Senescence across multicellular organisms, with a focus on social species

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Declaration

I declare that this thesis was composed by myself and that the work contained herein is my own except where explicitly stated in the text. This work has not been submitted for any degree or professional qualification except as specified.

Mark Roper

Michaelmas term 2022

Acknowledgements

I think to call completing a PhD a roller coaster is fairly accurate. However, no one tells you that its not as predictable. The climb to the top of the ride isn't straightforward, there are constant back and forths, jolts and bumps, and sometimes you start going backwards. The euphoria moments of speeding down a drop, or through a loop, can last moments before you spot the next insurmountable climb. The end of the ride is seemingly somewhere in the distance, before here I am, with this lazy attempt at poetry. Overall, however, I'm deeply grateful to the experience I was given to get on this ride, and I have too many people to thank for ensuring I got to the end (fairly) in tact. I will be forever grateful to you all.

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Abstract

Senescence, the increasing risk of mortality and/or decreasing rate of reproductive success, is, at first glance, detrimental to individual fitness. Developments in evolutionary theory for senescence across the 20th century, however, suggested that despite its detrimental effects, senescence is an inevitable outcome of evolution. The variable patterns of age-specific mortality and reproduction observed in nature suggest otherwise. In this thesis, I explore the variation in senescence across multicelluar organisms, with a particular focus on social species. Specifically, I: *(i)* review the current state of theoretical and empirical senescence research, suggesting evolutionary explanations for the variation in age-specific mortality and reproduction observed across species; *(ii)* quantify levels of covariation between metrics of actuarial (mortality) and reproductive ageing; *(iii)* model how social interactions between individuals might modulate ageing and senescence; *(iv)* comparatively test predictions of the model in social species; and *(v)* test for senescence in a wild population of a cooperative breeder. Overall, I suggest that the force of selection is still integral to understanding the evolution of senescence. The force of selection, introduced by Hamilton (1966), allows for the quantification of how strongly a detrimental allele will be selected against as a function of the age at which the allele invades. Hamilton's results suggested that the force of selection is always a monotonically declining function with increasing organism age. By breaking assumptions from Hamilton's model, however, we can observe forces of selection that need not always decline with age, and thus better predict the diversity of ageing patterns observed in nature.

Publications

The following chapters have resulted in publications:

Chapter Two

Roper, M., Capdevila, P. & Salguero-Gómez, R. (2021). Senescence: why and where selection gradients might not decline with age. *Proc. R. Soc. B.* 20210851

Chapter Six

Roper, M., Sturrock, N., Hatchwell, B., & Green, J. P. (2022). Individual variation explains ageing patterns in a cooperatively breeding bird, the long-tailed tit *Aegithalos caudatus*. *J. Anim. Ecol*. **91,** 1521-1534.

In addition, **Chapter Four** is in review at *Nature Ecology & Evolution*, and **Chapter Three** is being written up for publication. While producing this thesis, I also co-wrote the following published reply and bibliography, which are included in **Appendix B** and **Appendix C**.

Appendix B

Roper, M. & Salguero-Gómez, R. (2022). Reply to: Senescence, trait parameterization and (st)age-specific forces of selection. *Proc. R. Soc. B*. 289: 20212610.

Appendix C

Salguero-Gómez, R., Roper, M., Bernard, C. & Deere, J. 2021. Senescence. *Oxford Bibliographies*. DOI: 10.1093/OBO/9780199830060-0236

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1 | Introduction

1.1 Senescence: a never-ageing question

The causes and consequences of the physiological deterioration that accompanies old age in humans continues to stimulate academic research. The loss of physiological integrity that comes with ageing is a primary risk factor for many major human ailments, including cancer, diabetes, cardiovascular disorders, and neurodegenerative diseases (López-Otín *et al.* 2013). The actuarial focus of ageing research, and the importance of human demography for government policy (and the back pockets of life insurance companies), means the academic study of ageing is ever-expanding (Fig.1a).

In recent years, there has also been a revolutionary shift in focus to mechanisms of prevention, or even reversal, of the ageing process (Fig. 1b). This shift has been facilitated by major academic achievements, from the discovery that the rate of ageing is at least partially controlled by evolutionary conserved genetic pathways and biochemical processes (Gems & Partridge 2012), to the Hovarth clock (Hovarth 2013) and Yamanaka factors (Takahasi & Yamanaka 2006) highlighting mechanisms for potential ageing interventions.

For some, ageing is seen as a disease to be cured (de Grey & Rae 2007). Indeed, ageing now has a code (MG2A) in the International Classification of Diseases (ICD-11) and, recently, Altos Labs, the most heavily-funded biotechnology start-up to date (Kulcher 2022), has announced its mission to “reverse the diseases associated with ageing" (Ahuja 2021). The pursuit of increasing human health and well-being into older age, and even deterring death beyond current norms, are now an absolute scientific reality and focus.

In an influential review, López-Otín and colleagues (2013) describe nine ‘tentative hallmarks’ representing common denominators of ageing across organisms (López-Otín *et al.* 2013), albeit with a particular focus on mammals. These interconnected hallmarks summarise hundreds of potential mechanisms hypothesised to play a role in the ageing process, with their overall effect being to reduce the functional capacity of an organism in a time-dependent manner. When such a loss in functional capacity manifests itself at the whole-individual level, demographers observe senescence as an increasing risk of mortality and/or a reduction in reproductive success of the individual with increasing age

(Comfort 1964; Shefferson *et al.* 2017; Salguero-Gómez *et al.* 2021).

Despite being intuitively detrimental to evolutionary fitness, demographic senescence is observed across many wild populations (Nussey *et al.* 2013; Gaillard & Lemaître 2020). Longitudinal studies of wild populations, involving the repeated sampling of individuals across their lifespans, have provided depositories of high-resolution demographic data for numerous species (*e.g.* DATLife; Salguero-Gómez *et al.* 2015; Salguero-Gómez *et al.* 2016). Strikingly, cross-species analyses of data from these longitudinal studies have revealed that patterns of senescence are more variable than thought. In fact, some species not only show no apparent functional decline with age, but in some cases show demographic improvement with age (Finch 1990; Jones *et al.* 2014; Roper *et al.* 2021). These findings suggest that different ecological and evolutionary contexts can, and have, driven varying patterns of senescence. Understanding why and where across the tree of life senescence evolves is, therefore, an essential goal in our pursuit of increasing healthspan in humans (Baudisch & Vaupel 2012). This evolutionary perspective is fundamental to our understanding of the ageing process itself, and essential for identifying species for future model study. This doctoral thesis focuses on the variable rates of demographic senescence across species, and seeks to explore ecological and evolutionary explanations for such variation.

1.2 Definitions

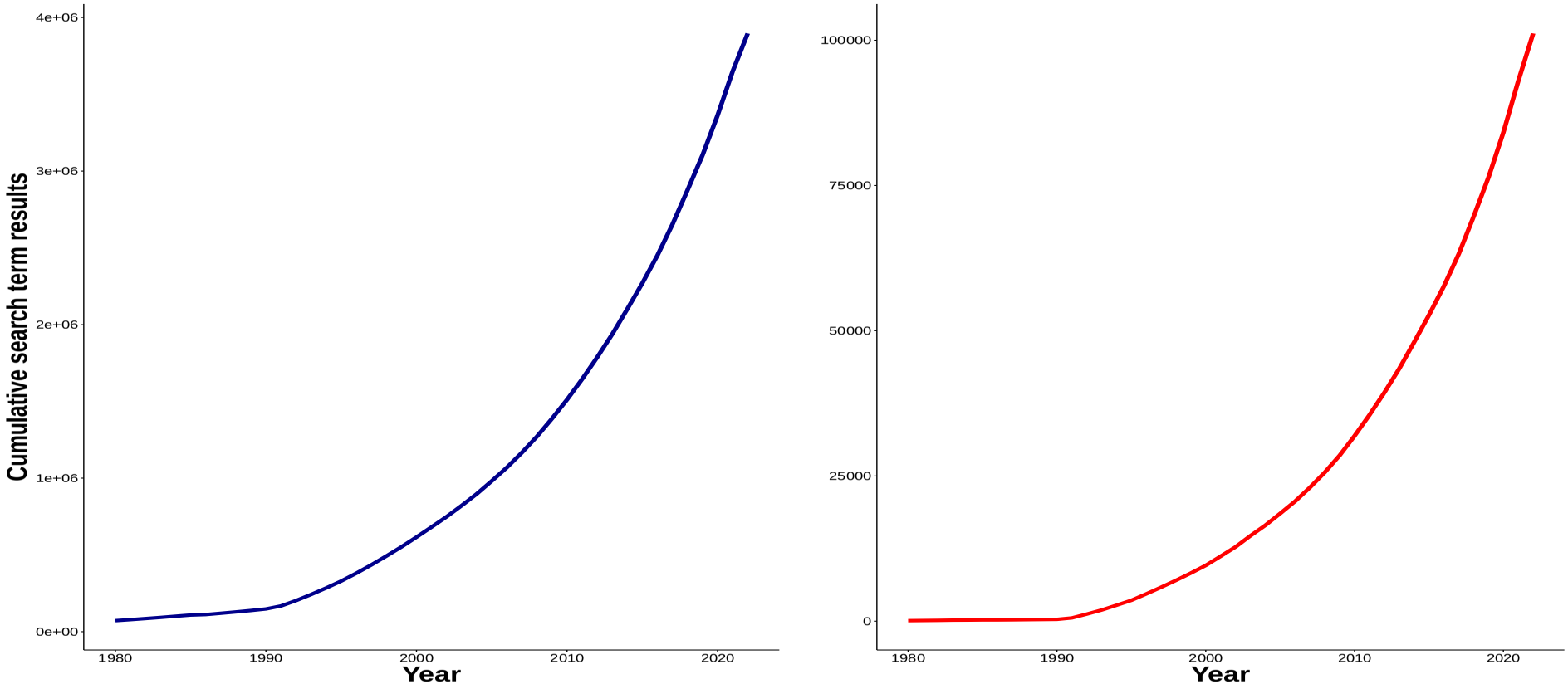
Ageing and senescence are often used interchangeably in the literature, both referring to the progressive loss of functional capacity with age. At the demographic level, however, more precision is necessary with these definitions. Following Medawar (1952), and numerous authors since, I define ageing as merely the passing of time as an organism survives, whereas senescence refers to the increasing risk of mortality (actuarial senescence) and/or decreasing rate of reproductive success (reproductive senescence) with increasing age. All organisms age so long as they survive a second, an hour, or a year, but not all necessarily undergo senescence. Following this perspective, I then further distinguish between the pace and the shape of ageing (Keyfitz 1977; Baudisch 2011). Pace refers to expected (reproductive) lifespan within a cohort, *i.e.* how long individuals live and reproduce, whereas shape describes the distributions of mortality and reproduction across the life course (Baudisch 2011; Baudisch & Stott 2019). In this

framework, pace and shape are mathematically orthogonal: a species' shape of mortality or reproductive distribution can be independent of how long they live. This approach has been supported by findings that patterns of senescence do indeed appear to be independent of lifespan (Jones *et al.* 2014; Péron *et al.* 2019).

1.3 Evolutionary theories of senescence

Hypotheses for evolutionary explanations for senescence date back to Weismann (1889) (**see A particular focus on social species**). However, it was insights in the 20th century from Fisher (1930), Medawar (1952), Williams (1957), amalgamated and mathematically formalised by Hamilton (1966), that put senescence on a contemporary evolutionary footing (Charlesworth 2000). I discuss these developments extensively in **Chapter 2**, so I refrain from doing so here. To summarise, senescence evolves because the 'force of natural selection' declines with increasing organismal age. This decline occurs because an omnipresent threat of mortality (Medawar 1952) and the production of new offspring (Wensink *et al.* 2017) leads to distinguishable age classes within a population, with the distributions of these age classes (stable age distribution) often being right-skewed (Fig. 2a). When this skew occurs, if individuals in less-represented age classes also have lower reproductive value – the relative contribution of an individual in an age class to the ancestry of future populations (Fisher 1930; Grafen 2006) – then any detrimental allele will have a lower probability of elimination than if it invaded at a younger age class. More exactly, the force of natural selection acting on an allele at age x can be estimated as the change in fitness due to the effect of the allele, which is proportional to the product of the stable age distribution of age and reproductive value (Hamilton 1966; Caswell 1978; Charlesworth 1994; Taylor & Frank 1996; Rose *et al.* 2007; Caswell 2010; Roper *et al.* 2021). When this product declines with age, senescence can evolve.

Figure 1. Cumulative search term results on Web of Science from January 1st 1980 to February 1st 2022 for search terms a) 'ageing' OR 'ageing' OR 'senescence' and b) 'anti-ageing' OR 'anti ageing'.



Since the classical evolutionary framework was introduced, further theoretical work has sought to explore necessary conditions for senescence. For example, Williams (1957) and Kirkwood (1977) posited that a segregation between germ line and soma is a necessary condition for senescence. In such organisms with a germ-soma divide, maintenance of the soma should only be selected for as long as such maintenance is required for successful reproduction (Kirkwood & Austad 2000). However, Partridge & Barton (1993) argued that even an organism comprised entirely of germ-cells would still have a non-zero probability of death. In a population of this hypothetical organism, older age classes would still contribute less to future populations, retaining a declining force of selection with age. Partridge & Barton's (1993) argument is supported by findings of senescence in unicellular organisms (no soma) with asymmetric division (Martinez & Levinton 1992; Ackermann *et al.* 2003). Interpreting these results leads to the argument that some form of asymmetry between individuals, that importantly is a function of age, is the ultimate necessary condition for senescence (Pen & Flatt 2021). This asymmetry will usually come from the rejuvenation found in new offspring. If all reproduction was symmetrical, and therefore all individuals had the exact same probability of death or rate of reproduction at any time, then 'age-structure' becomes meaningless, and senescence would not evolve.

Other research has attempted to decipher ecological and evolutionary factors that might make senescence more pronounced or, on the contrary, more strongly selected against. For example, there is debate in the literature as to the role extrinsic mortality plays in determining the rate of senescence (Williams 1957; Abrams 1993; Caswell 2007; Moorad *et al.* 2019; Day & Abrams 2020; de Vries *et al.* 2022). This debate stems from a prediction in Williams' (1957) seminal work that "Low adult death rates should be associated with low rates of senescence, and high adult death rates with high rates of senescence". Numerous authors have since argued over the interpretation of Williams' prediction, and considered how it might be modulated by age-specificity (Caswell 2007); density-dependence (Abrams 1993; Moorad *et al.* 2019; Day & Abrams 2020), condition-dependence (Williams & Day 2003), or might even be irrelevant (Moorad *et al.* 2019). In this thesis, I argue that the simple presence of extrinsic mortality is important for the evolution of senescence by, all else being equal, reducing the contributions of later age classes to future populations. Wensink *et al.* (2017) modelled a population with immortal individuals, however, and showed that births alone can bias the

age class distribution towards younger classes. In turn, this can facilitate a declining force of selection with age without any mortality in the population. Biologically speaking, however, threats of extrinsic mortality are ubiquitous across species, and are therefore still likely to contribute, in tandem with reproduction of newborn individuals, to a decline in the force of selection with age. From a theoretical perspective, the exact effect of extrinsic mortality on the rate of senescence, however, will depend on the assumptions one also makes about condition-and/or density-dependence (de Vries *et al.* 2022).

Given the classical evolutionary theoretical framework for senescence and its extensions, one can make simple or more nuanced predictions for how senescence should evolve across the tree of life. Keeping things simple, many studies still consider the logic of Hamilton (1966) – that the force of selection should decline from birth for reproduction, and from maturity for mortality – as baseline predictions to test in wild populations (Jones *et al.* 2014). As this logic is rather general and its only requirements are the existence of age-specific risk of mortality and rate of reproduction, Hamilton's model is interpreted as predicting that senescence should be universal (Hamilton 1966).

1.4 Senescence across wild populations

Understanding senescence from an evolutionary ecology perspective relies on studying wild populations, which in turn relies on intensive study of a species' demography. From longitudinal records (Nussey *et al.* 2008; Clutton-Brock & Sheldon 2010) of births and deaths of known-age individuals, one can separate within- and between-individual variation in survival and reproduction (van de Pol & Verhulst 2006; Metcalf 2016) and ask how senescence has evolved in the wild. This 'gold-standard' of senescence research naturally leads, however, to taxonomic biases in the populations studied (Troudet *et al.* 2017; Conde *et al.* 2019). These biases are likely to influence general conclusions for how senescence has evolved. For example, studying the life cycles of some species are well beyond the scope of the active research careers of bio-demographers, let alone lifespans of research grants. In other species, study in wild settings might be challenging or even unfeasible (Zajitschek *et al.* 2020). Many corners of the tree of life are still vastly under-represented with respect to demographic research of natural populations (Shefferson *et al.* 2017), but are beginning to be explored thanks to longitudinal

explorations of more traditionally under-represented taxa (Barks & Laird 2015; Zajitschek *et al.* 2020; Jordan *et al.* 2021), aided by advancements in methodology (Horvitz & Tuljapurkar 2009; Colchero *et al.* 2012; Koons *et al.* 2014).

Exhaustive literature reviews of the prevalence of senescence across wild populations are provided in **Chapters 2** and **5**, and so they are not conducted here. However, a general picture of senescence across the tree of life can be detailed by noting that age-related functional decline from maturity is neither inevitable nor universal, and that instead patterns of mortality and reproduction across species are highly variable (Finch 1990; Vaupel *et al.* 2004; Baudisch *et al.* 2013; Jones *et al.* 2014; Jones & Vaupel 2017; Roper *et al.* 2021). There appears to be some phylogenetic inertia: most mammals display senescence (Gaillard & Lemaître 2020), whereas 93% of angiosperms studied do not (Baudisch *et al.* 2013). Growth form (Colchero & Schaible 2014) and modularity (Bernard *et al.* 2019) might be important explanatory variables for determining senescence outcomes. Many species with deterministic growth (*i.e.* grow to a fixed adult size) appear to display senescence, whereas indeterminate growth appears to be somewhat correlated with negative senescence (Vaupel *et al.* 2004). More modular plants (*i.e*. those that are physiologically more compartmentalized) have a greater chance of escaping senescence than those that are unitary (Bernard *et al.* 2019). Furthermore, many species appear to display asynchronous rates of actuarial and reproductive senescence (Jones *et al.* 2014; Hayward *et al.* 2015; Gaillard & Lemaître 2017; Roper *et al.* 2021). Explaining this widespread variation in rates of actuarial and reproductive senescence from an eco-evolutionary perspective is the main focus of this thesis.

1.5 Why might patterns of senescence vary?

If we take Hamilton's (1966) model as a baseline for predicting the evolution of senescence, one can consider how each of his assumptions might play a role in determining the outcome. Hamilton assumed *(i)* that the intrinsic growth rate of a population, r, is an appropriate measure of fitness, *(ii)* that genetic mutations multiply survival probabilities, *(iii)* that changes to mortality and reproduction occur independently; *(iv)* a population with no structure or social interactions, and, finally, *(v)* that population dynamics can be adequately described according to age-structure. When one alters these assumptions, different predictions for the evolution of senescence might

arise.

As I discuss in **Chapter 2**, Caswell's (2010) generalisation of Hamilton's results reveal that the product of reproductive value and the stable age distribution of an age class provides a proxy for the relative contribution of such an age class to future populations. In many species, this product will decrease with age due to mortality and reproduction (Fig. 2A). When this decline occurs, selection against senescence becomes weaker, *i.e.* the force of selection declines with age. If some set of ecological or evolutionary factors drive a population towards age structure and reproductive value distributions for which the product of these distributions does not decline with age, then senescence should be more strongly selected against (Fig. 2B). Breaking Hamilton's assumptions might therefore facilitate model solutions in which the product of these distributions does not decline monotonically with age, or declines at a slower rate, thus generating variation in predicted outcomes of rates of senescence.

Numerous authors have deviated from Hamilton's original assumptions to seek explanation for the variable rates of senescence in nature. For example, Lee (2003) introduced the concept of nutritional intergenerational transfers, showing how the force of selection acting on mortality can increase during the juvenile period and remain above zero for post-reproductive adults. Vaupel and colleagues (2004) introduced growth into a life history optimisation model of senescence. They showed that increased reproduction with age (negative senescence) can be favoured when it pays to sacrifice current reproductive output if such a sacrifice markedly increases body size, and therefore, future reproductive output. Baudisch (2005) altered the genetic assumptions of Hamilton's model, showing how alternative parameterisation of mutational effects can lead to forces of selection that do not decline with age. Finally, Caswell & Salguero-Gómez (2013) modelled the force of selection allowing mortality and reproduction to depend both on the age and stage of individuals. In such populations, the force of selection need not always decline with age. From this work, a general explanation has emerged for the diversity of senescence rates we find in nature – not all species' demography can be adequately explained by the assumptions Hamilton made.

In **Chapter 3**, I test for covariation between the pace and shape of actuarial and reproductive ageing across multicellular plants and animals to assess the validity of Hamilton's assumption *(iii)* above. In **Chapter 4**, I relax two more of Hamilton's assumptions. First, that r is an appropriate measure of evolutionary fitness for all

species, instead exploring how senescence might evolve differently when inclusive fitness is considered as the metric which natural selection works to optimise. This approach necessarily involves also breaking Hamilton's assumptions of no population structure and no social interactions between individuals. Altering these assumptions will generate predictions for how senescence should evolve across social species.

1.6 A particular focus on social species

"The word 'direct' is important. Grandparents, though no longer fertile may promote (or impede) the welfare of their grandchildren, and so influence the mode of propagation of their genes... Selection for grandmotherly indulgence I should describe as 'indirect', and the indirect action of selection becomes important whenever there is any high degree of social organization"

Peter Medawar *in* An Unsolved Problem of Biology (1952)

Post-reproductive survival provides the most obvious challenge of Hamilton's models when considering the effects of sociality on senescence. According to Hamilton's results, the strength of selection on further survival of any age class with no future reproduction is equal to zero. We therefore should not expect individuals to live long at all beyond the age of last reproduction, but post-reproductive survival is observed in humans, across a few other mammals (Cohen 2004; Ellis *et al.* 2018; Chapman *et al.* 2019), and aphids (Uematsu *et al.* 2010). Hamilton himself noted (pg. 23, Hamilton 1966) that "the inclusive fitness of an individual is maximized by continually acting in ways that cause increases in its inclusive reproductive value". Yet, his models of senescence only considered conventional reproductive value and r as the measure of fitness, not inclusive reproductive value and inclusive fitness (Hamilton 1964a; 1964b). There remains a gap in the literature for how considering inclusive fitness will alter the moulding of senescence by natural selection and by kin selection.

Even in one of the earliest accounts of an evolutionary theory for senescence, August Weismann (1889, 1891) suggested that senescence might have evolved in part due to some social aspect. Weismann's theory suggested that senescence had evolved to remove older individuals from the population who contribute less to fitness. Although clearly circular – senescence was assumed but the theory aimed to actually explain its evolution – and adopting a naïve group selection perspective, Weismann was the first to consider

the potential for senescence to be under selection for its effects on other individuals.

In the quotation above, Medawar (1952) also referenced the importance of indirect selection when demography is socially structured for post-reproductive survival. It was not until 50 years later, however, that Bourke (2007) hypothesised verbal mechanisms for a relationship between kin selection and the evolutionary theories of ageing. Bourke's essay, and further recent reviews (Lucas & Keller 2020; Korb & Heinze 2021), represent a budding field of senescence literature – the sociality-senescence interaction – that has been lingering in the minds of theoreticians and empirical biologists since the conception of senescence research itself. I detail the work that has been explored on social senescence, key results, and limitations, extensively in **Chapters 4**, **5** and **6**.

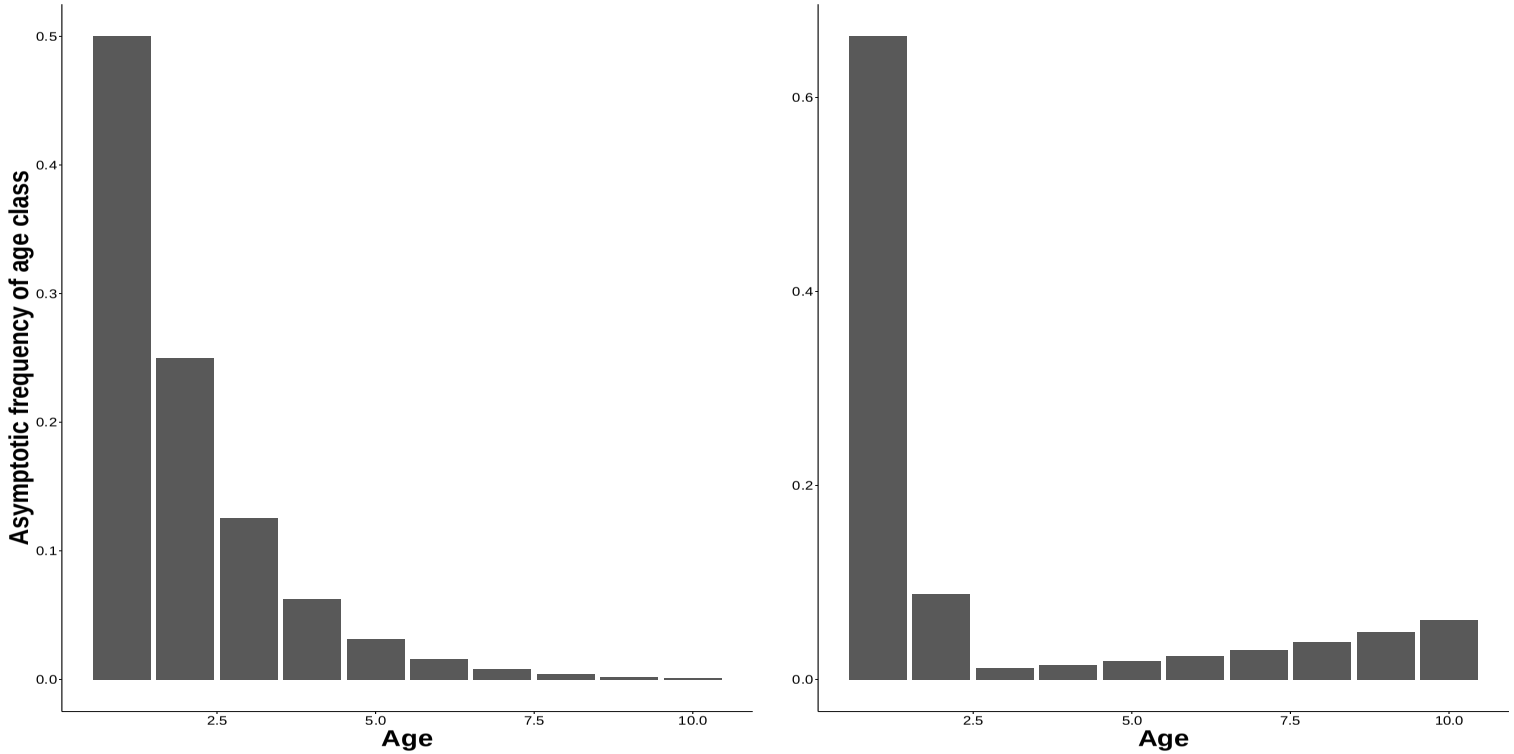
In considering the scope for social interactions to shape senescence, this thesis takes particular inspiration from the models of Lee (2003) and Ronce & Promislow (2010). As discussed earlier, Lee's (2003) model was a major development in the field, quantifying how the force of selection might differ in the face of social interactions. Yet, Lee's model assumed no population structure, and so the full consequences of kin selection could not be explored. The model also did not formally use inclusive fitness as a measure of fitness. Ronce & Promislow (2010) did model population structure; however, their model was limited to single individuals competing for patches, and was therefore limited to exploring parent-offspring conflict. Here, I use insights from both of these models to develop a general inclusive fitness model of senescence (**Chapter 4**) that considers multiple individuals with social interactions. Social populations have many features that can be hypothesised to alter the evolution of senescence. These include:

- Helping behaviour that leads to direct or indirect fitness returns might alter inclusive reproductive value and expected age class distributions. For example, juveniles in classical models gain no fitness until maturity, but might do so in social models.
- Mortality and reproduction of a focal individual not only affects the fitness of a focal individual, but also have the potential to alter the fitness of other local individuals (Bourke 2007).
- Subordinate *vs.* dominant, or breeder vs non-breeder phenotypes may be modelled with stage structure, and as such might have divergent senescence phenotypes (Kreider *et al.* 2021).
- Other demographic features of social systems, *e.g.* how groups form or levels of

reproductive skew, also have the potential to modulate the expected age class and inclusive reproductive value distributions, and therefore the evolution of senescence.

I explore these features and develop hypotheses for how age-specific inclusive fitness and inclusive reproductive value, combined with social demography, might shape senescence in social species in **Chapter 4**. In **Chapter 5**, I test these hypotheses comparatively across longitudinal dataset of social vertebrates.

Figure 2. Simulated asymptotic frequencies (stable age distributions) for two hypothetical iteroparous populations. In both populations there are 10 age classes beyond which the probability of survival is zero. Reproduction begins at age class 3 and is constant throughout adult life. In population A, survival probability is constant throughout life, leading to a right-skewed age class distribution. In population B, survival probability to maturity is 0.01, and then constant (and high = 0.95) throughout adulthood. The age class distribution of population B also has right skew, but the older age classes are better represented. All else being equal, senescence is therefore more likely to be under stronger purifying selection in older age classes in population B – conditional on the shape of reproductive value.



1.7 Thesis Outline

Chapter 2 sets the foundations for the thesis. Performing both a literature review of classical and contemporary evolutionary theory of senescence, as well as empirical data for senescence in wild populations, the chapter then proposes key explanatory predictors for variation in senescence rates across multicellular species. I argue that Hamilton's (1966) logic is foundational for any theory of senescence, and that understanding why different patterns of senescence evolve first requires an understanding of how ecological and evolutionary factors alter two key variables: population structure and reproductive value. I discuss how asynchronous patterns of survival and reproduction, stage structure, and social interactions might all alter these two key variables, and thus patterns of senescence, which sets the seed for the remaining chapters of the thesis. I conceived the project with Roberto Salguero-Gómez. Pol Capdevila and I conducted the analyses, with input from Roberto Salguero-Gómez, and produced all visualisations. I drafted the first version of the manuscript and, together with Pol Capdevila and Roberto Salguero-Gómez, revised and edited the manuscript.

In **Chapter 3**, collaborating with Iain Stott (University of Lincoln) and using a pace-shape framework of ageing, I test for covariation between the pace and shape metrics of actuarial and reproductive ageing. Using data from open access repositories of age and stage based species demography, I show systematic differences in the magnitude and direction of covariation between ageing metrics across animals and plants. Roberto Salguero-Gómez, Iain Stott and I conceived the project. I conducted all data collection, analyses, and produced all visualisations with input from Iain Stott and Roberto Salguero-Gómez. I drafted the first version of the manuscript and, together with Iain Stott and Roberto Salguero-Gómez, revised and edited the manuscript.

In **Chapter 4**, I switch attention to the evolution of senescence in social species. In this chapter, I develop a general model that quantifies inclusive fitness forces of selection in an age-structured population. The model consider how social interactions and other demographic characteristic of social species might shape forces of selection relative to Hamilton's (1966) original models. Using an infinite island modelling framework, I show analytically that the force of selection on survival rate at age x is a balance between inclusive reproductive value at age x+1 and the inclusive reproductive value of newborns, weighted by the coefficients of relatedness between an individual age x and a newborn. I

also show that the force of selection on reproduction at age x is a balance between the direct benefits and indirect costs of displacing related offspring. Michael Bonsall, Jonathan Green, Roberto Salguero-Gómez, and I conceived the project. I conducted all modelling and derivations with Michael Bonsall, and produced all visualisations with input from Michael Bonsall, Jonathan Green, and Roberto Salguero-Gómez. I drafted the first version of the manuscript and, together with Jonathan Green, Roberto Salguero-Gómez, and Michael Bonsall, revised and edited the manuscript.

In **Chapter 5**, I conduct a comparative analysis of variation in lifespan and rates of actuarial and reproductive senescence across species that exhibit alloparental care. To do so, I built an exhaustive database of age-specific estimates of survival probability and rate of reproduction, derived from longitudinal demographic studies of each species. Using predictions from the model in **Chapter 4**, I explored the relationships between variation in key features of these social species (relatedness, skew, magnitude of help) and three response variables: expected adult lifespan, actuarial senescence, and reproductive senescence. I found that increased relatedness within a group was associated with a reduced expected adult lifespan, and a shift in reproduction towards older age classes (*i.e.* weaker reproductive senescence. Jonathan Green, Roberto Salguero-Gómez, Michael Bonsall and I conceived the project. I conducted all data collection and tidying with input from Jonathan Green. I conducted all analysis, and wrote the Chapter with input from Michael Bonsall, Roberto Salguero-Gómez, and Jonathan Green.

Finally, in **Chapter 6**, I use empirical data from a wild population of the cooperatively-breeding bird, the long-tailed tit (*Aegithalos caudatus*). I chose this system to investigate senescence due to the available quantification of lifetime reproductive success for a large number of individuals (>800), the ability to split fitness effects between helpers and breeders due to a simple helping system, and the ability to test for senescence in helping behaviour. I conducted this study before developing the theoretical work in **Chapter 4**, but applications of the theory to empirical systems are presented in the **Discussion**. Using data collected over 25 years, I examined the age-trajectories of survival and reproduction in adult long-tailed tits to determine how these were affected by the presence or absence of helpers, and how helper behaviour changed with age. There was evidence for increased reproductive performance with breeder age, but no effect of age on the probability of survival. Within a breeding season, breeders that were

assisted by helpers enjoyed greater reproductive success through enhanced offspring recruitment in the following year. I found no evidence that age affected an individual's propensity to help, or the amount of indirect fitness accrued through helping. I also found a positive correlation between lifespan and multiple components of reproductive success, suggesting that individual variation in quality underpins age-related variation in fitness in this species. Jonathan Green and I conceived the project. Ben Hatchwell (University of Sheffield) manages the field study and has collected data on which analyses are based over 25 years. I analysed all data with input from Nicole Sturrock and Jonathan Green. I wrote the first version of the manuscript, and along with all other authors revised and edited the manuscript.

1.8 General Introduction Bibliography

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2 | Senescence: why and where selection gradients might not decline with age

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Senescence: why and where selection gradients might not decline with age

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Patterns of ageing across the tree of life are much more diverse than previously thought. Yet, we still do not adequately understand how, why and where across the tree of life a particular pattern of ageing will evolve. An ability to predict ageing patterns requires a firmer understanding of how and why different ecological and evolutionary factors alter the sensitivity of fitness to age-related changes in mortality and reproduction. From this understanding, we can ask why and where selection gradients might not decline with age. Here, we begin by summarizing the recent breadth of literature that is unearthing, empirically and theoretically, the mechanisms that drive variation in patters of senescence. We focus on the relevance of two key parameters, population structure and reproductive value, as key to understanding selection gradients, and therefore senescence. We discuss how growth form, individual trade-offs, stage structure and social interactions may all facilitate differing distributions of these two key parameters than those predicted by classical theory. We argue that these four key aspects can help us understand why patterns of negligible and negative senescence can actually be explained under the same evolutionary framework as classical senescence.

1. Classical evolutionary framework of senescence

Senescence, the increasing risk of mortality and decline in reproduction with age after maturity, has long been explained by a collation of theories defining the 'classical evolutionary framework of senescence'. The central logic common to these theories argues that the force of natural selection weakens with age [1–4]. Selection becomes too weak to oppose the accumulation of genes that negatively affect older age classes [1], or favours these genes if they also have beneficial effects at earlier ages in life [2], when the contribution individuals make to future populations is assumed to be greater. Selection should therefore favour resource investment into earlier reproduction rather than late-life maintenance [4], or at least only invest in non-reproductive functions for as long as they would naturally be required in the wild.

Underpinning all evolutionary theories of senescence is the omnipresence of extrinsic mortality. The later age classes of such a cohort should, all else being equal, contribute less to the ancestry of future generations [5]. Medawar [1] predicted this will create a 'selection shadow', whereby late-age-acting deleterious alleles will be less effectively removed by selection (mutation accumulation). Building on this, Williams [2] instead developed the 'antagonistic pleiotropy' theory of ageing [6,7], where he argued that such late-acting detrimental alleles could actually invade under positive selection if they had beneficial pleiotropic effects at younger ages.

Kirkwood [4] also took an optimality approach but framed his 'disposable soma' theory from a physiological perspective. Because it is the germ line that survives through generations, natural selection will only favour a level of somatic investment that optimizes the success of the germline [8]. As laid out in a thought experiment by Partridge & Barton [9], however, a hypothetical

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organism composed solely of germ line cells will still experience a non-zero probability of death and/or become unable to reproduce. The intensity of selection on the organism's survival and reproduction will therefore still decline with age, and senescence will still evolve. Evidence that senescence can occur in asexual metazoans [10] and bacteria with asymmetrical division [11] support this notion that a germ–soma divide is neither necessary nor sufficient for the evolution of senescence. Instead, the ultimate requirement for senescence is asymmetry between parent and offspring, essentially age structure, that makes individuals different in the eyes of selection [9].

These verbal ideas for the evolution of senescence were formalized mathematically by Hamilton [3], who used Fisher's [5] assumption that the Malthusian parameter r is an appropriate measure of fitness

$$\int_0^\infty e^{-rx} l(x) m(x) dx = 1. \tag{1.1}$$

The above equation is the Euler–Lotka equation [7] and r, also known as the population growth rate, is the single real root. The terms $l(x)$ and $m(x)$ define the probability of survival to, and reproduction at, age x respectively. Using implicit partial differentiation of equation (1.1), Hamilton calculated the effects on r of small changes in survival or reproduction at some age x,

$$\frac{\partial r}{\partial m(x)} \approx \frac{e^{-rx} l(x)}{T} \tag{1.2}$$

and

$$\frac{\partial r}{\partial \mu(x)} \approx -\frac{\int_x^\infty e^{-ry} l(y) m(y) dy}{T}, \tag{1.3}$$

where $\mu(x) = -(d\ln l(x)/dx)$ and defines mortality risk at age x, and T is generation time. The resulting quantities represent 'forces of selection' on age-specific vital rates [3]. The larger the absolute value of the quantity, the stronger the response of selection to a given change [7,12]. Equation (1.2) shows how the force of selection on reproduction at age x is proportional to survival at age x should decrease with age in a stationary or growing population, whereas equation (1.3) formulates how the force of selection acting against (hence the negative sign) an increase in age-specific mortality declines from the age at first reproduction [3,7,12–16] (but see Baudisch [17] for other indicators of selection where the forces of selection do not necessarily decline with age).

How extrinsic mortality may drive variation in rates of senescence has been debated off the back of one of Williams's nine predictions (see Gaillard & Lemaître [18] for a review of each) for the evolution of senescence [2]. Williams stated that 'Low adult death rates should be associated with low rates of senescence, and high adult death rates with high rates of senescence' [2]. Although the legitimacy of this prediction is debated [19–21], a general consensus seems to suggest that a change in extrinsic mortality has to be age-dependent to alter the rate of senescence [22]. An age-independent change in mortality, by definition of being age-independent, will mean the overall selection gradient should still follow the same pattern over all age classes if exponential growth is assumed [14,22]. If extrinsic mortality interacts with physiological condition, however, this could alter ageing rates [23]. Williams & Day found such an interaction to have a general tendency to strengthen selection against senescence at all ages, but potentially stronger effects at younger ages, favouring slower senescence earlier in life and more rapid senescence later in life [23].

To summarize the classical evolutionary framework and subsequent theoretical elaborations for age-structured populations, senescence (i) has not evolved adaptively without pleiotropy, (ii) occurs as a by-product of weak selection, and (iii) requires asymmetry between parent and offspring (i.e. requires age structure). Based on Hamilton's model [3] (equations (1.2) and (1.3)), we should expect to see the risk of mortality rise and reproduction decline throughout adulthood in any age-structured population. As we will show for 475 species of animals and plants, supporting previous research [24], many species in fact display negligible [25] or even negative [26] senescence, where the risk of mortality remains constant or decreases with age, and reproduction remains constant or increases with age. This phenomenon occurs because these species are not solely age-structured, in the sense that chronological age *per se* is not the principal driver of their demography [27]. For such species, selection gradients may not monotonically decline with age throughout adulthood, or a decline may be delayed [28,29].

2. Current data

With ever-growing amounts of readily available longitudinal demographic datasets [30,31], comparative demography offers a tool to reveal the diversity in ageing patterns across the tree of life [24,32]. Using high-resolution demographic information for wild populations of 80 animal and 395 plant species worldwide (see electronic supplementary material for methods), we provide a quantitative evaluation of the rates of actuarial senescence—the change in mortality risk with age after maturation—across a subset of multicellular organisms. We use a 'pace-shape' framework of ageing [33,34]. In it, *pace* of ageing quantifies the speed of life via mean life expectancy [34], whereas the *shape* of ageing (i.e. senescence rate) quantifies the spread and timing of mortality events, normalized by mean life expectancy. The shape metric, S, is bound between −0.5 and 0.5, where $S > 0$ indicates that more mortality events occur at advanced ages (i.e. positive actuarial senescence), while $S < 0$ indicates low mortality late in life (i.e. negative actuarial senescence [35]). We quantify the role of evolutionary history on actuarial senescence across our 475 species by estimating its phylogenetic signal [36] using phylogenies for animals [37] and plants [38], respectively. Finally, we use derived life tables [39] from both age and stage-based models (see electronic supplementary material) to quantify age-specific reproduction rates ($m(x)$) to evaluate whether they match their patterns of actuarial senescence.

The majority of animal species (59 out of 80 species) display a negligible change in their risk of mortality with age. Positive actuarial senescence is especially scarce across invertebrates in our data, with the water flea (*Daphnia pulex*; figure 1*a*) as the sole example of positive actuarial senescence. The remaining 14 invertebrate species display negligible actuarial senescence, as in the case of the long-wristed hermit crab (*Pagurus longicarpus*; figure 1*a*), or even negative actuarial senescence, for example, the sea whip (*Leptogorgia virgulata*; figure 1*a*). Across vertebrates, 72% of species display little change in the risk of mortality with age (figure 1*a*; electronic supplementary material, table S1). Positively senescent species, however, are

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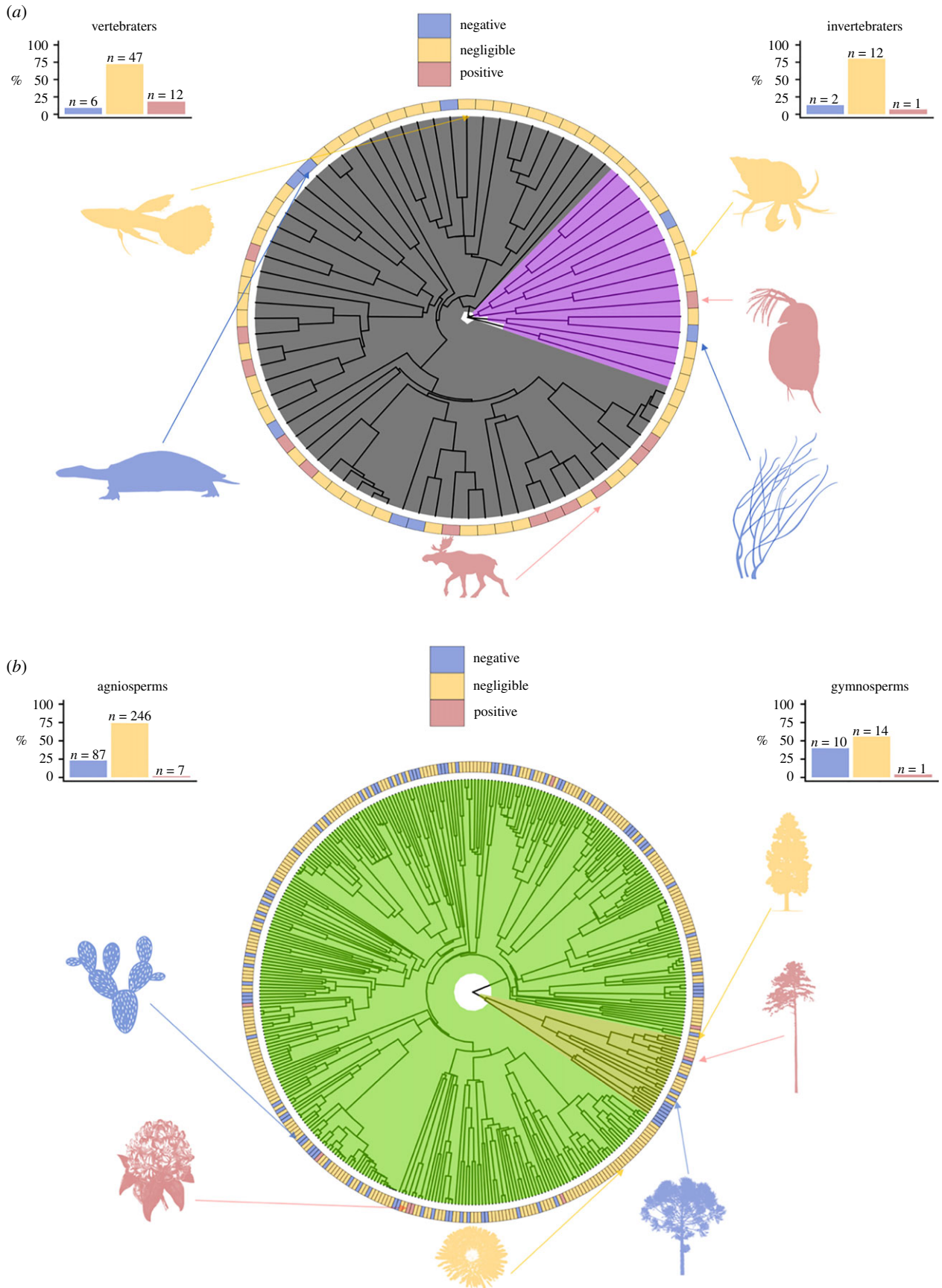


Figure 1. The evolution of and escape from senescence across multicellular life. Positive, negligible and negative patterns of actuarial senescence are dispersed throughout the four examined clades, with the percentages of each pattern within each clade shown in the bar charts of each of the figures. (*a*) Actuarial senescence across animals. Depicted around the phylogeny are six representative species, displaying positive (red), negligible (yellow) and negative (blue) senescence from each clade. Clockwise, representing invertebrates, these species are *Pa. longicarpus*, *D. pulex* and *L. virgulata*. For vertebrates, again clockwise, these species are *A. alces*, *P. expansa* and *Poecilia reticulata*. (*b*) Actuarial senescence across plants. Depicted around the phylogeny are six representative species, displaying positive (red), negligible (yellow) and negative (blue) senescence from each clade. For gymnosperms, these species are *Pi. lambertiana*, *Pi. sylvestris* and *Taxus floridana*. For angiosperms, these species are *Hypochaeris radicata*, *Rhododendron maximum* and *Opuntia rastrera*. (Online version in colour.)

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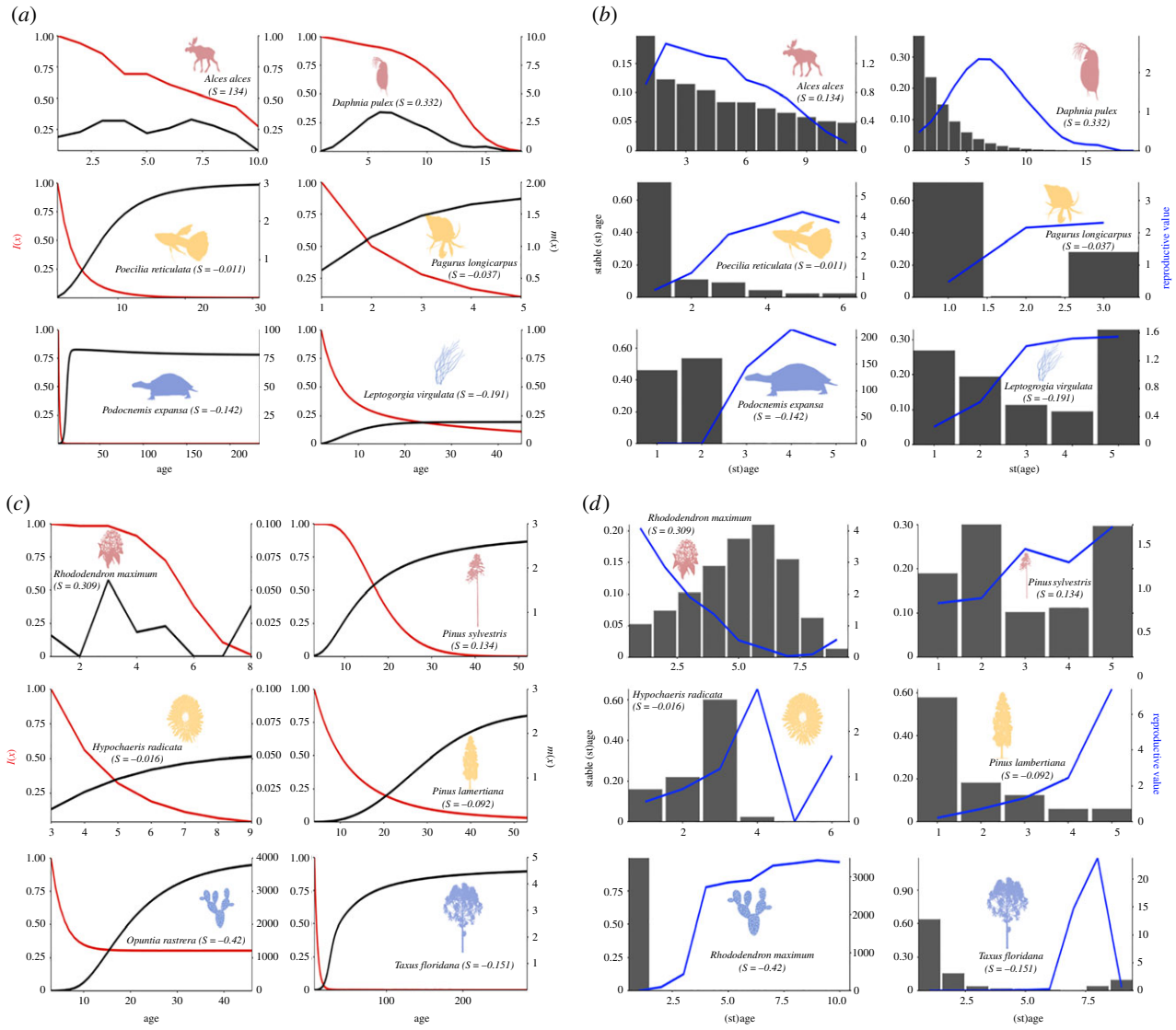


Figure 2. Age-based patterns of survivorship ($l(x)$—red) and reproduction ($m(x)$—black) are often decoupled, whereas reproductive value and stable age distributions predict the patterns of senescence. (*a*) $l(x)$ and $m(x)$ trajectories for the six selected animal species from figure 1. (*b*) Stable (st)age and reproductive value trajectories for the six selected animal species from figure 1. (*c*) $l(x)$ and $m(x)$ trajectories for the six selected plant species from figure 1. (*d*) Stable (st)age and reproductive value trajectories for the six selected plant species from figure 1. $l(x)$ and $m(x)$ trajectories are conditional upon reaching the age of maturity, at which the mature cohort is defined to have entered adulthood with a survivorship of 1. The trajectories of $l(x)$ and $m(x)$ run from age at maturity to the age at which 5% of the mature cohort is still alive. Stable (st)age and reproductive value trajectories are displayed for the whole life course. (Online version in colour.)

more common in vertebrates (18%; 12 out of 65 species) than invertebrates (6%; 1 out of 15 species); these species are primarily mammals (75%; electronic supplementary material, table S1) such as the moose (*Alces alces*; figure 1*a*). The majority of examined plant species also display negligible senescence. Indeed, only 2% of the 375 examined plant species exhibit positive senescence, including the scots pine (*Pinus sylvestris*) and the great laurel (*Rhododendron maximum*; figure 1*b*). Approximately 23% of angiosperms show a decreasing risk of mortality with age (e.g. *Opuntia rastrera*; figure 1*b*), compared to 40% of gymnosperm species (e.g. *Pinus lambertiana*; figure 1*b*). Overall, 98% of our studied plant species do not display a substantial increasing risk of mortality with age.

Estimates of phylogenetic signal on actuarial senescence are weak across the pool of examined animals (electronic supplementary material, figure S1 and table S3). Specifically, Pagel's λ [36], which ranges from zero (weak signal) to one (strong signal), is not significantly different from zero for across all the examined animal species ($\lambda = 0.22$, $p = 0.18$), nor when considering vertebrates and invertebrates separately (electronic supplementary material, table S3). These results indicate that the patterns of senescence across animals cannot be explained by phylogenetic relatedness, assuming a Brownian model of evolution. More thorough phylogenetic analyses are required, however, to rule out the effects of ancestral constraint on the patterns of senescence in animals. On the other hand, phylogenetic relatedness plays some role in senescence patterns across plants (electronic supplementary material, figure S2 and table S3). The analysis including angiosperms and gymnopserms raises a Pagel's λ of 0.31 ($p < 0.001$), most likely due to the significant phylogenetic signal in angiosperms ($\lambda = 0.27$, $p = 0.001$), compared to the non-significant signal in gymnosperms ($\lambda = 0.27$, $p = 0.08$).

Patterns of reproduction ($m(x)$) are diverse and appear somewhat independent of whether the examined species displays actuarial senescence (figure 2*a*,*c*; electronic supplementary material, figure S3). In plants, for example, both the scots pine and the great laurel display actuarial

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senescence (figure 1*b*), but their reproductive outputs do not decline with age (figure 2*c*). This pattern is contrasting to both examples of animals displaying positive senescence, where the moose (*A. alces*) and water flea (*Daphnia pulex*) also display reproductive decline with age (figure 2*a*). The flatweed exhibits negligible actuarial senescence and a relatively constant $m(x)$ trajectory, whereas the long-wristed hermit crab and the sugar pine also display negligible actuarial senescence but have increasing $m(x)$ trajectories. It appears from our study species that survivorship and reproduction can follow independent age-based trajectories.

Long-term individually based studies remain the gold standard for studies of senescence [40–42]. Only by tracking cohorts of individuals throughout their entire lifespans—or by being able to back-track age linked to performance [43]—can one account for variation in quality of individuals, and the subsequent issues of selective appearance and disappearance [44]. Long-term individually based studies are currently taxonomically biased [45], however, mostly limited to birds and mammals [46]. The matrix population models we use here, and the robust methods to derive age-based trajectories of survival and reproduction from them [27], offer an insight into the diversity of ageing rates in a taxonomically broad manner. By observing these broad-scale patterns, we can now isolate and understand these mechanisms behind this variation of age trajectories of mortality and reproduction, elucidating why some species succumb to senescence while others appear to escape its forces [28,29].

3. Why and where species may not senesce

It is practical to refer back to Hamilton's models as a starting point. Caswell's [15] reformulation of equations (1.2) and (1.3) show what factors drive the change in the force of selection with age [14–16],

$$\frac{\partial r}{\partial m(x)} \approx \frac{c(x)}{bT} \tag{3.1}$$

and

$$\frac{\partial r}{\partial \mu(x)} \approx \frac{-c(x)v(x)}{bT}, \tag{3.2}$$

where $c(x)$ gives the proportion of individuals aged x at the stable age distribution, $v(x)$ is the reproductive value [5] (the average contribution to the ancestry of future generations), b is the birth rate $\left(b = \left[\int_0^\infty \mathrm{e}^{-rx} l(x)\,\mathrm{d}x\right]^{-1}\right)$ and bT scales the values of the force of selection. For any given (st)age of a life cycle, the force of selection on an increase in the reproduction or mortality of that (st)age is proportional to the product of two key components: (i) the stable age distribution of individuals at that (st)age and (ii) the reproductive value of individuals that the (st)age contributes to the population (new offspring or their surviving selves). In equation (3.1), the stable age distribution of individuals aged x is weighted by the reproductive value of newborns, which is defined as 1. Importantly here, selection gradients need not always decline with age, or their decline may be delayed. This is because demographic or ecological factors may alter the stable age distribution or reproductive value profile of a population in such a way that maintains stronger selection with age.

Figure 2*b*,*d* exemplifies the importance of reproductive value and population structure for explaining divergent patterns of senescence (see also electronic supplementary material, figure S3). All species (except *Pinus sylvestris*; figure 2*d*) display reproductive value and stable age distributions that match their classification of actuarial senescence. Species that display positive actuarial senescence have distributions biased to earlier (st)ages, and those that do not have distributions that are more heavily skewed towards later (st)ages. Our assumption of each population sampled being at a stable state at the time of data collection provides the most intuitive reason why the patterns (including for *Pinus sylvestris*) may not match. It is clear from our analysis that reproductive value and population structure are key to understanding variation in patterns of senescence.

In identifying ecological and evolutionary mechanisms that might lead to alternative ageing patterns than those suggested by Hamilton, we suggest to ask two questions:

(1) How does the ecological and/or evolutionary mechanism alter the reproductive value and stable age distributions of the population from that expected of a standard age-structured population?
(2) How do the resulting distributions affect the evolutionary outcome for senescence?

Here, we consider how (i) the many components of reproductive senescence and trade-offs between survival and reproduction, (ii) the stage structuring of populations, and finally (iii) the spatial structuring of populations can act as ecological and evolutionary mechanisms that alter reproductive value and stable age distributions in ways that drive the evolution of senescence in different directions than those predicted from the classical evolutionary framework.

(a) Reproductive senescence

Survival and reproduction are intrinsically linked; the evolutionary outcomes for one make little sense without consideration of the other. Considered in isolation, however, there is a relative simplicity afforded by the monotonic nature of survival curves—everybody must eventually die. This simplicity has facilitated the conception of measures of shape [34], entropy [33] or lifespan equality [47] to describe the distribution of deaths, and afforded the ability to describe mortality curves with simple functions (e.g. Weibull or Gomerptz models [48,49]). Only recently have metrics become available to also quantify rates of reproductive senescence across species [35,50]. Unlike survivorship, reproductive schedules can be highly variable, including multiple peaks. This greater complexity in reproduction has made extensive cross-species comparisons more challenging for reproductive senescence.

As reviewed by Lemaître & Gaillard [51], quantifying reproductive senescence is complicated by the multitude of different components that can comprise the reproductive schedule. When broken down into these components, from egg production to juvenile survival and everything in between, heterogeneous rates of reproductive senescence can be revealed. In the grey-leaved cistus (*Cistus albidus*), for example, older plants produce fewer flowers but show no evidence of a decline in germination capacity [52]. In meerkats (*Suricata suricatta*), litter size displays a concave quadratic relationship with maternal age, but litter survival is independent of maternal age [53]. Parental effect senescence, where offspring age-specific vital rates are affected by the age of their parents

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[54], is also rife but variable across species [55]. Accounting for these heterogenous rates and trade-offs between these different components of the reproductive schedule will be of paramount importance for future studies of reproductive senescence [51].

Adding further complexity to quantifying reproductive senescence is the variety of shapes age-specific reproductive trajectories can take. Across birds and mammals, increases in a component of reproduction followed by decline from a peak are common [56–58], but indefinite increases with age can also occur across taxa [24] (electronic supplementary material, figure S3). In plants, vegetative dormancy [59] and seed banks [60] add remarkable extra layers of complexity [61,62]. Baudisch & Stott's [35] pace and shape metrics of fertility offer an attempt to condense this complexity into two metrics using the insight of cumulative reproduction. Our results here show that age trajectories of reproduction are often uncoupled from the pattern of actuarial senescence (figure 2; electronic supplementary material, figure S3). Species-specific studies that investigate both actuarial and reproductive senescence are becoming more frequent, and both uncoupling [63] and concurrent [64] patterns of actuarial and reproductive senescence appear common.

Overall, the prediction from Hamilton's model for actuarial senescence is clear: senescence should occur in any age-structured population, and start at the age of first reproduction [12]. Equation (3.1) tells us that the force of selection for reproduction is proportional to the abundance of mothers at a given age [14]. With efflux of the oldest due to mortality and influx of the youngest due to births, overall stable age distributions are likely to often be biased towards younger age classes [14]. Yet, there is no theoretical expectation that the stable age distribution will be centered at the age of maturity. Future theoretical work should seek to pinpoint how ecological and demographic forces will shape the stable age distribution, allowing for clearer hypothesized comparative tests for the onset, rate and/or escape from reproductive senescence. Links can then be made to how reproductive and survivorship trajectories should co-evolve when constrained by physiological trade-offs [65,66], unveiling mechanisms behind the variation in synchrony of actuarial and reproductive senescence across species. Only by studying age-patterns of reproduction and mortality in tandem, can we fully understand how different ecological and demographic mechanisms have promoted different patterns of senescence.

(b) Stage structure and growth forms

Not all species' mortality and reproduction patterns are best predicted by age. Caswell & Salguero-Gómez [67] showed how within a stage, age-specific selection gradients can actually increase with age, a pattern that might commonly be found in plants [32]. Spanning across taxa, many stage-categorized species have the capacity to grow indefinitely [26,68]. Extremely common in plants [69], indeterminate growth is also found in insects [70], fish [71], reptiles [72] and corals [73]. A pioneering study by Vaupel *et al.* [26] hypothesized that such indeterminate growth can favour negative senescence. The authors modelled an organism whose reproductive capacity increases with size. For such an organism, it can pay to sacrifice current reproductive output if such a sacrifice markedly increases size, and, therefore, potential future reproductive output. An increase in size over time implicity carries with it an increase in age. Negative senescence, a decrease in mortality rate from the age at reproduction and/or increasing reproduction with age, may, therefore, be observed.

In our display of currently available demographic data, 98% of studied plant species and all of our studied corals show little evidence of an increase in risk of mortality with age (e.g. *Paramuricea clavata*; figure 1; electronic supplementary material, tables S1 and S2). We also found evidence for negative actuarial senescence in the South American river turtle (*Podocnemis expansa;* figure 1), an indeterminately growing reptile. Vaupel and colleagues suggested that negligible and negative patterns of senescence may mostly be characteristic of species that attain sizes at reproductive maturity that are not their maximum size, and gain significant reproductive capacity as they grow. Some indeterminately growing species do still display senescence [74], and perhaps the current treatment of growth form as a discrete variable (determinate versus indeterminate) offers limited scope for predictions [28,75]. Transforming growth form into a continuous variable, for example, as size at maturity as a proportion of maximum size, will allow for more quantitative comparative tests on the effects of growth form on senescence.

The concept of negative senescence can be generalized as a resource allocation decision based on a trade-off between current and future reproduction within a life course [76]. Often, this trade-off favours the former [1–4], but Vaupel *et al.*'s insight is that growth can provide a mechanism shifting the optimal balance of the trade-off in the favour of future reproduction. If reproduction is much lower at younger, mature ages (lower sizes) and increases disproportionately with size and age, then the relative reproductive value of older ages classes and the abundance of mothers will be biased towards older age classes. Growth and increasing reproductive capacity with age provide just one mechanism to alter a population's reproductive value and stable age distributions in favour of delaying senescence.

(c) Social interactions

Hamilton's model assumed an infinite population in which individuals do not interact. A recent themed issue in *Philosophical Transactions B* 'Ageing and sociality: why, when and how does sociality change ageing patterns?' [77] is testament to a growing interest that such interactions between individuals could alter the evolution of senescence [78]. Given the strong relationship between the social environment and mortality risk in humans [79], it is perhaps not surprising that researchers have now begun to focus their attention on trying to test for similar phenomena in other social animals.

Sociality appears to be correlated to some extent with longer lifespan [58,80,81]. Extreme sociality is associated with a 100-fold increase in lifespan in social insects [80]; cooperative breeding birds live longer on average than non-cooperative birds [81] and a recent comparative review of reproductive senescence in birds and mammals reveals a relationship between proxies of sociality and a slower pace of life [58]. However, current evidence suggests that this relationship is likely to be due to longer lifespan first being driven by a reduced threat of extrinsic mortality [81]. Lower individual turnover and overlapping generations then facilitate more opportunities for individuals to interact, and can favour the evolution of cooperation [82,83]. Quantitative theoretical predictions are lacking as to how, or even if, more complex sociality can causally alter selection on age-specific mortality and reproduction, and, therefore, senescence.

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Some theoretical treatments for senescence in a social context do exist, with the most influential being that of Lee [84]. Lee extended Hamilton's models to include a 'transfer effect', whereby the author showed how selection gradients on mortality and reproduction can also depend on resources given and received throughout the life course of the individual. Most other models have focused specifically on the interaction between limited dispersal and senescence. These studies have shown that actuarial senescence may favour reduced juvenile dispersal with increasing maternal age [85], that reduced dispersal may favour the evolution of shorter lifespans [86], and that in some scenarios, actuarial senescence can actually be favoured in viscous populations [87,88]. In general, however, a comprehensive ageing theory of social animals is lacking [77].

Cooperative breeding systems, defined by the presence of alloparental care by helpers [89], offer an excellent system for empirical and theoretical study for how social interactions could directly alter patterns of senescence. For example, helpers can provide load-lightening benefits to breeders [80] that may delay breeder senescence and extend lifespan [90–92]. Helping tendencies may also change with age [93], due, for example, to changing relatedness to its group as an individual helper ages [94], or to changing benefits of independent reproduction [95]. Age trajectories of how individuals could affect both helper and breeder senescence profiles, and lead to plastic senescence patterns within a species.

The classification of helpers and breeders, or subordinates and dominants, means that cooperative breeding systems can be modelled as stage-structured populations. Rank or stage may be quantified by the proportional access of total group reproduction an individual gains, which will be determined by the level of reproductive skew within the group [96]. In more despotic societies, such as the naked mole-rat [97] (*Heterocephalus glaber*), the breeding pair of individuals control all reproduction, whereas in plural breeding societies such as Guira cuckoos [98] (*Guira guira*), all individuals have access to reproductive opportunities. This stage-related access to reproduction will bias the reproductive value distribution towards certain classes of individuals, which if they correlate with age, will alter late-life selection.

Furthermore, groups can be composed of kin, non-kin or a mix, thus also potentially creating indirect fitness trajectories of survival or reproduction [99]. The formation of these different types of groups via dispersal patterns will drive the competitive dynamics within groups [100], and how the relatedness of an individual to their group changes with age, which can have extreme consequences such as favouring complete reproductive suppression [94]. The interactions of all of these features of cooperative breeding systems (e.g. helping, stage structure, and compeititve and cooperative interactions) may lead to novel predictions for the evolution of senescence when individuals interact with one another.

4. Extending the classical evolutionary framework of senescence

The decline in the force of natural selection with age provides an ultimate explanation for the counterintuitive prevalance of senescence in nature [3]. We have reviewed recent—and provided new—reasons why this decline in the force of natural selection may be postponed, or even reversed during adulthood. Although the forces of selection may always ultimately decline in the extremes of any species's lifespan [26], the variable patterns of ageing from maturity for both survival and reproduction (figures 1 and 2; electronic supplementary material, figure S3) observed in nature [24] deserve explanation [28,29].

We have found it conceptually useful to consider lifespan and senescence as orthogonal characterisitcs of a species, using a pace-shape framework [34]. Indeed, there is now strong evidence that senescence rates are independent of a species's lifespan [24,101]. Discussions often conflate the two, which can be confusing for predictions and prevent further progress in the field. For example, a delayed age of maturity will be associated with longer lifespan and postponed onset of senescence, but should not *a priori* alter the rate of senescence. Likewise, an age (and condition [23]) independent reduced threat of extrinsic mortality should select for longer lifespan but not, all else being equal, make any difference to the pattern of senescence [22]. A consensus on a definition of senescence as the change in mortality and reproduction with age independent of lifespan, we argue, is the first important, albeit trivial, step towards extending the framework.

The identification of different mechanisms that might be associated with variable ageing patterns has relied upon research of previously underexplored taxa. High-resolution demographic data on some major groups of species are still yet limited [102]. We hope that our work contributes to stimulating the next generation of demographers to explore these under-represented corners of biodiversity. There are also other potential mechanisms that may shape senescence outcomes not explored here, such as how sexual selection interacts with ageing rates [103]. It may be easy for one to then imagine any number of mechanisms that could alter ageing patterns, so how can we provide a rigorous framework for accepting or declining these mechanisms?

We show in our review of theory and analyses (figure 2*b*,*d*; electronic supplementary material, figure S3) that reproductive value and population structure are key to understanding senescence. The two-question process outlined in §3 can offer a stepping stone framework for evolutionary research of senescence. Senescence can be viewed as a result of early versus late-life trade-offs [104]. Stage structure, whether by size, social hierarchies or division of labour [105], provide mechanisms to weight this trade-off in favour of late life by shifting the population's distributions of reproductive value and class frequencies to reduce the decline of selection in later age classes. Ultimately, as Hamilton displayed [3], and Caswell made explicit [15], it is these two fundamental properties of a population that determine the strength of selection, which in turn determines the ease at which late-acting detrimental alleles may be able to invade. We must first ask how a mechanism can shift these distributions, which will then allow us understand which patterns of senescence will evolve. Hamilton's work provided fundamental insights for the evolution of senescence. Now that we have access to an unparalleled amount of data from a more diverse range of species, it is our duty as demographers, empiricists and theoreticians, to extend the framework and once and for all solve the problem of the evolution of senescence.

Data accessibility. Data are available from the COMPADRE Plant Matrix Database and COMADRE Animal Matrix Database (www.compadre-db.com). Code used for analysis is available in the electronic supplementary material.

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The data are provided in electronic supplementary material [106].

Authors' contributions. M.R. and R.S.-G. conceived the review project. M.R. and P.C. conducted the analyses with input from R.S.-G. and produced all visualizations. M.R. drafted the first version and, together with P.C. and R.S.-G., revised and edited the manuscript.

All authors gave final approval for publication and agreed to be held accountable for the work performed therein.

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Supplementary Information

Senescence: why and where selection gradients might not decline with age

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This file includes:

Methods
Figures S1 to S2
Table S3
References for Supplementary Information

Note – Figure S3 and Tables S1-S2 are moved to the Appendix due to space limitations.

Methods

Data

We used the COMADRE Animal Matrix Database (v. 3.0.0) (Salguero-Gómez *et al.* 2016) and COMPADRE Plant Matrix Database (v. 5.0.0) (Salguero-Gómez *et al.* 2015) to obtain age trajectories of survival and reproduction. These open-access data repositories consist of a collection matrix population models (MPMs) (Caswell 2001) incorporating high-resolution demographic information on the survival and reproduction patterns of over 1,000 animal and plant species worldwide and associated metadata (Salguero-Gómez *et al.* 2015; Salguero-Gómez *et al.* 2016). Both databases include information on species for which the data have been digitised and thoroughly error-checked. We imposed a series of selection criteria to restrict our analyses to data of the highest quality possible:

(i) MPMs were parameterised with field data from non-disturbed, unmanipulated populations (*i.e.* natural populations) to best describe the species' age trajectories.

(ii) MPMs had dimension ≥3 × 3 (i.e. rows × columns). Generally, low dimensions MPMs lack quality for the estimation of life history traits (Salguero-Gómez, R. & Plotkin 2010). This selection criterion also helps avoid problems with quick convergence to stationary equilibrium, at which point the estimates of life history trait values and rates of senescence become unreliable (Jones *et al.* 2014; Horvitz & Tuljapurkar 2009).

(iii) MPMs were only used when the entire life cycle was explicitly modelled including recordings of survival, development, and reproduction for all life cycle stages.

(iv) When multiple studies existed for the same species, we considered only the study of greater duration to ensure the highest temporal variation in the population dynamics was captured.

(v) Studies of annual species modelled using seasonal projection matrices were not included; we chose only species using an annual time step. This is due to the difficulties of converting their population dynamics to an annual basis to compare with all other species' models.

(vi) Included MPMs have stage-specific survival values ≤ 1. In a small number of published models, the stage-specific survival values can exceed 1 due to clonality being hidden in the matrix, rounding errors, or other mistakes in the original model (Salguero-Gómez *et al.* 2015; Salguero-Gómez *et al.* 2016).

(vii) MPMs were from species of which phylogenetic data was available, to ensure we were able to account for phylogenetic relatedness.

The result of these criteria was a subset of 475 species of animals and plants from the initial databases, which we used for our analysis. Of these, 80 were animals, with 15 invertebrates and 65 vertebrates. The remaining 395 species were plants, with 25 gymnosperms and 370 angiosperms We provide a list of all the species used, their categorisation of senescence including a value of *S*, and their relevant source study in the supplementary information (Table S1).

Quantifying actuarial senescence

MPMs are a summary of the population dynamics of a given species, from which we can calculate several life history traits. To do so, we first must decompose an MPM (***A)*** into its sub-components (Caswell 2001):

U **–** containing the stage-specific survival rates

F – containing the stage-specific per-capita reproduction rates

C – containing stage-specific per-capita clonality rates

$$\boldsymbol{A} = \boldsymbol{U} + \boldsymbol{F} + \boldsymbol{C}$$

equation 1

This decomposition facilitates the estimation of key life history traits, including a rate of senescence (*S*) (Baudisch & Stott 2019). Calculating *S* requires first obtaining the age-specific survivorship curve $l(x)$ from $\boldsymbol{U}$. To obtain $l(x)$ we first have to define age, and the definition of age requires a choice of a stage that corresponds to "birth". Following Jones *et al.* (2014), we defined the stage corresponding to birth as the first established non-propagule stage (e.g., not seeds or seed bank in the case of plants, nor larvae or propagules in animals) due to the estimate uncertainty of parameters involved in those stages. The calculation of $l(x)$ was then implemented according to Caswell (p. 118-21) (2001).

$$l(x) = e^t U^x e^j \qquad x = 0, 1, \ldots,.$$

equation 2

Where *e* is a vector of ones, and we start with a single individual in the stage *j* defined to correspond to birth.

We considered survivorship trajectories beginning at the age of maturity (α - calculated following subsections 5.47–5.54 in Caswell (2001)) and ending at the age at which 5% survivorship from maturity occurs (ω). This is because a cohort modelled by iteration of the ***U*** matrix eventually decays exponentially at a rate given by the dominant eigenvalue of ***U***, and converges to a quasi-stationary distribution given by the corresponding right eigenvector **w**. Once this convergence has happened, mortality remains constant with age, and so to prevent our conclusions being overly influenced by this assumption, we calculated the age at which the cohort had converged to within a specified percentage (5%) of the quasi-stationary distribution (Jones *et al.* 2014, Horvitz & Tuljapurkar 2009).

Following Baudisch & Stott (2019), the function *H(x)* defines the cumulative hazard of mortality up to age x as

$$H(x) = \int_{\alpha}^{x} \mu(t)dt$$

equation 3

Where $\mu(x)$ denotes the age-specific mortality function capturing the average hazard of death of an individual at age *x*, and *H(x)* corresponds to the logrartihmic transformation of the survivorship trajectory (*H(x) = -lnl(x)*). *S* is quantified as the difference in areas under the age-specific survivorship curves of a standardised survivorship curve that assumes constant mortality, and therefore has a value of 0.5, and the survivorship curve in question:

$$S = 0.5 - \int_{\alpha}^{\omega} H(x)$$

equation 4

Theoretically, the maximum and minimum values of the second term in equation 4 are 1 and 0 respectively. The value of S is therefore bound between -0.5 and 0.5. If most mortality occurs later in life, $S > 0$, the population is displaying actuarial senescence. On the contrary, if $S < 0$, the risk of mortality declines with age and the population escapes actuarial senescence. Values of $S \sim 0$ indicate negligible senescence, where risk of mortality remains relatively constant with age. We determined a bound around zero to infer which values of *S* should be considered as negative, negligible, or positive senescence respectively for the species in our dataset. By using bounds around negligible senescence, we aim to not exaggerate reports of both negative and positive senescence. Although somewhat arbitrary, we argue that it is also non-sensical to describe everything above some value (0 here) as positive senescence, and everything below such value as negative senescence, with negligible senescence constrained to the single value. In an ideal world, when more demographic data are collected on numerous populations for a given species, we hope these bounds will be replaced by an approprite statistical metric. For both animals and plants separately, we assumed that the root mean squared difference between a species' value of *S* and zero is less than or equal to some value, ε, such that:

$$\sqrt{\frac{\sum(S(i)-0)^2}{n}} \leq \varepsilon$$

Where *S(i)* is the value of *s* for species *i*, and *n* is the total number of species in our dataset which are animals (80) or plants (395), respectively. We quantified bounds of $-0.109 \leq S \leq 0.109$ for animals and $-0.129 \leq S \leq 0.129$ for plants. For each taxonomic kingdom, values of *S* that fall within the bound are considered not different from zero and therefore categorised as negligible senescence. The inequality assumes no statistical distribution of the values of *S* across species.

The metric of actuarial senescence, *S* (Baudisch & Stott 2019), measures the spread of mortality throughout the life course of a cohort, but does not distinguish between extrinsic and intrinsic causes. If, however, mortality is biased towards the latter life stages ($S > 0$), *i.e.* older age, then this is indicative of older ages classes succumbing to mortality at a greater rate relative to their younger counterparts. Whether the ultimate cause of death is internal or external is irrelevant, *S* merely describes which age classes are more vulnerable to such mortality. If older ages classes are more vulnerable to mortality then this is indicative of demographic actuarial senescence.

Phylogenetic analyses for actuarial senescence

After quantifying each species' rate of actuarial senescence, we accounted for the phylogenetic relatedness of the species studied to determine the influence of a species' evolutionary history on its value of *S*. To explore the effects of phylogenetic relationships between the species included in this study, we obtained animal and plant species-phylogenies from different sources. The plant phylogeny was obtained using the *V.PhyloMaker* R package (Jin and Qian 2019). *V.PhyloMaker* allows one to build a rooted

and time-calibrated phylogeny using a species list, based on already built plant phylogenies (Smith & Brown 2018; Zanne *et al.* 2014). The animal phylogeny was produced using the *datelife* R package (Sánchez-Reyes & O'Meara 2019), a service that uses publically accessible phylogenetic source data to build a chronogram – rooted and time-calibrated tree - given an input phylogeny that we sourced from the Open Tree of Life (Hinchliff *et al.* 2015). In some cases, for both plant and animal phyologenies, we detected polytomies (*i.e.* >2 species with the same ancestor), which can interfere in our phylogenetic signal analyses (see Revell 2012). Polytomies were resolved using the function "multi2di" from *ape* package (Paradis, Claude & Strimmer 2004), which transforms polytomies into a series of random dichotomies with one or several branches of length zero. Trees were visualised using the *ggtree* R package (Yu *et al.* 2017).

To evaluate the role of phylogenetic relatedness in determining the patterns of variation of actuarial senescence we estimated Pagel's λ (Pagel 1999). This metric is an index bounded between zero and one, where values ~0 indicate that the evolutionary history of the species explains little about the variation of the trait measured, and values ~1 suggest that that evolutionary history mostly explains the observed variation of the trait across the studied species. To estimate Pagel's λ we used the R package *phytools* (Revell 2012). A full summary of the phylogenetic signals obtained for each of the four monophyletic groups can be found in the Supplementary Information (Table S3).

Reproduction analysis

We calculated reproductive age-trajectories for the species in our analysis to investigate whether reproductive trajectories matched patterns of actuarial senescence. Age-specific reproduction (*m(x)*) was calculated following Caswell (p. 118-21) (2001). Briefly, the proportional structure of the cohort at age *x* is given by:

$$\mathbf{p}(x) = \frac{U^x e_j}{e^T U^x e_j} \quad x = 0,1,\ldots.$$

equation 5

The total sexual reproductive output per individual at age *x* is given by:

$$m(x) = \mathbf{e^T F p}(x)$$

equation 6

For the remaining 463 species that are not displayed in Figure 2, the *l(x)* and *m(x)* trajectories are found in the Supplementary information (Fig.S3).

Reproductive value and stable (st)age distributions

We calculated reproductive value and stable (st)age distributions for the populations in our study to visualise how strong the strength of (st)age-specific selection should be against senescence, under assumptions of stable (st)age distributions and exponential growth (Hamilton 1966; Moorad *et al.* 2019). For each population, we quantified reproductive value and the stable (st)age proportions of each (st)age using the 'eigs' function in the popdemo (Stott *et al.* 2012) package in R. Stable (st)age distributions sum to 1.

Fig. S1. Phylogenetic clustering of actuarial senescence across animals. Values of *S*, a metric of actuarial senescence, across 80 animals are generally not dictated by phylogenetic relatedness, with estimated insignificant values of Pagel's λ (Table S3). Values of *S* range from -0.222 (high negative actuarial senescence) to 0.332 (high positive actuarial senescence).

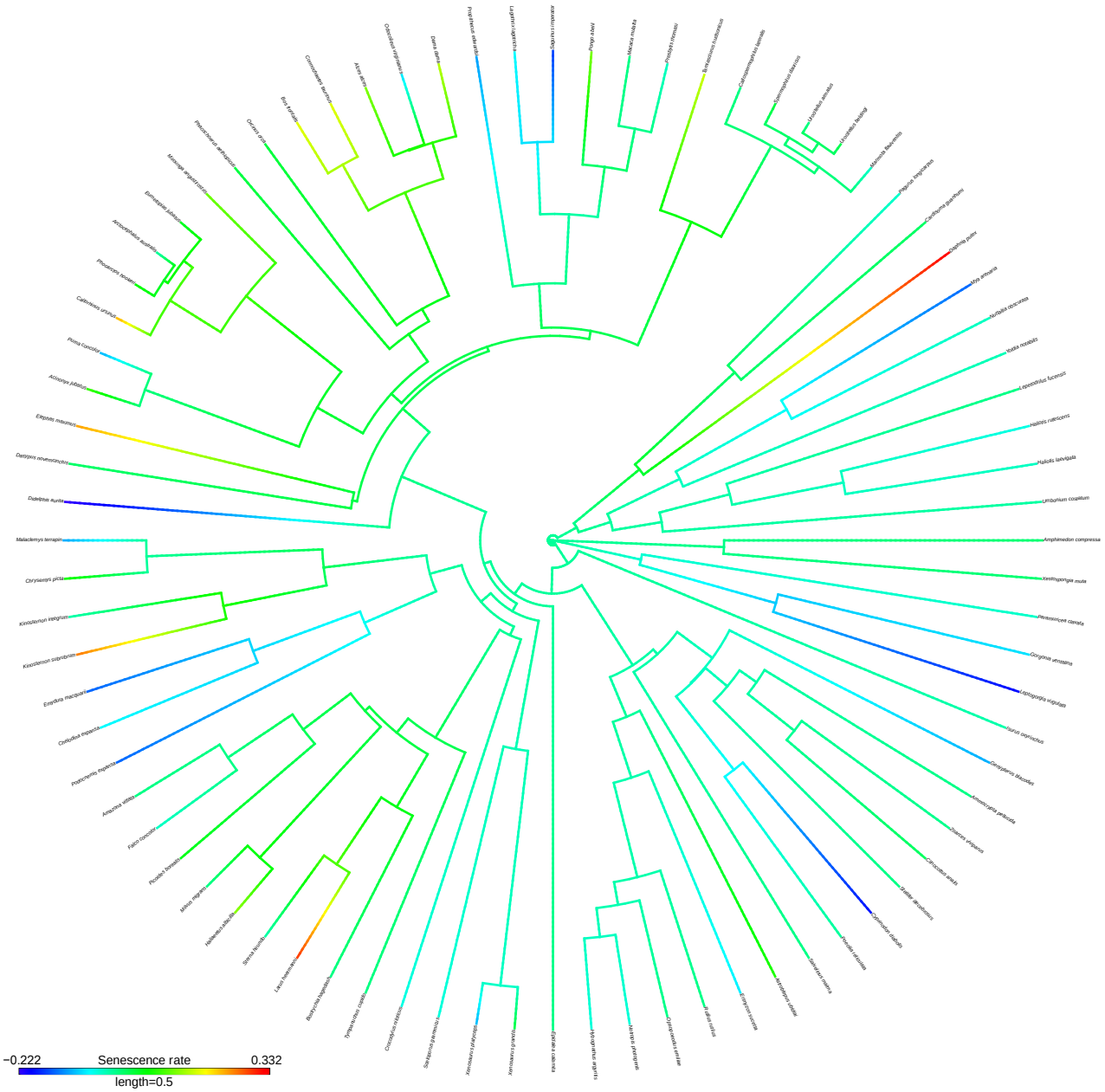


Fig. S2. Phylogenetic clustering of actuarial senescence across plants. Values of *S*, a metric of actuarial senescence across 395 plants are fairly well predicted by phylogenetic relatedness (Table S3). Values of *S* range from -0.445 (high negative actuarial senescence) to 0.309 (high positive actuarial senescence).

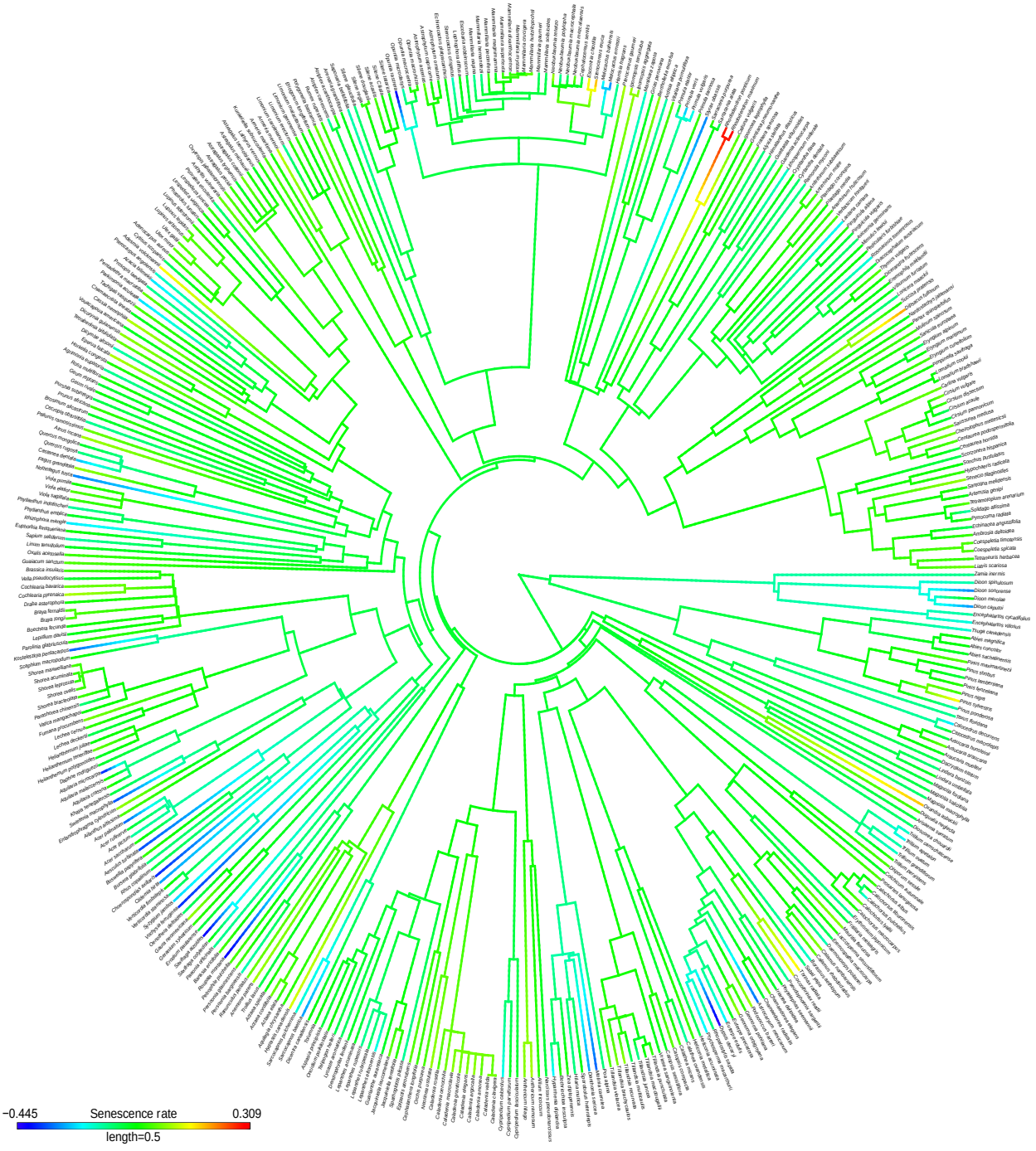


Table S3. Phylogenetic signals of actuarial senescence for each major taxonomic group of studied. Estimates of Pagel's λ and their respective p values within the phylogenetic analysis of actuarial senescence across our study species. Pagel's λ is an index bounded between 0 and 1, where values ~0 indicate that the evolutionary history of the species explains little about the variation of the trait measured, and values ~1 suggests that that evolutionary history mostly explains the observed variation of the trait across the studied species. Values of $p < 0.05$ indicate that Pagel's λ is significantly different from 0. Values of Pagel's λ can be > 1 in the case of small sample sizes (17).

Taxonomic breadth	**Pagel's λ**	**p**
Plants	0.31	0.00
Animals	0.22	0.18
Angiosperms	0.27	0.00
Gymnosperms	0.27	0.09
Vertebrates	0.17	0.33
Invertebrates	1.63	0.13

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3 | Covariation between metrics of aging systematically differs across animals and plants

Covariation between metrics of ageing differs systematically among animals and plants

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Abstract

How long species live, for how long they reproduce, how their risk of mortality is distributed throughout their life course, and how their rate of reproduction is distributed across their life course collectively determine their fitness. These four components of a species' life history have been modulated in numerous ways over evolutionary time across taxa, resulting in the tremendous diversity of life histories we see today. Yet, species face trade-offs between these four life history traits – no species can simultaneously maximise them all. The trade-offs species face within their lifetimes due to constraints imposed by resource availability, environmental limitations, phylogenetic history or organismal forms and functions are likely to have driven covariation across taxa between these pillars of fitness. Yet, the extent of this hypothesised covariation is unknown, as is the extent to which it systematically differs across taxa. Here, we use high resolution demographic data on 106 animal and 232 plant species with age-specific estimates of mortality and reproduction to quantify each species' pace of ageing (*i.e.*, how long they are expected to survive or reproduce) and shape of ageing (*i.e.*, the distributions of mortality risk or reproduction from maturity to death). After accounting for body mass, we reveal residual covariation between ageing metrics that differ in magnitude and direction across major animal clades, but little role of shared ancestry in shaping covariation between metrics. Birds with longer reproductive lifespans tend to show less-senescent survival curves, and in both birds and mammals, senescence in mortality and senescence in reproduction tend to be associated with one another. Conversely in plants, covariation is predominantly shaped along shared ancestry, with lifeform and ecoregion accounting for negligible estimates of residual covariation between ageing metrics. Our results indicate that covariation between ageing metrics is common across taxa but differs in magnitude and direction across major groups of animals and plants.

Main

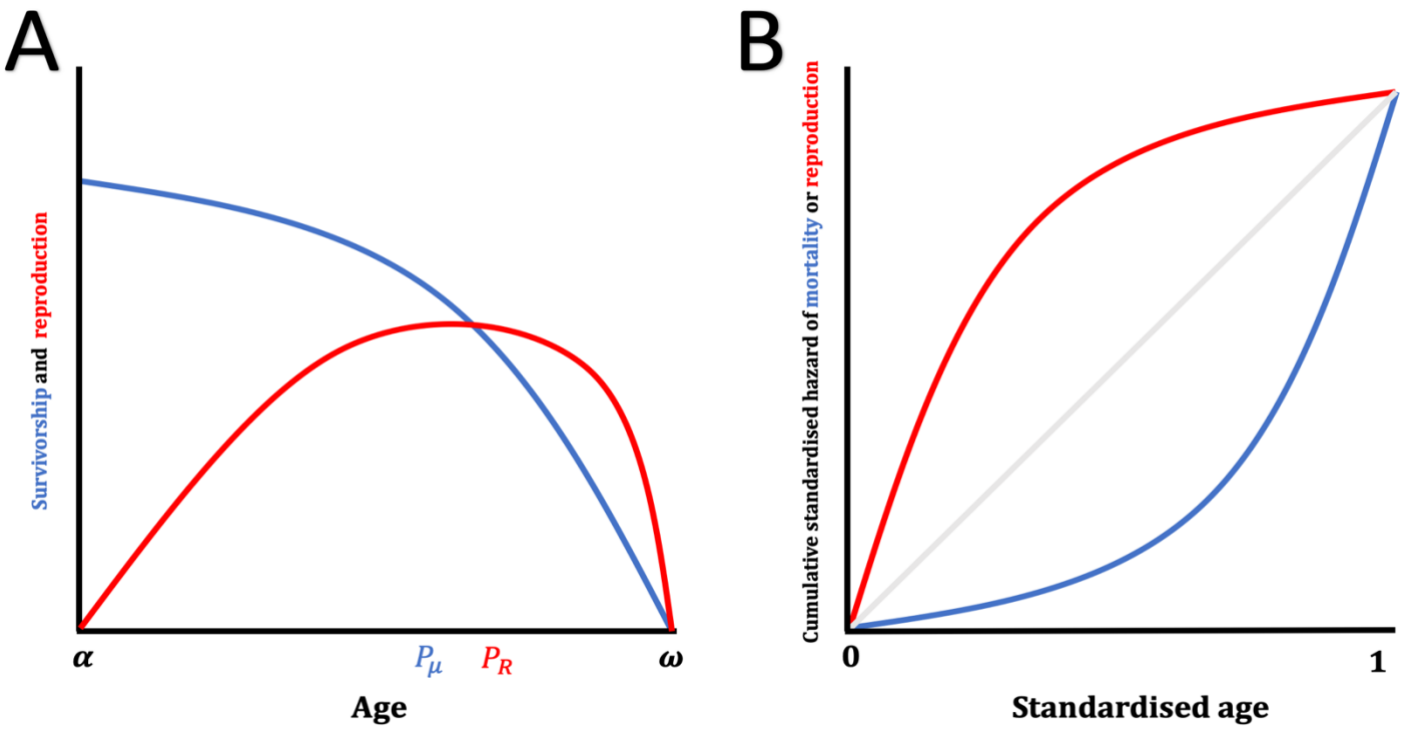
All species face a common conundrum: how to allocate limited resources between survival and reproduction to maximise fitness[1,2]. Under the pressures of environmental resource limitations and evolutionary constraints, natural selection has innovated a huge diversity of strategies to overcome this problem, the understanding of which is the focus of life history theory[2,3]. Pacific salmon (*Oncorhynchus tshawystcha*) produce thousands of small eggs at once[4], whereas Kiwis (*Apteryx spp*) produce a single egg at a time up to a quarter of their own size[5]. Aphids are born reproductively mature[6], whereas some plant species set seeds that may remain viable yet not germinate, below-ground, for over hundreds of years[7]. The Greenland shark (*Somniosus microcephalus*) only becomes sexually mature after decades[8]. Many species display increasing frailty with age (*i.e.*, senescence)[9,10], while others do not[11], and many display asynchrony[11] (**Chapter 2)** between how their rates of mortality (actuarial senescence) and reproduction (reproductive senescence) change with age. This diversity of life history strategies reflects modifications in the same demographic currencies (survival, development, and reproduction) under varied environmental, physiological, and phylogenetic constraints[2]. However, the extent to which these constraints dominate patterns of covariation in ageing remains unknown. For instance, are longer-lived species more likely to have mortality and reproduction distributions skewed to older ages? Are species that display actuarial senescence also more likely to display reproductive senescence? To what extent do these patterns of covariation differ across taxa?

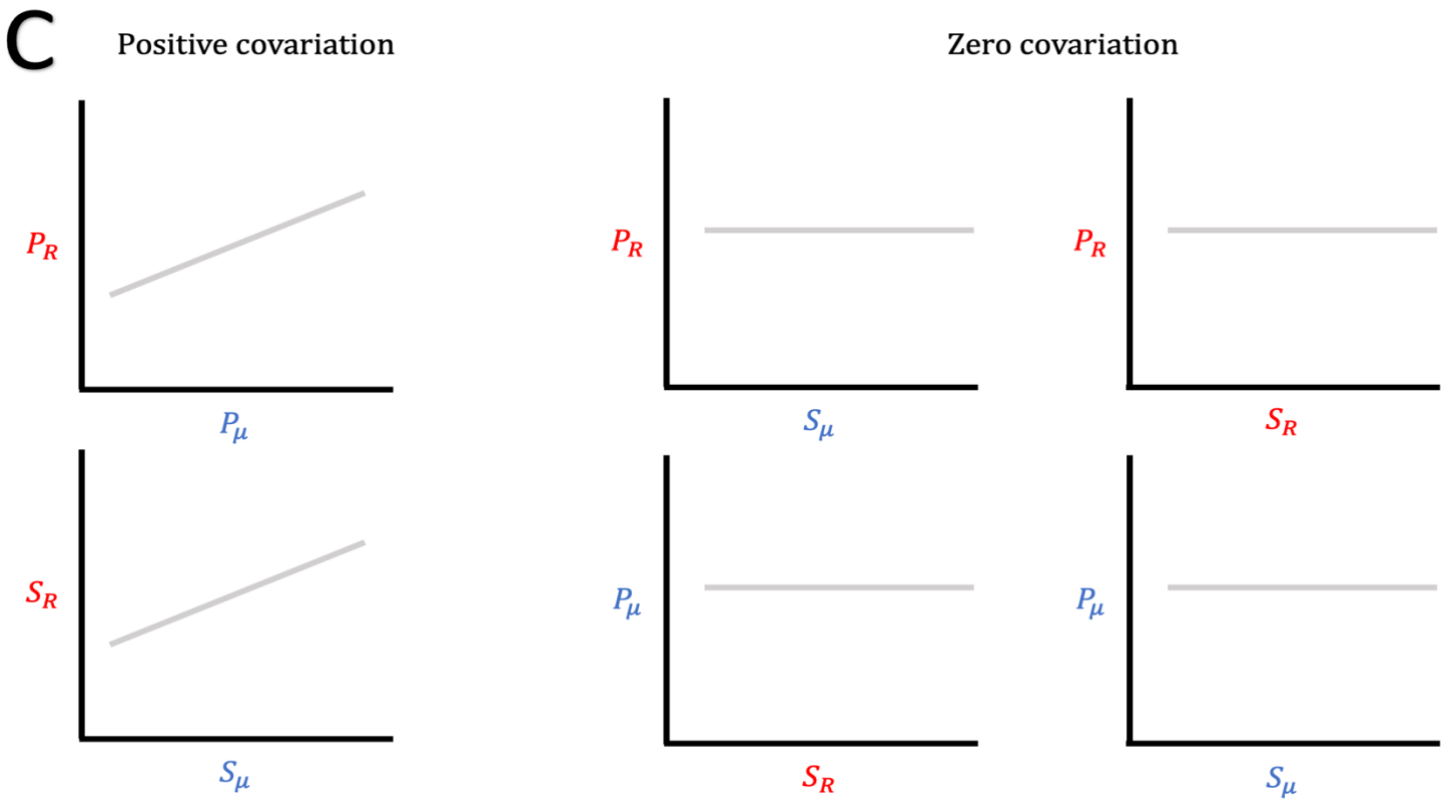
To answer these questions, we utilise the pace and shape perspective of ageing[12,13,14] (Fig.1). 'Pace' is a quantity calculated from actuarial or reproductive schedules, measured in units of time. Commonly, the pace of actuarial ageing (P_μ) measures expected lifespan from reproductive maturity, and the pace of reproductive ageing measures the average age that a mother gives birth (P_R) (Fig. 1A). Species that live and reproduce over the span of days would

therefore have a lower value of pace than species that live and reproduce for years. 'Shape' is a dimensionless measure, standardised by lifespan, that describes the age-specific distribution of mortality (S_μ) or reproduction (S_R) across the life course[13,14] (Fig. 1B). Positive shape describes senescence (*i.e.*, characterised by relatively higher mortality risk and lower reproductive output towards later life) whilst negative shape describes negative senescence (*i.e.*, characterised by relatively low mortality risk and higher reproductive output towards later life). For a given longevity and/or age at maturity, pace and shape are linked: S_μ and S_R describing senescent life histories will have higher P_μ and lower P_R compared to negatively-senescent life histories. Despite this link, for a given longevity and/or age at maturity, pace and shape are mathematically orthogonal. As longevity and age at maturity may freely vary across species[13,14] (see Methods), species may theoretically show any combination of pace and shape across the four measures.

The pace and shape metrics of ageing are orthogonal, and so the theoretical null expectation is zero covariation between pace and shape metrics of actuarial and reproductive ageing. Despite this expectation, life-history theory predicts the emergence of covariation in demographic processes over evolutionary time. Indeed, substantial evidence exists regarding the fast-slow continuum[15,16], which ranks species according to the speed of their life history. In this continuum, 'fast' species allocate more resources to rapid development and early reproduction, resulting in shorter lifespans. On the other hand, 'slow' species allocate more resources to maintenance (survival), thus growing slower, reproducing later and producing fewer offspring over their lifetimes. Although P_μ and P_R are calculated as independent metrics, we expect strong positive covariation between P_μ and P_R, since a longer expected lifespan should drive a greater average age of a mother, all else being equal (Fig. 1C).

Figure 1 | Visualisation of the pace and shape of ageing metrics and our predicted levels of covariation. A) The pace of mortality (P_μ) is quantified from the logarithmic transformation of the survivorship curve (blue; see Methods) and describes the average expected adult lifespan, where the adult lifespan spans from the age of maturity (α) to the final reproductive age class (β). The pace of reproduction (P_R) represents the age of an average mother, independent of survival. **B)** Shapes of mortality (S_μ) and reproduction (S_R) are calculated from the cumulative standardised hazard of mortality or rate of reproduction, over standardised age. The area under the curve is weighted against the 'benchmark case' of constant mortality or reproduction (grey line) to give the value of shape (See Methods). When reproduction is biased towards older age classes, this represents negative reproductive senescence. When mortality is biased towards older ages classes, this represents positive actuarial senescence. **C)** We predicted positive residual covariation between P_R and P_μ, and S_R and S_μ, and zero covariation between all other ageing metrics (see Main text).


A
Survivorship and reproduction
α
P_μ
P_R
ω
Age
B
Cumulative standardised hazard of mortality or reproduction
0
1
Standardised age


C
Positive covariation
Zero covariation
P_R
P_μ
P_R
S_μ
P_R
S_R
S_R
S_μ
P_μ
S_R
P_μ
S_μ

Classical evolutionary theories of senescence suggest that a declining efficacy of natural selection with increasing age favours trading-off late-life performance for early life reproductive success[17,18,19,20]. A previous comparative study across mammals and birds revealed a positive covariation between actuarial senescence and reproductive senescence[21], suggested synchrony between trade-offs of survival and reproduction across the adult life course. However, recent studies have gathered increasing evidence that reproductive and survival related traits do not always change synchronously over age within individuals[11,22] (**Chapter 2**), and the full extent to which both co-vary across different taxonomic groups remains unquantified.

Importantly, temporal correlations between vital rates[23], and in particular an association between the average length of life and its variability (P_μ-S_μ) across human populations and primates has been revealed in recent work[24,25]. However, this relationship seen across human and primate populations weakens as the phylogenetic scope is extended more widely across mammals[26]. When considering a closely related group of species, if lifespan is fairly constrained, then most differences in the pace of ageing between species will likely be constrained to shifts in the distributions of mortality and reproduction (*i.e.* shape). However, as the phylogenetic scope widens, and lifespan becomes more labile when considering a greater range of species with distant shared common ancestry, this association may weaken. Given the broad scope of our analyses across distantly related taxa (*e.g,* from gymnosperms to mammals), we retained the theoretical null hypotheses of zero covariation between all four combinations of pace and shape of mortality and reproduction: (1) P_μ-S_μ, (2) P_R-P_R, (3) P_μ-S_R, and (4) S_μ-P_R

Despite this set of null hypotheses, there are distinct known consequences of life history strategies or environment that may facilitate differing rates of covariation between ageing

metrics across different groups of species. For example, body mass correlates with longevity[3], but may have a different effect on rates of covariation between ageing metrics depending on the growth form of the species. For example, greater body mass might bring increased reproductive output in indeterminate growers, but not necessarily determinate growers[27]. In plants, there is some evidence, albeit with sampling issues, that tropical and subtropical plant species seem to live longer than species in arid or temperate regions[28]. We do not consider an ecoregion equivalent for animals given their mobility and, especially in some birds and mammals, long distance migration. Additionally, the Raunkiær life form[29] of a plant species, a classification based on the location of the dormant shoot apical meristems in relation to the ground during season of adverse conditions, may drive variation in pace and shape due to different allocations decisions between growth, maintenance and reproduction[28].

Using the pace and shape of ageing framework and high-resolution structured demographic data for 106 animal and 232 plant species, we test for covariation between the six pairwise combinations of the aforementioned four key metrics of ageing: pace and shape of mortality and reproduction. By *(i)* accounting for phylogeny and adult body mass in animals, and ecoregion and Raunkiær life form in plants, and *(ii)* focusing on populations with high resolution estimates for both mortality and reproduction, we provide a robust framework that captures the results of natural selection's solutions to a variety of environmental and evolutionary pressures across a breadth of taxa. We uncover systematic differences in covariation between ageing metrics across taxa. In animals, residual covariation between ageing metrics differs in magnitude and direction in the four examined animal classes. Conversely in plants, phylogeny has the strongest influence on covariation between ageing metric.

Methods

Data collection

We used the COMADRE[30] v. 4.21.8.0, COMPADRE[31] v.6.22.1.2 and DATLife[32] databases to compile demographic data for 106 animals and 232 plants. The COMADRE and COMPADRE databases host species-specific structured demographic data in the form of Matrix Population Models (MPMs)[33]. MPMs contain age or stage specific rates of survival and reproduction from which age-specific survival and reproduction can be easily derived[34]. We restricted our sample of species to those with MPMs *(i)* parameterised from wild and unmanipulated populations so we had the best chance of uncovering age-specific estimates of mortality and reproduction that had evolved under natural conditions, *(ii)* containing data on both mortality and reproduction so that covariation within the same population could be tested, removing the effect of different environmental conditions on different populations, and *(iii)* meeting necessary requirements to calculate life tables[33,35] (See further details in SOM). An MPM containing both survival (which can be converted to mortality estimates – see Methods) and reproduction data is labelled as $\boldsymbol{A}$, and can be decomposed as:

$$\boldsymbol{A} = \boldsymbol{U} + \boldsymbol{F} \tag{1}$$

where $\boldsymbol{U}$ represents a matrix with survival-dependent changes between (st)ages, and $\boldsymbol{F}$ represents a matrix with reproduction estimates. This decomposition facilitates the automated calculation of life tables across the species from COMADRE[30] and COMPADRE[31] using the Rage R package[34].

Life tables for each population were produced from the MPMs to quantify the probability of survival from birth to age x ($l(x)$) and reproduction at age x ($m(x)$). Each life table was

trimmed to consider only the adult life course, which was defined as beginning at the age of first reproduction and ending at maximum longevity (post-reproductive life stages were not present in any species in our analysis). We therefore designated $l(x) = 1$ at the age of first reproduction. The end of the adult life course depended on the type of MPM. For Leslie matrices[36] (those with nonzero entries only on the sub-diagonal of the $\boldsymbol{U}$ matrix, and non-zero entries in the first row of the $\boldsymbol{F}$ matrix), the end of the adult life course corresponded to the final age class in the matrix. For other types of MPM, with non-zero elements in their bottom right element and/or elsewhere in the matrix defining other stage transitions, we trimmed the adult life course to an age at which 5% of the adult cohort would still be alive (therefore all $l(x) \geq 0.05$). This was to limit the effect of mathematical artifacts that constant estimates of mortality and fertility in a final stage can give rise to, including mortality and reproductive plateaus[37]. We only used one MPM per species in our calculations for simplification with phylogenetic analysis. If estimates of survival and reproduction were available for multiple populations of the same species, we first compared the dimensions of each population's MPM. If the populations had the same MPM dimension (*i.e.* same number of st(age) classes), we calculated a single element-by-element arithmetic mean MPM from these. If one population had a higher MPM dimension than the others this population was chosen to represent the species as it should have higher-resolution survival and reproduction data, and if multiple higher-dimension matrices were available then we used the arithmetic element-by-element mean of these. Data in the DATLife database[32] contains life tables of animal species. We supplemented our 94 animal species data from COMADRE with 12 further species from the DATLife database, which had estimates of both age-specific survivorship (to be converted to a hazard of mortality) and reproduction from the same population. We replaced one species (*Papio cynocephalus*) from COMADRE with data from DATLife due to the higher resolution estimates that age-specific data provides rather than stage.

Quantifying pace and shape metrics

We used age-specific reproduction $m(x)$ and survivorship $l(x)$ from the adult life tables to quantify the pace and shape of actuarial and reproductive ageing for each species (Fig. 1). First, to quantify the pace and shape of actuarial ageing, the function $H(x)$ captures the cumulate hazard of mortality up to age x, and corresponds to the logarithmic transformation of $l(x)$, $H(x) = -\ln(l(x))$:

$$H(x) = \int_{\alpha}^{x} \mu(t)dt \tag{2}$$

where α is equal to the age of first reproduction (maturity). The shape of mortality considers the distribution of mortality across the adult life course, and is given by:

$$S_{\mu} = \frac{1}{\tau H}\left(\frac{\tau H}{2} - \int_{\alpha}^{\omega} H(x)dx\right) \tag{3}$$

Where $\tau = \omega - \alpha$, with ω denoting the maximum age class, and so standardises the measure of shape according to adult lifespan. The term $\frac{\tau H}{2}$ represents the value of shape when age-specific mortality is constant (the 'benchmark case'[13,14]), rendering the value of S_{μ} positive if mortality increases with adult age (actuarial senescence), zero if mortality is constant with adult age and equal to the benchmark cases (negligible actuarial senescence) and negative if mortality decreases with adult age (negative actuarial senescence). Shape values range within $-0.5 < S_{\mu} < 0.5$. Actuarial pace is calculated as the life expectancy from age at maturity.

To quantify the pace and shape of reproductive ageing, the function $B(x)$ captures the cumulative reproduction (total number of births) up to age x as:

$$B(x) = \int_{\alpha}^{x} m(t)dt \tag{4}$$

Then, let β represent the last age of reproduction (in most cases, $\beta = \omega$). The shape of reproductive ageing considers the distribution of reproduction across the adult life course, and is given by:

$$S_R = \frac{1}{\tau B}\left(\int_{\alpha}^{\beta} B(x)dx - \frac{\tau B}{2}\right) \tag{5}$$

where, in this case, $\frac{1}{\tau B}$ provides standardisation of the reproductive lifespan, and $\frac{\tau B}{2}$ provides the 'benchmark case' of a constant rate of reproduction across the adult ages. This means the value of S_R will be positive if reproduction declines with adult age (reproductive senescence), zero if the rate of reproduction is constant with adult age and equal to the benchmark cases (negligible reproductive senescence) and negative if reproduction increases with adult age (negative reproductive senescence). Shape values again fall into the range $-0.5 < S_R < 0.5$. The pace of reproduction can be interpreted as 'birth expectancy': the average age that a mother 'gives birth', taken from the distribution of expected offspring across the entire reproductive life. This metric is impacted both by age and age-specific birth rate, in the same way that life expectancy is impacted by age and age-specific death rate. Pace of reproduction uses S_R to calculate the weighted standardized age of 'birth', which is then scaled by reproductive lifespan to give age-specific 'birth expectancy':

$$P_R = \tau - \left(S_R\tau + \frac{\tau}{2}\right) = \tau(0.5 - S_R) \quad (6)$$

Equation (6) illustrates the point made in the introduction: pace and shape are linked by equation 6, therefore shape S_R determines pace P_R for a given reproductive lifespan τ, but as τ can freely vary, any pace and shape combination is theoretically possible.

Covariates and statistical analyses

Our ultimate goal is to estimate the residual covariation between pairwise combinations of metrics of the pace and shape of actuarial and reproductive ageing, alongside estimating the impact of phylogenetic relationships and key covariates such as proxies to size (body mass in animals, Raunkiær life form in plants), and ecoregion in plants. To do so, we apply multivariate Bayesian generalized linear mixed models (glmms), with one bivariate model (see Fig. 1) for each of the six possible pace-shape combinations, and separate analyses for each of the two Kingdoms represented (animals and plants). Therefore, we develop a total of 12 models.

For animals, we include adult body mass (log-transformed), their taxonomic class (Mammalia: n = 45; Aves: n = 31; Reptilia: n = 19; Actinopterygii: n = 11) and the database (COMADRE vs DATLife) from which the data were collected as fixed effects. The latter allows us to test for potential differences emerging from assumptions made in the modelling species demography on estimates of ageing metrics. We used the Amniote Life History Database[38] and fishbase[39] to collect estimates of adult body mass for each species. Body mass (W) in fish was estimated from length (L) by the relationship $W = aL^b$, where a and b parameter estimates for each species are recorded in fishbase. For each fish species, we used the 'common length' estimate for L if available, and the maximum recorded length if not. We obtained estimates of

adult body mass for all other animals in our dataset (Mammalia, Aves and Reptillia) in the Amniote Life History Database.

For plants, we included the Raunkiær life form[29] (Chamaephyte: n = 27, Epiphyte: n = 15; Geophyte: n = 30; Hemicryptophyte: n = 100; Phanerophytes: n = 60), in which we collapsed megaphanerophytes, mesophanerophytes and nanophanerophytes in to one group for model simplification as a fixed effect. We also included ecoregion, for which we condensed the 13 different ecoregions in COMPADRE for our plant species into five categories (following[27]): (*i)* Temperate (temperate broadleaf and mixed forests; temperate coniferous forests; temperate grasslands, savannas and shrublands): n = 90, *(ii)* Tropical (flooded grasslands and savannas, tropic/subtropical dry broadleaf forests, tropical/subtropical grasslands, savannas and shrublands, tropical/subtropical moist broadleaf forests, and tropical/subtropical coniferous forests): n = 70, *(iii)* Desert/xeric shrublands: n = 23, *(iv)* Mediterranean forests, woodlands and shrubs: n = 36, and *(v)* Tundra, Montane and Boreal Ecoregions: n = 13.

There are six possible combinations between the pace and shape metrics of actuarial and reproductive ageing for which covariation can be estimated, each of which is termed a *'Trait'* in the glmms below. For animals, our linear predictor took the functional form:

$$E(Trait\ 1, Trait\ 2) = f([Adult\ body\ mass: Class + Data\ source]: trait), \quad (7)$$

We allowed for an interaction between adult body mass and the class of the species, due to potentially different impacts of greater size on rates of mortality and reproduction. For plants, our linear predictor took the functional form:

$$E(Trait\ 1, Trait\ 2) = f([Lifeform + Ecoregion]: trait).$$

(8)

In models for both animals and plants, Trait 1 and Trait 2 represent the pace of mortality, pace of reproduction, shape of mortality and shape of reproduction. We assumed gaussian error distributions for all response variables, and performed log transformation to values of pace, and a logit transformation to shape values after adding 0.5 to each data point (*i.e.* adjusting shape values so that $0 > S > 1$) so that the *Trait* values met the assumptions of normality. To account for non-independence in pace and shape values that result from common ancestry, we generated an animal phylogeny (Fig. 2) using the Open Tree of Life[40], and used the plant phylogeny (Fig. 3) available in COMPADRE that is derived using the V.PhyloMaker package[41]. In all models, we included phylogeny as a random effect that estimated a variance-covariance matrix for each trait and their covariance in a bivariate model, assuming a Brownian motion model of evolution.

We fitted Bayesian phylogenetic mixed models using the MCMCglmm package[42]. We ran three model chains for each of the 12 multi-response models for 203000 iterations, with a thinning interval of 100 and a burn-in period of 3,000. For animals, in each pace-shape combination model, we estimated a 2×2 residual variance-covariance (VCV) matrix for each of the four animal classes in the dataset. Likewise, for plants, in each pace-shape combination model, we estimated a residual VCV matrix for each of the five Raunkiær life forms and each of the five ecoregions. Our models therefore took the full format of:

$$(Trait\ 1, Trait\ 2) = f([Adult\ body\ mass: Class + Source\ data]: trait), random$$
$$= \sim us(trait): Species, rcov$$
$$= \sim us(at.level(Class,\ x_1): trait): units + \cdots$$
$$+\ \sim us(at.level(Class,\ x_n): trait): units \quad (9)$$

for animals, where x_n represents each major class (Mammalia, Aves, Reptilia, and Actinopterygii), and

$$(Trait\ 1, Trait\ 2) = f([Lifeform + Ecoregion]: trait),$$
$$random = \sim us(trait): Species, rcov$$
$$= \sim us(at.level(Lifeform,\ x_1): trait): units + \cdots$$
$$+\ \sim us(at.level(Lifeform,\ x_n): trait): units$$
$$+\ \sim us(at.level(Ecoregion,\ y_1): trait): units + \cdots$$
$$+\ \sim us(at.level(Ecoregion,\ y_n): trait): units \quad (10)$$

for plants, where x_n represents each major Raunkiær life form and y_n represents each major Ecoregion.

In both sets of analyses for animals and plants, we used uninformative priors with an inverse-Wishart distribution with variance for each trait, V, set to 0.5, covariance, CV, set to 0, and belief parameter, nu, set to 0.002. For the estimation of the phylogenetic VCV matrix we used a parameter expanded prior (a half-Cauchy distribution with parameters V = 0.5, CV = 0, nu = 1, alpha.mu = 0 for both traits and alpha.V = 100) to improve chain mixing, decrease autocorrelation in the random and residual posterior chains, and improve our effective sample size. We checked for convergence between model chains by confirming that the potential scale reduction factor, the Gelman-Rubin statistic, was below 1.1 for all models. Following the

recommendations of Hadfield[42], we also checked that autocorrelation between successive stored iterations was less than 0.1 in all models. All analyses were carried out in the R Statistical and Programming Environment[43]. For visualisations, estimates of covariation from the outputs of models are displayed as posterior distributions (Fig. 2), whilst raw data values of pace (log transformed) and shape of actuarial and reproductive ageing are displayed around phylogenies[44] (Fig. 3 and 4).

Results

Covariation between pace of mortality and reproduction

As per predictions from the fast-slow continuum[15,16,28,45], we expect a positive residual covariation between the pace of mortality (P_μ) and of reproduction (P_R); species that have a slower pace of survival (*i.e.,* longer mature lifespan) should generally also have a slower pace of reproduction (*i.e.*, have a relatively later average birth-age). Across animals, our results support this hypothesis for mammals (95% CIs: 0.040, 0.280; Fig. 2) and birds (95% CIs: 0.005, 0.310; Fig. 2), but not in reptiles (95% CIs: -0.040, 0.100; Fig. 2) or ray-finned fishes (95% CIs: -0.060, 0.130; Fig. 2), for which covariation is distributed across zero. Although we treat these results with caution due to low sample sizes (especially those for ray-finned fishes), they are suggestive that changes in pace of life across these species can be driven more by shifts in the distribution of reproduction and mortality (*i.e.* shape) than flexibility in lifespan (like mammals and birds). In contrast to animals, where phylogeny was found to be unimportant in structuring model (co)variation (Extended Data Fig. 1), common ancestry was found to be highly important in structuring model covariation between ageing metrics in plants (Fig 2; see **Covariation of ageing metrics in plants correlates with common ancestry**). The estimate of residual covariation being structured by phylogeny was most pronounced for the relationship between P_μ and P_R (Fig. 2).

Covariation between shape of mortality and reproduction

Positive rates of actuarial (S_μ) and reproductive senescence (R_μ) tend to occur more frequently in mammals than in the other examined animal classes (Fig. 2). Indeed, mammals are the species that best match the assumptions behind much of the classical evolutionary theories of senescence: determinate growth[27] and a clear distinction between parent and offspring[46]. These characteristics both facilitate a faster declining force of natural selection with increasing age[17]. We report a trend towards positive covariation between S_μ and S_R in mammals (95% CIs: -0.002, 0.080; Fig. 2) and birds (95% CIs: -0.002, 0.030; Fig. 2), highlighting a common trade-off across the lifespan. Both mammals and birds appear to favour increased performance of both survival and reproduction in younger age classes at the expense of performance in later life. Reptiles, meanwhile, show a distribution of covariation with greater overlap across zero (95% CIs: -0.030, 0.110; Fig. 2), however the vast majority of these species display negligible or negative rates of actuarial and reproductive senescence (S_μ or $S_R < 0$; Fig. 3). In fact, the loggerhead sea turtle (*Caretta caretta*) is the only reptile to display a (relatively weak) signal of reproductive senescence ($S_R = 0.02$). In particular, tortoises, turtles and crocodiles (11/19 of the reptiles here) continue to grow throughout life[47], which, when coupled with increased reproduction capacity with age[27], can limit the weakening of natural selection with age and favour increased performance in later life. Our results support recent work on the pace and shape of ageing in reptiles[48,49], which has showed that, although reptiles display significant variation in rates of senescence, there is a trend towards improving performance with age. Fish, like reptiles, have the capacity to continue to grow throughout life and display increased reproductive output with age[50]. Indeed, across the species of Actinopterygii in our study, all except the Eastern sand darter (*Ammocrypta pellucida*; $S_R = 0.13$) display negligible or negative reproductive senescence. The estimate for covariation between S_μ and S_R in fish, however, spanned across zero (95% CIs: -0.180, 0.150; Fig. 2).

Covariation between pace and shape of ageing metrics

The mean posterior estimate of covariation between P_μ and S_μ in mammals overlaps zero (95% CIs: -0.500, 0.110; Fig. 2). This finding suggests a lack of an association between the average length of life and its variability across the mammal species in our study. For example, the American ground squirrel (*Tamiasciurus hudsonicus*) and Red deer (*Cervus elaphus*) both displayed actuarial senescence despite their pace of life differing by an order of magnitude (Fig. 3). In contrast to mammals, birds exhibited positive covariation between P_μ and S_μ (95% CIs: 0.020, 0.150; Fig. 2), revealing an association between the average length of life and its variability. In our study, the variation in P_μ across mammals (Ω = 10.82) is more than double that in birds (Ω = 5.05), indicating that that S_μ likely plays a bigger role in determining P_μ in birds than mammals, leading to positive covariation between the two metrics. Despite this apparent covariation between average lifespan and its variability in birds, across all major animal clades the estimate of covariation between P_R and S_R overlapped zero (Fig. 2), highlighting a lack of association between the average age of a mother and its variability.

Covariation of ageing metrics in plants is structured by common ancestry

In plants, the phylogenetic component of the glmm accounts for the vast majority of random and residual variance. This key finding indicates that residual covariations between all combinations of metrics of ageing in our 232 examined plant species are predicted to be more likely structured by common ancestry than to (co)vary independently across species regardless of ancestry (Fig. 2). However, we do find some prevailing differences in the absolute values of the pace and shape of ageing across ecoregions and Raunkiær life form. For example, phanerophytes (tall plants such as trees) tend to have a slower pace of life and reproduction (Extended Data Fig. 4), and species from tropical ecoregions typically have more negative

reproductive senescence (Extended Data Fig. 5). However, the residual covariation between ageing metrics in the species examined in our study is mostly explained by common ancestry.

Figure 2. The covariation between pace and shape metrics of ageing across animals and plants.

The covariation between the six possible combinations of ageing metrics: pace of mortality (P_μ), pace of reproduction (P_r), shape of mortality (S_μ), and shape of reproduction (S_R) ($P_\mu - S_\mu, P_\mu - P_r, P_\mu - S_R, S_\mu - P_R, S_\mu - S_\mu$ and , $P_R - S_R$) differs across the four taxonomic classes in animals (Actinopterygii – blue; Mammalia – red; Aves – purple and Reptilia; green; upper right half of contingency plot), and is explained mostly by phylogenetic relationships in plants (bottom left half of contingency plot). Displayed are posterior distributions from MCMCglmm models of the residual variance covariance matrix for each class (animals) and the phylogenetic variance covariance matrix (plants). The grey line running through each plot is aligned with an estimate of zero covariance, and black dashes indicate the upper and lower 95% credible intervals for the estimate of mean covariance.

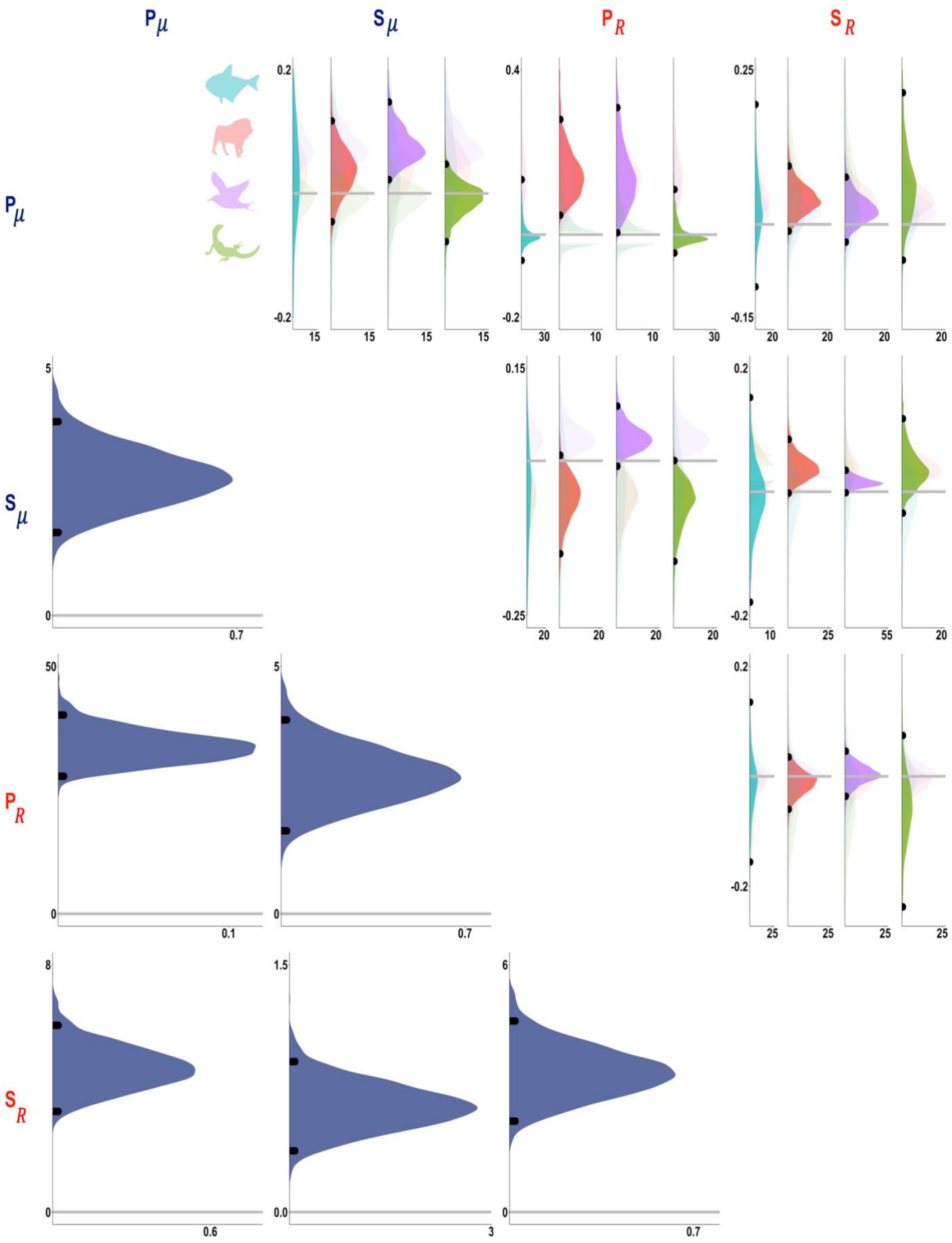

P_μ
S_μ
P_R
S_R
P_μ
S_μ
P_R
S_R

Figure 3. The pace and shape of ageing across animals. Depicted around the phylogeny are nine representative species, with values in the purple outlined boxes displaying values of (in order from inner to outer on the heatmap surrounding the phylogeny) actuarial pace of ageing (P_μ), reproductive pace of ageing (P_R), actuarial shape of ageing (S_μ), and reproductive shape of ageing (P_S). Highlighted clades represent the four examined taxonomic classes of animals from Figure 1 (Actinopterygii – blue; Mammalia – red; Aves – purple and Reptilia; Green). Clockwise from below the legend, the species are *Parus major*, *Sterna hirundo*, *Gopherus. agassizii*, *Drymarchon couperi*, *Panthera leo*, *Cervus elaphus*, *Cebus capucinus*, *Tamiasciurus hudsonicus*, and *Oncorhynchus tshawystcha*. Animal silhouettes used to visually represent species downloaded from PhyloPic (http://www.phylopic.org).

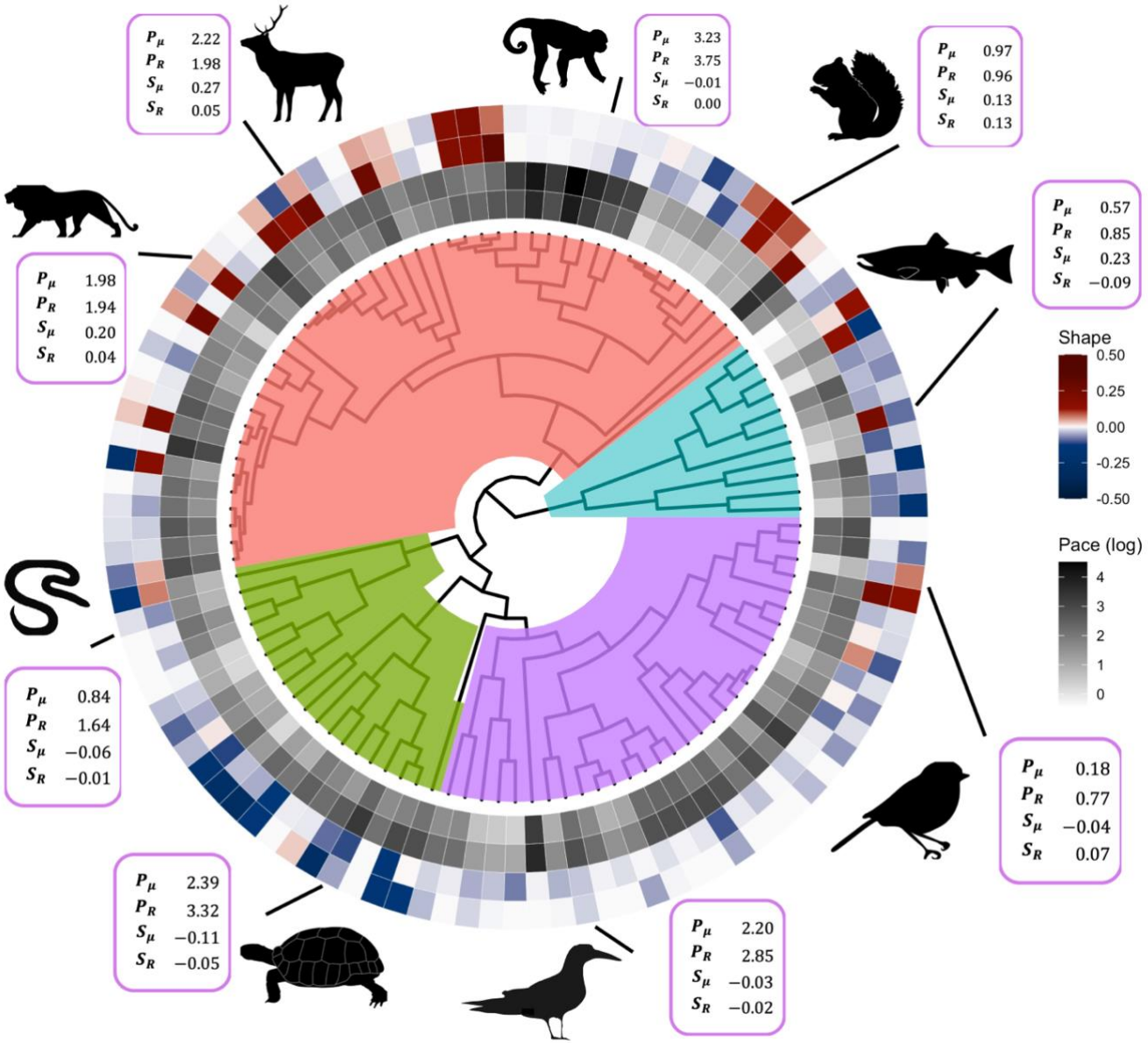

P_μ 2.22
P_R 1.98
S_μ 0.27
S_R 0.05
P_μ 3.23
P_R 3.75
S_μ −0.01
S_R 0.00
P_μ 0.97
P_R 0.96
S_μ 0.13
S_R 0.13
P_μ 0.57
P_R 0.85
S_μ 0.23
S_R −0.09
P_μ 1.98
P_R 1.94
S_μ 0.20
S_R 0.04
Shape
0.50
0.25
0.00
-0.25
-0.50
Pace (log)
4
3
2
1
0
P_μ 0.84
P_R 1.64
S_μ −0.06
S_R −0.01
P_μ 0.18
P_R 0.77
S_μ −0.04
S_R 0.07
P_μ 2.39
P_R 3.32
S_μ −0.11
S_R −0.05
P_μ 2.20
P_R 2.85
S_μ −0.03
S_R −0.02

Figure 4. The pace and shape of ageing across plants. Depicted around the phylogeny are six representative species, with values in the purple outlined boxes displaying values of actuarial pace of (in order from inner to outer on the heatmap surrounding the phylogeny) ageing (P_μ), actuarial shape of ageing (S_μ), reproductive pace of ageing (P_R) and reproductive shape of ageing (P_S). Clockwise from below the legend, the species are *Veronica fontqueri*, *Silene acualis*, *Oxalis acetosella*, *Oscularia deltoides*, *Caladenia macroclavia* and *Pinus nigra*. Plant silhouettes used to visually represent species downloaded from PhyloPic (http://www.phylopic.org).

Shape
0.50
0.25
0.00
-0.25
-0.50
Pace (log)
6
4
2
0
P_μ 3.75
P_R 4.46
S_μ −0.04
S_R −0.04
P_μ 0.93
P_R 1.47
S_μ 0.04
S_R −0.04
P_μ −0.56
P_R 0.36
S_μ −0.19
S_R 0.02
P_μ 1.55
P_R 2.32
S_μ −0.11
S_R 0.06
P_μ 0.54
P_R 1.46
S_μ −0.05
S_R −0.12
P_μ 4.75
P_R 5.50
S_μ −0.04
S_R −0.07

Discussion

Here, we show that whilst life histories strategies across animals and plants show remarkable diversity, significant levels of covariation between ageing metrics have arisen in different directions and magnitudes across taxa. In particular, birds and mammals, as determinate growers, exhibit similar underlying trade-offs across the lifespan. Furthermore, our study provides support for the strong positive covariation between expected lifespan and the average age of reproduction predicted by the fast-slow continuum[15,16,28,45]. We also displayed trends towards positive covariation in rates of senescence, suggesting that, broadly, the mammals and birds in our study trade-off survival and reproduction across the life course in synchrony. Despite these similarities, birds displayed positive covariation between their pace and shape of mortality, whilst mammals did not. Furthermore, we report a general lack of association across ageing metric combinations in reptiles and fishes, both indeterminate growers, suggesting that these groups face more variable ecological and evolutionary pressures. These key findings highlight the importance of examining ageing patterns across broad phylogenetic groupings[11,51], and provide a framework for identifying and uncovering the ecological and evolutionary drivers, and historical constraints, underpinning covariation between mortality and reproduction.

Although the pace and shape of ageing differed across ecoregions and across Raunkiær[29] life forms, covariation between ageing metrics is instead primarily driven by phylogenetic relationships in plants. Plant longevity can range from weeks to millennia[52], and reproduction through seed mass and production ranges over several orders of magnitude[53,54]. Although extremely variable, previous studies have reported that approximately 50% of plant variation in life histories is explained by the fast-slow continuum[28]. This proportion is lower than seen in animals (approx. 80%)[16], likely due to extra, complex, stages in the plant life cycle such as

seed banks or dormant stages[28] compared to the examined animal species. Despite this variation, expanding amounts of demographic data across plants have revealed that most species tend to occupy the space of negligible and negative rates of senescence[55]. Often, for plants, size, rather than age *per se*, is a better predictor of mortality risk and rate of reproduction[33]. Virtually all plants are indeterminate growers, and increasing size can result in a reduced risk of mortality and increased rate of reproduction, similar to reptiles and fish. When size and stage are incorporated into classic evolutionary theory for senescence, selection gradients can be modelled that do not necessarily decline with age[27,56], and negligible and negative rates of senescence may be expected. Indeed, despite our study sample containing plant species with values of P_μ and P_R varying over several orders of magnitude (Fig. 3), only 16/232 of our plant species (7%) displayed positive shapes of actuarial ageing and only 5/232 (2%) displayed positive shapes of reproductive ageing (Fig. 4).

The ageing metrics that we have discussed and analysed here are mathematically independent, allowing for any possible combination in theory. Yet, as we have discussed, we recovered signals of covariation between ageing metrics that was structured by class in animals, and by phylogeny in plants. The next step for future research is to identify the mechanisms behind such covariation, and to explore how general these patterns are as we extend the scope beyond our sample to more species. In animals, for instance, is it distinct organismal features such as growth determinism[27] (determinate in mammals and birds *vs* indeterminate in reptiles and ray-finned fish) or ectothermy[48,49] that drive rates of covariation between ageing metrics, or distinct differences in the ecological environment these different groups of species face? On the other hand, our results for plants suggest that demographic covariation is primarily due to evolutionary rather than ecological factors, but full satisfaction with this result likely requires more sophisticated phylogenetic approaches. Our model of brownian motion assumes a

constant rate of evolution throughout the modelled tree[57], which may not be the most accurate picture especially in angiosperm evolution[58].

Despite our study utilising high-resolution demographic data, there are still some limitations that warrant caution when considering the results presented here. When estimates of mortality and reproduction are parameterised into MPMs[30,31] or life tables[32], we lose some resolution. For example, (st)age specific estimates of older age classes might be limited in sample size[9], or biased towards high quality individuals who may escape trade-offs due to unequal ability in resource acquisition[59]. Additionally, environmental conditions in studies conducted over a relatively short period of time may result in biases. For example, if the population is measured at a time of relatively good environmental conditions, estimates of mortality risk may be reduced and estimates of reproduction may be greater relative to their long term averages[23,60]. Ideally, we should estimate ageing metrics from each individual population using an analysis that accounts for selective disappearance[12], lower sample sizes at later age classes[62], and heterogeneity of mortality and reproduction estimates between years[23]. Despite these limitations, the population level estimates we have used here provide a robust starting point for understanding the extent of covariation between ageing metrics.

The variation in patterns of mortality and reproduction throughout the lifespan across species continues to fascinate biologists studying life history theory[2,15]. Individuals are tasked with acquiring and allocating finite amount of resources[59], and ultimately trading off these limited resources over the full life course to maximise fitness. Over evolutionary time, this common problem has led to numerous solutions exemplified by the diversity of species in this study. Nonetheless, we have identified ubiquitous trade-offs and historical constraints across species, highlighted by the significant levels of covariation between ageing metrics. Though

taxonomically broad, our study did not consider certain groups of species, notably invertebrate animals, where a suite of traits including modularity[53], sociality[54], and variable growth forms might also significantly impact both the pace and shape of ageing, and covariation between. Expanding demographic research to previously under-studied taxa[65,66] is an obvious goal that will only allow for a deeper understanding of the common rules and drivers of life history strategies, and provide a more robust framework for our understanding of life history theory in general.

Data availability

Data used for the analysis presented in this manuscript are available in Extended Data Table 1 (Animals) and Extended Data Table 2 (Plants). COMADRE v. 4.21.8.0, COMPADRE v.6.22.1.2 are available for download at https://compadre-db.org/ or can be downloaded directly into R using the RCompadre package. Matrix IDs, representing the MPMs for which data was collected from for use in the analyses here are labelled in Extended Data Tables 1 and 2.

Code availability

R code needed to reproduce the analysis is included as a supplementary file.

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Covariation between metrics of ageing differs systematically among animals and plants

Extended Data Figures & Methods
A manuscript in preparation for submission to Nature as a Research Article

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Competing Interest Statement: The authors declare no competing interests.

This file contains:

Extended Methods

Extended Data Figure 1: Phylogenetic covariance estimates from MCMCglmm outputs for each aging metric combination in animals and plants

Extended Data Figure 2: Scatter plots of body mass *vs.* each aging metric in animals

Extended Data Figure 3: Violin plot of data source *vs.* each aging matric in animals

Extended Data Figure 4: Violin plots of Raunkiær lifeform *vs.* each aging metric in plants

Extended Data Figure 5: Violin plots of Ecoregion *vs.* each aging metric in plants

Extended Methods

We imposed a series of selection criteria for MPMs[1] from COMADRE[2] and COMPADRE[3] to restrict our analyses to data of the highest quality possible:

(i) MPMs were parameterised with field data from non-disturbed, unmanipulated populations (*i.e.* natural populations) to best describe the species' age trajectories.

(ii) MPMs had dimension ≥3 × 3 (i.e. rows × columns). Generally, low dimensions MPMs lack quality for the estimation of life history traits[4]. This selection criterion also helps avoid problems with quick convergence to stationary equilibrium, at which point the estimates of life history trait values and rates of senescence become unreliable[5,6] .

(iii) MPMs were only used when the entire life cycle was explicitly modelled including recordings of survival, development, and reproduction for all life cycle stages.

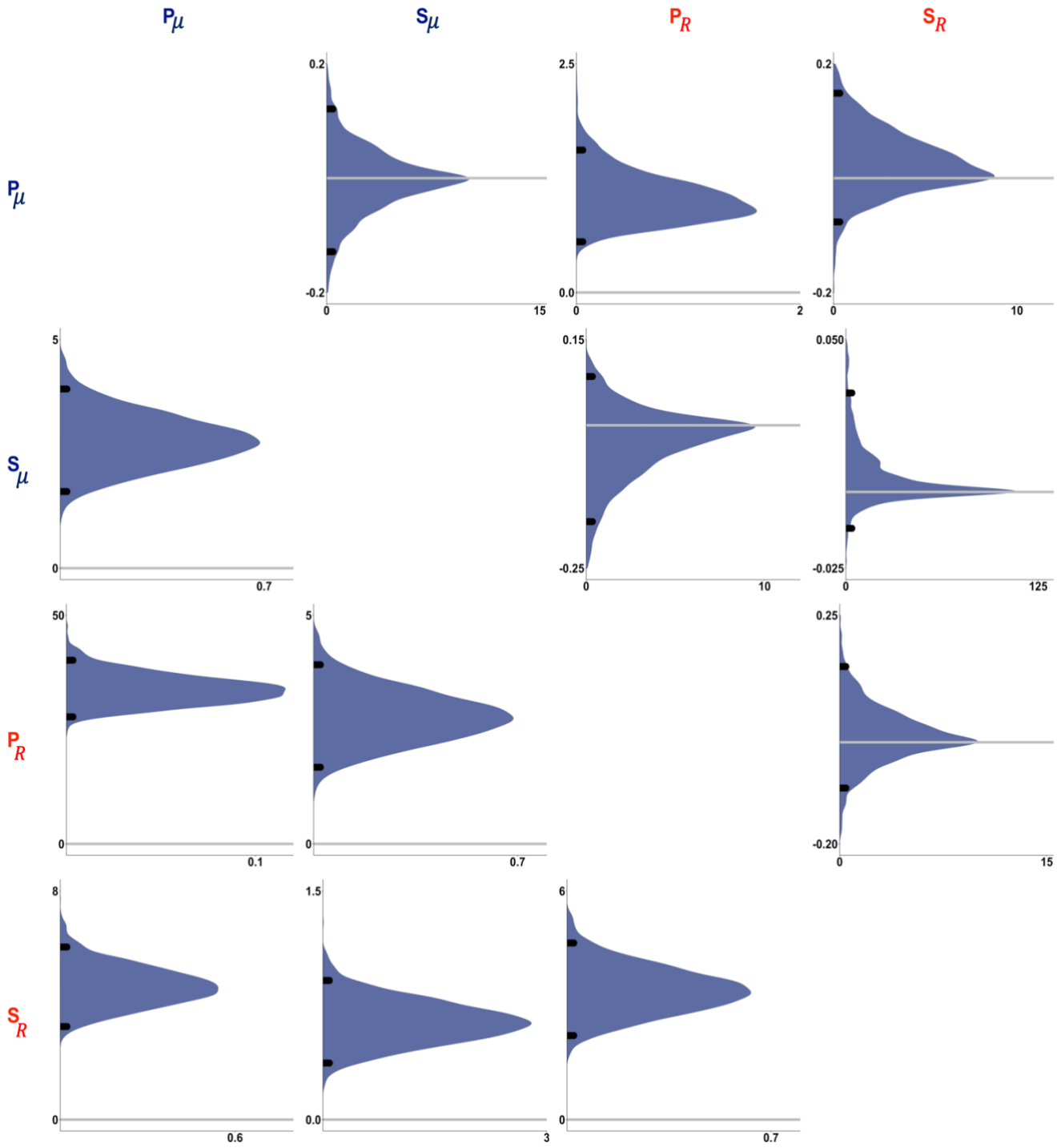
(iv) When multiple studies existed for the same species, we only averaged population MPMs if they all had both survival and reproduction data, and had the same matrix dimension.

(v) Studies of annual species modelled using seasonal projection matrices were not included; we chose only species using an annual time step. This is due to the difficulties of converting their population dynamics to an annual basis to compare with all other species' models.

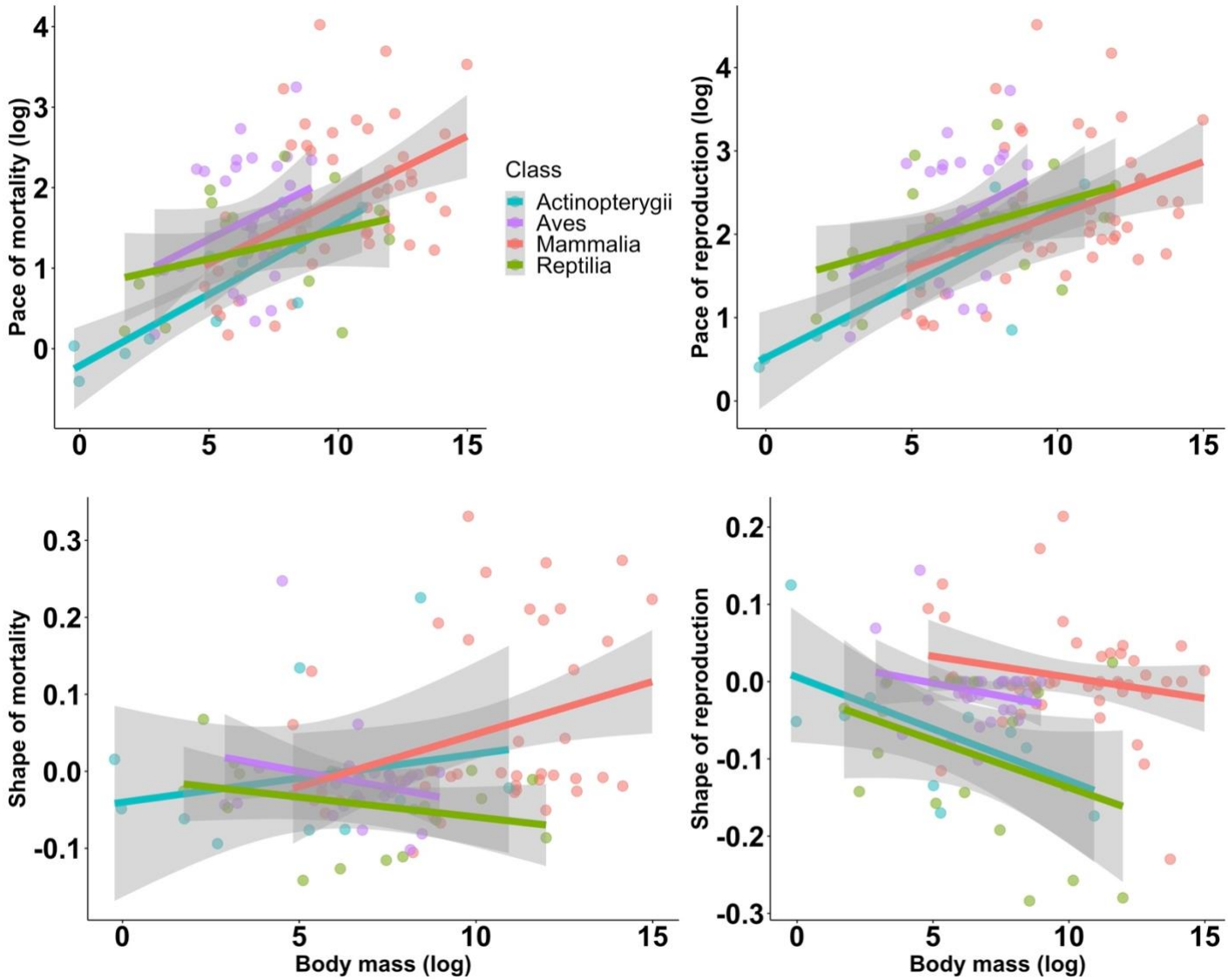
(vi) Included MPMs have stage-specific survival values ≤ 1. In a small number of published models, the stage-specific survival values can exceed 1 due to clonality being hidden in the matrix, rounding errors, or other mistakes in the original model[2,3]

(vii) MPMs were from species of which phylogenetic data was available, to ensure we were able to account for phylogenetic relatedness.

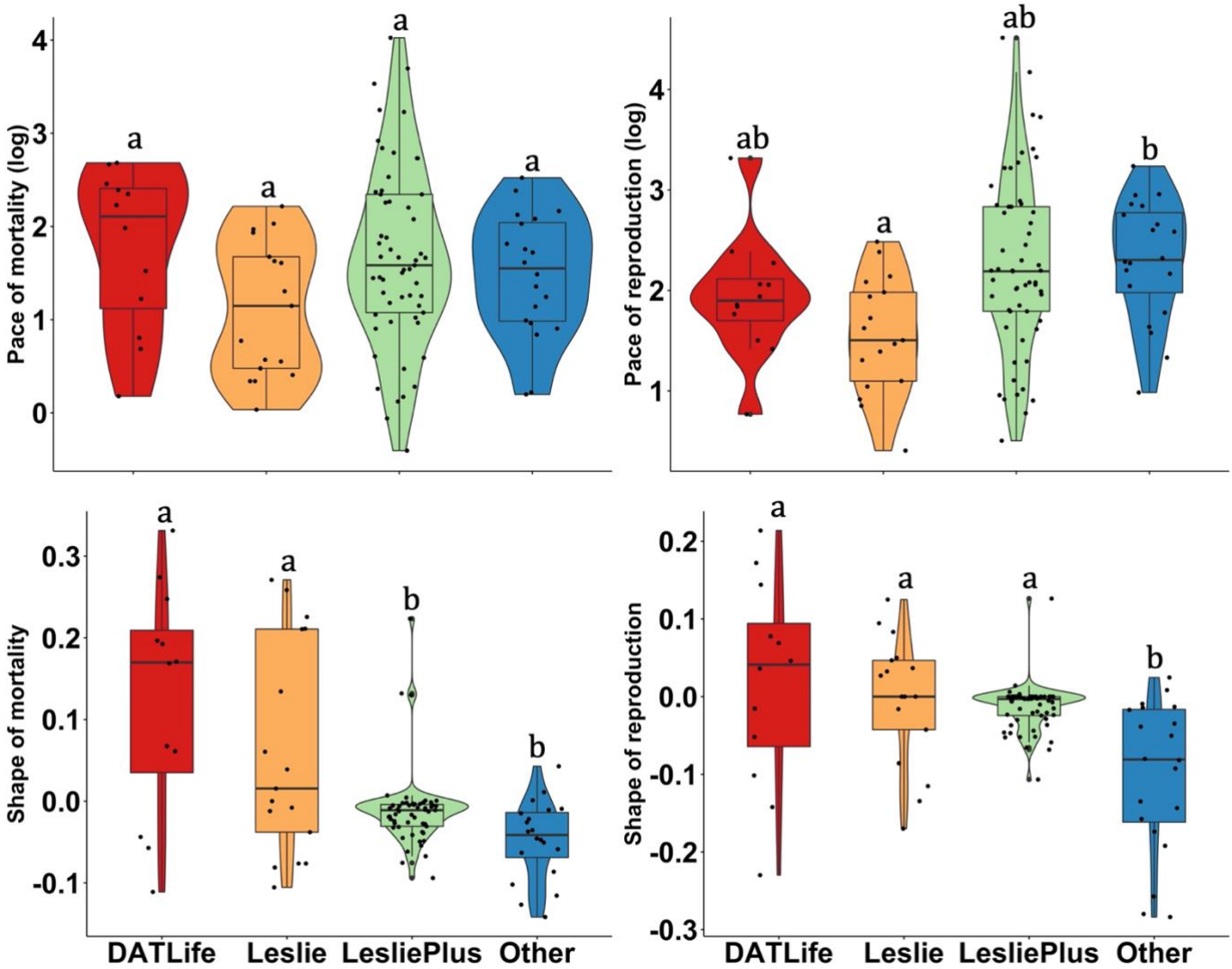
Extended Data Fig. 1 | Phylogenetic covariance estimates from MCMCglmm outputs for each aging metric combination in animals and plants. Unlike plants (left half of contingency table), most phylogenetic covariance estimates for animals (right half of contingency table) overlap zero (grey horizontal bar) for each pace and shape of aging metric combination. Displayed are posterior distributions of the estimated covariance from MCMCglmm models of the phylogenetic variance covariance matrix. Black dashes indicate the upper and lower 95% credible intervals for the estimate of mean covariance.



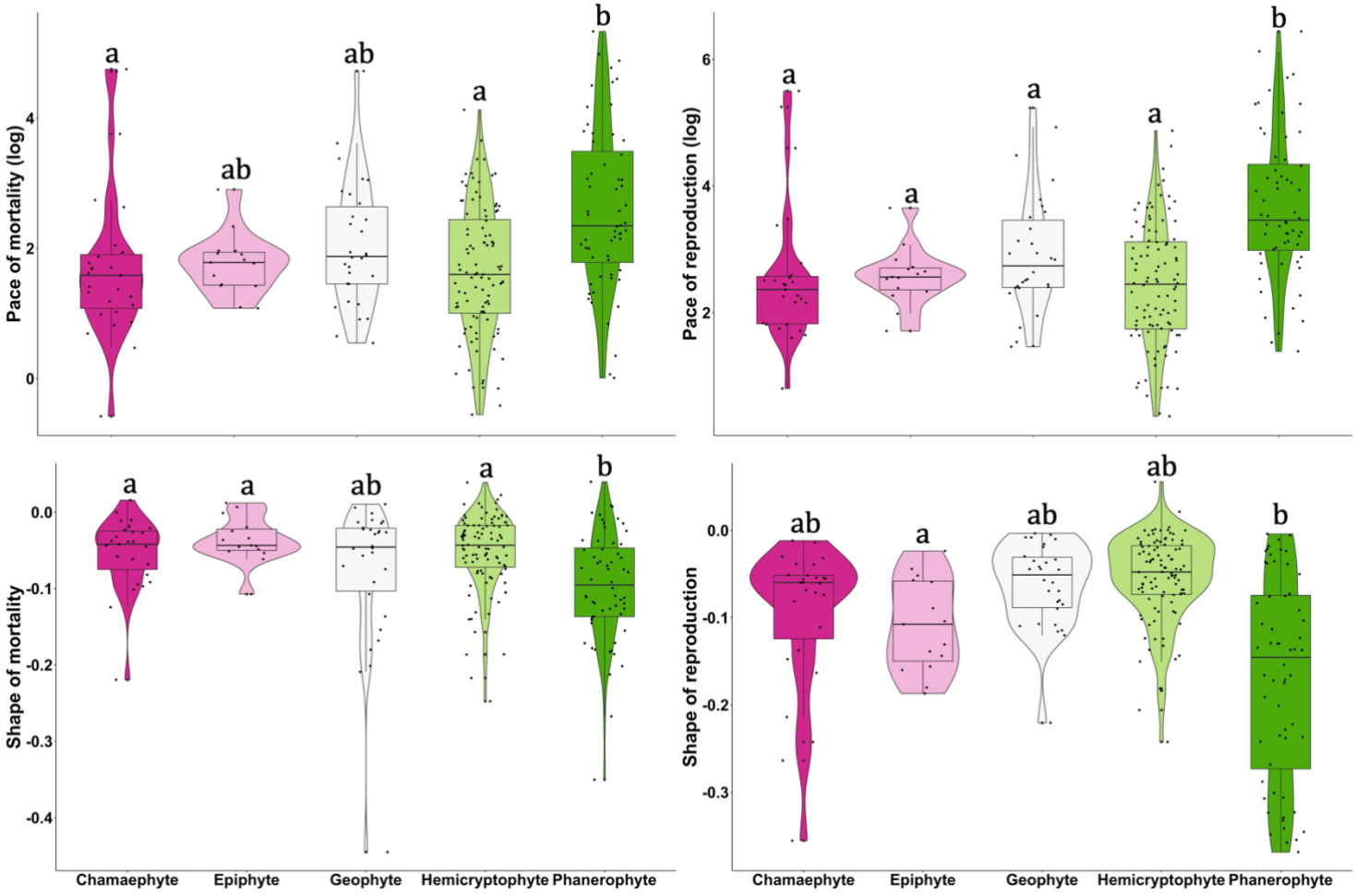
Extended Data Fig. 2 | Body mass is positively correlated with pace of mortality and reproduction, but not shape of mortality and reproduction across animals. Outputs from MCMCglmm model outputs show that log adult body mass (grams) is related to measures of pace but not shape across each of the aging metric combination models. Here, scatter plots display the relationship between adult body mass and a species' values of pace of mortality (P_μ, log), pace of reproduction (P_R, log), shape of mortality (S_μ) and shape of reproduction (S_R) (see Methods). The 106 examined animal species are grouped into the four main taxonomic classes, with a linear relationship and corresponding standard errors plotted for visualisation for each of the Classes.



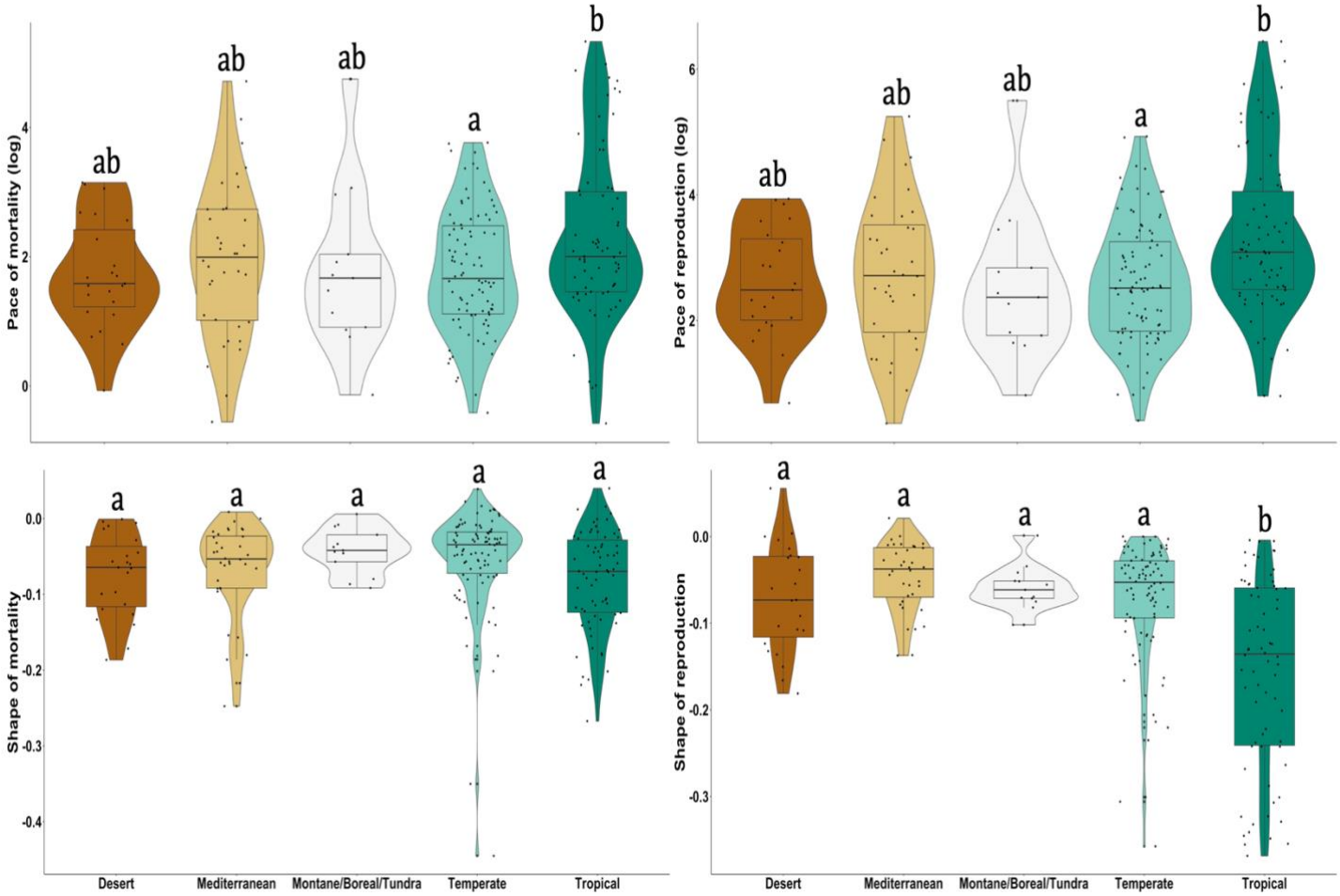
Extended Data Fig. 3 | The chosen method of modelling demographic data impacts the mean estimations of ageing metrics in animals. Shown are violin plots and results of the Tukey-Kramer post-hoc test between the chosen modelling/source of demographic data and the four ageing metrics used as response variables in our analysis. Different letters indicate statistically significant differences at a level of $p < 0.05$. DATLife provides data in the format of life tables (n = 12), whilst COMADRE (n = 96) stores data in the format of age and/or stage matrix population models (MPMs, see Methods). Leslie MPMs[7] are those with per-capita reproduction matrix elements in the top row of the matrix, between age p_x survival elements in the sub-diagonal, and zeros everywhere else, including the bottom right element of the matrix, which indicates a maximum age of survival. LesliePlus MPMs have the same format as a Leslie MPM, but contain non-zero element in the final age class indicating a constant survival rate, from which mortality and reproductive plateaus may emerge[6] (but see Methods for our approach to prevent them from entering this study). Other MPMs include stage transitions, *i.e.* non-zero elements that are not on the diagonal or top row of the matrix, e.g. Lefkovitch MPMs[8].



Extended Data Fig. 4 | Phanerophytes show distinct differences in ageing metrics compared to other Raunkiær lifeforms. Each plant species was labelled with one of five possible Raunkiær lifeforms[9], condensed from the 8 in COMPADRE[3] (see Methods). These were Chamaephytes, Epiphytes, Geophytes, Hemicryptophytes and Phanerophytes. Shown are violin plots and results of the Tukey-Kramer post-hoc test between Raunkiær lifeform and the four ageing metrics used as response variables in our analysis. Different letters indicate statistically significant differences at a level of $p < 0.05$. Phanerophytes showed distinct differences in across all ageing metric when compared to the other lifeforms.



Extended Data Fig. 5 | The Ecoregion of each plant species had a limited effect on the variation in ageing metrics. Each plant species was labelled with one of five possible Ecoregions, condensed from the thirteen in COMPADRE[3] (see Methods). These were Desert, Mediterranean, Montane/Boreal/Tundra, Temperate, and Tropical. Shown are violin plots and results of the Tukey-Kramer post-hoc test between Ecoregion and the four ageing metrics used as response variables in our analysis. Different letters indicate statistically significant differences at a level of $p < 0.05$. Plants in Tropical Ecoregions showed a greater pace of mortality and reproduction (longer actuarial and reproductive lifespan) than plants from Temperate Ecoregions. Plants from Tropical Ecoregions also showed significantly more negative reproductive senescence than plants from all four other Ecoregions.



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4 | Inclusive fitness forces of selection in an age-structured population

Main Manuscript for

Inclusive fitness forces of selection in an age-structured population

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Abstract

Current evolutionary theories of senescence predict that the force of selection on survival will decline from maturity to zero at the age of last reproduction, and the force of selection on reproduction will decline monotonically from birth. These predictions rest upon the assumption that individuals within a population do not interact with one another. This assumption, however, is violated in social species, where an individual's survival and/or reproduction may shape the fitness of other group members. In such species, it is inclusive fitness that natural selection optimises. Yet, it remains unclear how the forces of selection on survival and reproduction might be modified when inclusive fitness, rather than population growth rate, is considered the appropriate metric for fitness. Here, we derive inclusive fitness forces of selection for hypothetical populations of social species. We show that selection on survival is not always constant before maturity, and can remain above zero in post-reproductive age classes, contrary to conventional models of senescence. We also show how the trajectory of the force of selection on reproduction does not always decline monotonically from birth, as predicted by classical theory, but instead depends on the balance of benefits to direct fitness and costs to indirect fitness. Our theoretical framework provides a unique opportunity to expand our understanding of senescence across social species, with important implications to species with variable life histories.

Main Text

To date, there are no general theories for how senescence might evolve differently in groups of social species. At the demographic level, senescence is defined as the decline in organismal fitness with increasing age[1]. Hamilton[2] provided a mathematical explanation for the seemingly counter-intuitive evolution of senescence: the force of natural selection weakens with age, and so detrimental alleles acting late in life can persist despite their negative effects on fitness[3-6]. Two years prior, Hamilton[7,8] also introduced the concept of inclusive fitness, which has had a profound impact on our understanding of the evolution of social life histories[9-11]. Inclusive fitness quantifies *(i)* an individual's number of offspring in the absence of social effects and *(ii)* the effects an individual has on the number of offspring produced by other individuals, weighted by relatedness[7,8]. What has not yet been fully considered from a theoretical perspective, however, is how an individual's effects on the fitness of others may alter the evolution of senescence.

An age-specific force of selection describes the relative effect on fitness at different age classes of a mutant allele that impacts survival or reproduction. How might the components that contribute to such age-specific forces of selection differ between a solitary and a social species? First, consider an individual of a solitary species. When this individual dies, it loses access to any future reproduction it might have achieved. If a mutant allele arises in this population that increases the risk of dying at a certain age, say x, then the force of selection that acts against the allele is proportional to the expectation of residual reproduction that the individual may have realised[2]. Now, imagine instead a social species in which individuals within a group influence one another's survival and reproduction, for example, through the provision of alloparental care or through competition for limiting resources. For an individual, death means the loss of any future reproduction, just as in the solitary case. However, in social species, an

individual's death may also alter the survival and reproduction of other individuals[12,13]. For instance, the death of an individual providing alloparental care may lead to a reduction in breeder productivity. Alternatively, where there is competition within groups for resources, the death of an individual may release resources that other group members may use for survival and reproduction. If individuals within a group are related, then these effects will be under kin selection. For example, an increase in mortality late in life can be adaptive if relatives stand to benefit from the death of a focal individual[14-20]. On the other hand, mortality may be more strongly selected against if individuals can transfer beneficial resources to others[21-23]. When the death and reproduction of a focal individual not only impacts its own fitness, but also the fitness of relatives, the force of selection acting on a mutant allele at age x must also consider these complex social effects.

To incorporate social interactions into the evolutionary theory of senescence, we develop a general model for quantifying age-specific inclusive fitness forces of selection in social species. Here, we focus on the effects of cooperative interactions between individuals and the corresponding forces of selection, but note that our model also has scope to consider other scenarios, such as cases of harm (see **Discussion**). Using an infinite island framework to describe a resident social population[16,20,24-34], we explore the fate of a mutant allele that alters *(i)* the survival rate from age x to age $x+1$ and *(ii)* reproduction at age x. We derive inclusive fitness forces of selection acting on these mutant alleles, which indicate how the efficacy of natural selection changes with age with respect to socio-demographic parameters. After deriving general analytical results, we explore the applicability of our framework to different social settings by providing numerical solutions for two examples of social structures: *(i)* the grandmother hypothesis: post-reproductive individuals aiding juvenile survival and *(ii)*

cooperative breeding: juveniles aiding reproduction by adults. We conclude by discussing the implications and possible extensions for our model.

Results

Model

We consider a population divided into an infinite number of patches, and model the population dynamics of a focal patch. This infinite island approach[16,20,24-34] allows kin selection to be modelled while also considering the effects of demography, which is appropriate for considering an age-structured population in which individuals have effects on one another's fitness. Each patch, which could also be conceptualised as a territory, contains discrete groups of exactly N individuals that are, for simplicity, haploid and asexual. We also assume that patches produce a large number of offspring in each breeding season so that no position on any patch is vacant at the start of each breeding season (*i.e.* a density-dependent stationary population). Offspring that establish on to a patch are designated age 1 and can survive until some maximum age, ω, at which point they die. Time proceeds in a series of discrete breeding seasons, during which individuals have a probability of surviving to the next breeding season, $p(x)$, and a rate of reproduction, $b(x)$, that may vary with age, and can be described by a population matrix model ($\boldsymbol{A}$). Individuals may receive contributions to their survival and reproduction from the other $N-1$ individuals on their patch, and may themselves contribute to the survival and reproduction of the $N-1$ conspecifics on the patch.

Fundamental to this model is the concept of 'transfers'. Biologically, transfers represent the help or harm to other individual's fitness components: survival and reproduction. Transfers occur in the currency of genetic offspring equivalents, the same currency as survival and reproduction. Here, we assume that the transfers an individual makes to others is a function of

the ages of both the actor and recipient (Fig. 1). We display transfers between individuals as T_{yz}^{x}: if y = 1, this represents an individual in age class x's social effect on the reproduction of age class z, while $y = z + 1$ would represent an individual in age class x's social effect on the survival of age class z.

To quantify the inclusive fitness contributions of a focal individual of age x, a series of key considerations must be made. Specifically, we must *(i)* exclude the fraction of the class-y offspring of a focal class-x individual that are born or survive as a consequence of the social environment (the help or harm of other individuals), and *(ii)* augment the total production of class-y offspring from all other age classes, including other individuals in age class x, that are born or survive due to the social contributions of a focal class-x individual. These latter offspring contributions are weighted by the coefficient of relatedness between an individual of age class x and the class-y offspring of the recipient class[7,8]. For example, a focal individual aged x survives with probability $p(x)$ and has a rate of reproduction $b(x)$. A fraction of these rates of survival and reproduction may be due to social interactions. These fractions are excluded from the inclusive fitness of the focal individual, leaving $\dot{p}(x)$ and $\dot{b}(x)$, with dot notation representing the effect of a focal individual's own genotype on its own survival or rate of reproduction, *i.e.* direct fitness. Of the $\dot{b}(x)$ offspring produced due to the genotype of an individual aged x, a proportion d disperse, and a proportion $1 - d$ remain at their natal patch. A fraction c of the dispersing offspring die, representing a cost of dispersal. Surviving, dispersed offspring are evenly distributed among all sites, regardless of distance, and compete (fair lottery) for sites freed by adults that die in the current breeding season. Asymmetric competition is assumed so that juveniles do not displace resident adults, and die if they do not gain a breeding position on a patch. Offspring of a focal individual aged x face a probability of establishment $g(x)$ onto their natal patch, and $\bar{g}$ on a different, random patch in the

population. The term $\bar{g}$ represents the average probability of establishment for an offspring on another patch, which averages the survival of patch members according to the expected age distribution of patch members.

In a population with social interactions between patch members, we can populate a matrix ($\boldsymbol{W}$) with the inclusive fitness (genetic offspring) contributions of individuals in age class x to individuals in age class y (w_{yx}):

$$w_{yx} = \begin{cases} \dot{p}(x) + T^{x}_{x+1,x} & \text{if } y = x+1 \\ \dot{F}(x) & \text{if } y = 1 \\ 0 \; OR \; T^{x}_{yz} & \text{if } y = z+1 \end{cases}$$

[1]

where

$$\dot{p}(x) = p(x) - \sum_{z} T^{z}_{x+1,x}$$

[2]

and

$$\dot{b}(x) = b(x) - \sum_{z} T^{z}_{1,x}$$

[3]

and

$$\dot{F}(x) = \dot{b}(x) + \sum_{z} T^{x}_{1,z}\,[(1-d)g(x) + (1-c)d\bar{g}]$$

[4]

The proportions of the survival and reproduction of a focal individual aged x that are due to the genotypes of other individuals are represented in the summation terms on the right-hand

side of **[2]** (survival) and **[3]** (reproduction) (where $0 \leq \sum_z T^z_{x+1,x} < p(x)$, and $0 \leq \sum_z T^z_{1,x} < b(x)$). Importantly, these proportions are distributed to other age classes, thus ensuring that no offspring is 'double counted'[35,36]. A focal individual of age x may also contribute to the survival and reproduction of others, accumulating indirect fitness through the transfer of genetic offspring. Contributions to survival are captured as $T^x_{y,z}$ (where $y = z + 1, \text{and } y \neq 1$), and reproduction as $T^x_{1,z}$ (summed across age classes to equal $\sum_z T^x_{1,z}$). The magnitude of these contributions will depend on *(i)* the expected number of individuals in the recipient age class, *(ii)* the fraction of the total contribution of all age classes combined to the survival or reproduction of the recipient age class individuals that is due to a focal individual aged x, and *(iii)* the relatedness between a focal individual aged x and an individual in the recipient age class (see **Supplementary Information**). This approach to modelling social interactions assumes that there are fractions of survival and fecundity of each age class that are due to the social environment (which could equal zero), and that these fractions are distributed to other individuals across age classes. If there are no explicit social interactions between multiple individuals on a patch, equation **[2]** simplifies to a population with limited dispersal and Ronce & Promislow's[20] kin competition selection gradients can be computed. With full dispersal (no offspring stay at their natal patch) and no social interactions, equation **[1]** simplifies to Hamilton's panmictic population, and his forces of selection can be computed[2].

An inclusive fitness force of selection

To compute forces of selection, we are ultimately concerned with a hypothetical mutation that alters survival rate or rate of reproduction at age x. The derivative of the growth rate of the mutant population, λ, with respect to the phenotypic effect of the mutation, δ, gives an indicator of the force of selection acting on the mutant allele[2,20,37,38]. We consider mutations of weak effects (small δ) and first-order effects of selection[39]. Using this 'sensitivity' approach for an

age-structured population[20,37,38,40-42], the force of selection acting on a mutant allele can be written as:

$$S = \frac{d\lambda}{d\delta}_{\delta=0} = \sum_x \sum_y \frac{f_x v_y}{\mathbf{f} \cdot \mathbf{v}} \frac{dw_{yx}}{d\delta}_{\delta=0}$$

[5]

where **f** and **v** are, respectively, the vectors of asymptotic frequencies and inclusive reproductive values for the different age classes in the resident population. The term f_x denotes the asymptotic frequency of age class x, and **f** is the dominant right eigenvector of the demographic projection matrix (***A***). In this model, the term v_x represents the inclusive reproductive value of age class x, and is instead derived from an inclusive fitness matrix (***W***) that decomposes the demographic projection matrix into inclusive fitness contributions between age classes. Therefore, **v** is the dominant left eigenvector of ***W***. Thus, the growth rate of the mutant population, λ, represents an inclusive fitness growth rate of the allele. Finally, the term w_{yx} represents the class y offspring of a class x individual (genetic offspring equivalents). Therefore, dw_{yx} represents the difference in the contribution of an individual age x to individuals aged y in the mutant population compared to the resident population. Overall, the sign of *S* predicts the direction of selection on the mutant allele with respect to the resident population wild type allele, whilst the magnitude of *S* conveys information about the force of selection[2,20].

The inclusive fitness force of selection on survival

A mutant allele that alters the survival rate between age x and $x + 1$ changes inclusive fitness contributions between age class according to the following (see **Methods**):

$$dw_{yx} = \begin{cases} d\dot{p}(x) & \text{if y = x + 1} \\ -d\dot{p}(x)\left[\dot{h}(x) + \dot{k}(x)\hat{r}(x)\right] & \text{if y = 1} \\ 0 & \text{otherwise} \end{cases}$$

[6]

where $\dot{h}(x)$ is the proportion of offspring after dispersal at the local patch that are the direct and indirect contributions of a focal individual aged x, $\dot{k}(x)$ is the proportion of offspring that are born due to the genotypes of other individuals on the patch, and $\hat{r}(x)$ is the relatedness of an individual aged x to the offspring of other patch mates (see **Methods**). As we assume mortality occurs between breeding seasons, a focal individual's contributions to the survival and reproduction of other age classes are only affected at $x + 1$, not in the current breeding season.

Let $S_p(x)$ be the component of the force of selection due the effect of a mutant allele on the survival rate between age x and $x + 1$. Using equations **[5]** and **[6]**, in a stationary population with limited dispersal and social interactions between individuals, this can be written as:

$$S_p(x) = \frac{d\dot{p}(x)}{d\delta}\frac{f_x(v_{x+1} - \left[\dot{h}(x) + \dot{k}(x)\hat{r}(x)\right]v_1)}{\mathbf{f}.\mathbf{v}}$$

[7]

Equation **[7]** shows that the overall direction of the force of selection acting on a mutant allele that affects the survival rate between age x and $x + 1$ is a balance of two forces: the inclusive reproductive value at age $x + 1$ *vs* the reproductive value of offspring (displaced by the survival of the focal individual) that have varying relatedness to the focal individual aged x. The term $\mathbf{f}.\mathbf{v}$ acts to scale the forces of selection in terms of generation time[2,20].

The inclusive fitness force of selection on reproduction

A mutant allele that alters reproduction at age x changes inclusive fitness contributions between age class according to the following (see **Methods** and **Supplementary Information**):

$$dw_{yx} = \begin{cases} 0 & \text{if y = x + 1} \\ d\dot{b}(x)\left[(1-d)g(x)\left[(1-h(x)) - \dot{I}(x) - \dot{k}(x)\hat{r}(x)\right] + (1-c)d\bar{g}\right] & \text{if y = 1} \\ 0 & \text{otherwise} \end{cases}$$

[8]

Then, let $S_m(x)$ be the component of the force of selection due the effect of a mutant allele on reproduction at age x. Using **[5]** and **[8]**, in a stationary population with limited dispersal and social interactions between individuals, this can be written as:

$$S_m(x) = \frac{d\dot{b}(x)}{d\delta}\frac{f_x v_1}{\mathbf{f}.\mathbf{v}}\left[(1-d)g(x)\left[(1-h(x)) - \dot{I}(x) - \dot{k}(x)\hat{r}(x)\right] + (1-c)d\bar{g}\right]$$

[9]

where

$$\dot{I}(x) = \frac{\sum_z T_{1,z}^x (1-d)}{b(x)(1-d) + (N-1)\bar{b}(1-d) + N\bar{b}(1-c)d}$$

[10]

is the fraction of all offspring at the local patch after dispersal that exist due to indirect effects of the genotype of a focal individual aged x. Equation **[9]** shows that the overall force of selection acting on a mutant allele that affects the rate of reproduction at age x is also comprised of two components: *(i)* the effect of the allele on the probability of establishment of different

types of offspring onto the local patch and *(ii)* the effect of the allele on the dispersing offspring that are part of the direct fitness of the focal individual aged x. Selection for effect *(ii)* will always be positive; however, selection for effect *(i)* will depend on the relative weights each class of offspring contributes to the overall effect. In this model, an increase in direct reproduction is, all else being equal, beneficial for the direct fitness of a focal individual, but detrimental to the indirect fitness of the focal individual.

Applications of the model

Equations **[7]** and **[9]** provide general solutions for age-specific inclusive fitness forces of selection on individual survival and reproduction in group structured populations. To visualise the results, we consider two hypothetical populations of iteroparous individuals with social interactions (Fig. 2, Fig. 3). For each, we consider background demography described by age-specific vital rates, $p(x)$ and $b(x)$. We parameterise mortality risk at age x using the Siler model[43]:

$$\mu(x) = \alpha_1 e^{-\beta_1 x} + \alpha_2 e^{\beta_2 x} \qquad \textbf{[11]}$$

The probability of survival at age x, $p(x)$, is therefore equal to $e^{-\mu(x)}$. The probability of survival to age x ($l(x)$) is then $l(x) = \prod_1^{x-1} p(x)$, with $l(1) = 1$. As we assume all patches have no breeding positions available at the start of each breeding seasons (*i.e.*, a density-dependent stationary population), we can calculate the asymptotic frequency (f_x) of each age class as

$$f_x = \frac{l(x)}{\sum_y l(y)} \qquad \textbf{[12]}$$

We then parameterise individual rate of reproduction at age x as:

$$b(x) = \begin{cases} 0 & \text{if } x < \varepsilon \\ (x-\varepsilon)e^{-\varphi(x-\varepsilon)} & \text{if } x \geq \varepsilon \\ 0 & \text{if } x > \kappa \end{cases}$$

[13]

where ε designates the age of reproductive maturity, κ represents an age at which reproduction ceases, and φ modulates the shape of reproduction across age classes.

Fig. 2*A* and Fig. 3*A* illustrate the life cycles of the two hypothetical social populations. Fig. 2*A* considers a population with post-reproductive individuals providing care for juveniles, as seen in humans[44], orcas[45], and Asian elephants[46]. Fig. 3*A* considers a population with juvenile individuals providing help to the reproduction adult breeders, as is found in many cooperatively-breeding species[47]. Fig. 2*B* and Fig. 3*B* display the modelled survivorship and reproduction as a function of individual age. We then apply our methodology (see **Model)** to partition these vital rates into inclusive fitness contributions between age classes and compute a fitness matrix (**W**) with elements described in **[1]**. Fig. 2*C* and Fig. 3*C* show the forces of selection acting on survival and reproduction at age *x* in these hypothetical social populations according to equations **[7]** and **[9]**.

We show that the force of selection acting on survival in social populations is not necessarily constant before maturity, as predicted by classical theory[2]. The exact pattern depends on whether pre-reproductive individuals gain indirect fitness through transfers or not. When juveniles do not engage in helping behaviour, the force of selection increases in the juvenile period as relatedness to newborn offspring decreases with increasing juvenile age (Fig. 2*C*; Fig. 2*D*). This decline in local relatedness facilitates a more 'selfish' force of selection on survival throughout the juvenile period. On the other hand, when juveniles provide help to

adult reproduction, the force of selection on survival generally decreases from the age at which indirect fitness was first accrued (Fig. 3*C*; Extended Data Fig. 3), rather than the age of first reproduction. In both examples, the force of selection on survival then declines throughout adulthood as future inclusive reproductive value declines and the relatedness to newborn offspring increases. When post-reproductive adults continue to accrue indirect fitness, the force of selection on survival can remain above zero in post-reproductive age classes (Fig. 2*C*; Extended Data Fig. 1). The magnitude of the force of selection is greater in post-reproductive age classes when juvenile dispersal is lower (and so there is higher local relatedness) and the magnitude of help provided by post-reproductive individuals is higher (Extended Data Fig.1). In general, the force of selection on survival will always have a positive component until the final age at which inclusive fitness is accrued, rather than necessarily the age of last reproduction. At this age, when future survival is no longer possible, the first term on the numerator of Equation **[7]** is zero**,** and so, if there is some level of local relatedness (*i.e.* $\hat{r}(x) > 0$), selection will favour increased mortality as it will benefit the establishment of related juveniles.

In populations with relatively long lifespans, the force of selection on reproduction was weaker than the force of selection on survival. The force of selection acting on reproduction at age x generally declined from birth, as predicted by Hamilton's model[2], but not always (Extended Data Fig. 4), and the decline was more rapid when the rate of dispersal was lower (Extended Data Fig. 2). This more rapid decline is likely due to the greater inclusive fitness costs of increasing personal reproduction when local relatedness is higher. The force of selection on reproduction in early life is also weaker when post-reproductive adults have a more significant impact on juvenile survival. In all iterations of the model (Fig. 3*C*; Extended Data Fig. 3), there

was a slight increase in the force of selection acting on reproduction in the final age class, when the force of selection on rate of survival becomes negative.

When considering the evolution of demographic senescence, evolutionary biologists use population growth rate, r, as the measure of fitness[48] (but see[49]). The magnitude of the change in population growth rate due to an age-specific change in survival and/or reproduction generally declines with age (but see[50] for other indicators of the force of selection), and this decline facilitates the evolution of senescence[2]. However, for social species, it is crucial to consider explicitly the inclusive fitness of individuals as the quantity that natural selection seeks to maximise[10]. Indeed, the change in inclusive fitness due to an age-specific change in individual survival and/or reproduction must consider the combined effect on all individuals that are affected by the change[24]. Here, we show that, in an age-structured model for patch-structured social populations, considering the inclusive fitness effects of an allele significantly alters the form of the forces of selection acting on age-specific survival rate and rate of reproduction.

Our framework provides several key insights into the force of selection acting on survival and reproduction in social species. First, the force of selection acting on the survival rate of that age class is the product of future inclusive reproductive value (IRV), rather than conventional reproduction value (RV[48]), and the asymptotic frequency (stationary age distribution) of that age class. Since IRV remains above zero after reproduction ceases, if post-reproductive adults continue to accrue indirect fitness benefits, selection on survival of post-reproductive age-classes does not necessarily go to zero as in Hamilton's model[2]. Importantly, this finding provides a formal inclusive fitness framework for the 'grandmother hypothesis'[51,52], supporting work that has suggested indirect fitness benefits are essential to sustained post-reproductive

lifespan[23,31]. In our framework, the force of selection on survival of social species will remain non-zero until there is no future IRV. At this point, if there is some local relatedness, the force of selection on increased survival will be negative. Combined with an increase in the force of selection on reproduction at a 'final age class', a kin-selected terminal investment strategy, in which it pays to invest heavily in reproduction at the expense of survival to maximise the establishment of kin, may be favoured[19].

The incorporation of age-specific indirect fitness into the evolutionary theory of senescence means that selection on survival before maturity is not necessarily constant (Fig. 2*C*; Fig. 3*C*). This difference occurs because of the balance between the future IRV of the individual and the IRV of newborns displaced by increased survival. If relatedness to other individuals declines throughout the juvenile period as a focal individual ages, and the focal individual's own IRV increases as they approach maturity, the balance in Equation **[7]** is weighted more heavily towards the first term, and the force of selection on increased survival will increase. On the other hand, in populations where juveniles help and accrue indirect fitness, the force of selection on survival will decline from the age at which indirect fitness is first gained. This result implies that, in species with pre-reproductive help, senescence should start from the age at which inclusive fitness is first gained, rather than the age of first reproduction, as in conventional models[2,20].

An inclusive fitness force of selection acting on reproduction depends on the costs and benefits associated with increasing personal reproduction. In our framework, selection for increased reproduction will always have a positive component due to the increased probability of an offspring (whether philopatric or dispersive) establishing on to a patch. However, the subsequent decrease in probability of other locally produced offspring establishing on to the

patch reduces the magnitude of the force of selection acting on reproduction. This result may be especially important for groups experiencing strong competition over resources[12]. For example, a negligible force of selection on reproduction may favour reproductive restraint by some individuals within cooperatively-breeding groups, when access to reproduction is limited and inclusive fitness costs of increasing personal reproduction would be substantial[32].

Discussion

Our framework builds on previous work that has made significant ground in incorporating social effects into the evolutionary theory of senescence. Lee's[23] model showed that the force of selection acting on age-specific mortality can be modified by intergenerational transfers of resources. However, kin selection did not enter the formal model as no explicit spatial structured was considered. Here, by explicitly considering a patch-structured population with dispersal, we allow for variation in relatedness and thus a larger breadth of possible kin selection effects to be considered. Ronce & Promislow[20] derived analytical solutions that provided the baseline framework for the model here, showing that the force of selection on increased survival includes a negative component driven by the displacement of offspring from establishing on the local patch. This term is similar to the negative term in **[7]**; however, our framework also explicitly considers the impact of survival on the establishment of other locally produced offspring. By only considering single individuals on a patch, social interactions in Ronce & Promislow's model were limited to kin competition between parent and offspring over residency on the patch. Here, by including multiple individuals on the patch, we can also incorporate social effects into the form of the force of selection on reproduction (**[10]**). Finally, Moorad & Nussey[53] took a quantitative genetics approach to add indirect genetic effects, explicitly considering maternal effect senescence, but modelled no explicit demography. A

combination of explicit demography, as modelled here, and quantitative genetics could prove a major future step.

The framework we present here provides a base to expand our understanding of senescence across social species. For example, previous work has found mixed evidence for extended lifespan in cooperative breeders[54-57], and some evidence for differences in rates of senescence between cooperative and non-cooperative breeders[58]. Previous theory suggests that it is longer life and overlapping generations that initially favour cooperation[26], but also that a delayed age of first reproduction as a result of queuing for reproduction might be a self-reinforcing mechanism for extended lifespan in cooperative breeders[59]. However, multiple other facets of the demography of cooperative breeding systems, including the process of group formation[60], the structure of dominance hierarchies[61] and levels of reproductive skew[62], all have the potential to play a role in determining lifespan and rates of senescence. These group-level characteristic can, in theory, contribute to the shape of the age class asymptotic frequency and inclusive reproductive value distributions that, demonstrated in this model, underpin inclusive fitness forces of selection. Our model provides a framework to stimulate further theoretical work for how these features of cooperative breeding systems may impact the evolution of lifespan and senescence.

Here, we focused on how cooperative interactions between members of a group can alter age-specific inclusive fitness forces of selection. However, in many groups, competitive interactions over limited resources are also rife. In our model, transfers between age classes reflect the net effect of the presence of an individual in one age class on the survival and reproduction of an individual in another age class. If the net effect is negative, then the genetic offspring transfer is also negative. For example, consider again the social system illustrated in

Figure 2. Instead of post-reproductive individuals having a positive effect of the survival of juveniles, let us instead imagine a scenario in which the presence of post-reproductive individuals is harmful to the survival of juveniles. An allele that increases the rate of survival in such post-reproductive individuals will be selected against due to the inclusive fitness costs imposed from the negative effects on related juvenile individuals, potentially hastening the evolution of more rapid senescence. Finally, we only considered indirect fitness returns from social interactions. In many cooperative breeding systems, however, direct fitness returns from social interactions can be the main driver for alloparental care[47]. Some form of direct fitness benefits could be incorporated into the model by delaying the age at which returns from social interactions are realised, as hypothesised by group augmentation theory[63].

In summary, recent research has focused on the potential for social interactions to drive variation in senescence across species[1,64]. The model we present here shows that, when inclusive fitness consequences of increasing individual survival or reproduction are considered, age-specific forces of selection can vary markedly from previous asocial models. Our results thus support the hypothesis that sociality can shape patterns of senescence in nature. Further theoretical, empirical and comparative studies are now needed to determine the amount of variation in senescence patterns that can be explained by social modes of life.

Methods

Appendix A: Relatedness

In order to quantify indirect genetic contributions, it is essential to consider the relatedness between different age classes of individuals in the population. The relatedness of a focal individual aged x to other individuals on the patch, including themselves, can then be described as:

$$r(x) = \frac{1}{N} + \frac{N-1}{N}\hat{r}(x) \ .$$

[A1]

Then, let r_{yx} denote the probability that an allele sampled randomly from a given locus in an individual aged x is identical by descent (IBD) to an allele sampled randomly from the same locus in an individual aged y[26,27,32,34,65,66]. The term $\hat{r}(x)$ represents the average relatedness of a breeding individual aged x to another random breeder on the same patch[26,27], which is equivalent to the mean relatedness of a focal individual aged x across all age classes ($\hat{r}(x) = \overline{r_{yx}}$). Given the assumption of haploid genetics and asexuality, $\hat{r}(x)$ is therefore also the relatedness of a focal individual aged x to the offspring of the other individuals on the patch. Under the assumption of infinite patches, any immigrants arriving at the focal patch will not have any relatives when they arrive, and the relatedness of individuals on the patch of any age to these immigrants is equal to 0.

Let us define $h(x)$ as the proportion of offspring after dispersal at the local patch that are the offspring (not partitioned into inclusive fitness contributions) of a focal individual aged x:

$$h(x) = \frac{b(x)(1-d)}{b(x)(1-d) + (N-1)\bar{b}(1-d) + N\bar{b}d(1-c)}$$

[A2]

where $\bar{b}$ represents the average rate of reproduction. For simplicity, we assume no demographic stochasticity within patches (see **Discussion**). Then, let $k(x)$ define the proportion of offspring after dispersal at the local patch that are the demographic offspring of other individuals on the patch besides the focal individual aged x:

$$k(x) = \frac{(N-1)\bar{b}(1-d)}{b(x)(1-d) + (N-1)\bar{b}(1-d) + N\bar{b}d(1-c)}$$

[A3]

Using equations **[A2]** and **[A3]**, we can describe the relatedness between an individual aged x to a different individual on the patch aged y as a function of both individual's ages:

$$r_{yx} = \begin{cases} h(x-y) + k(x-y)\hat{r}(x-y) & \text{if } y < x \\ (1-d)^2\left[\bar{h}^2 + \left(1-\bar{h}^2\right)\hat{r}(1)\right] & \text{if } y = x \\ h(y-x) + k(y-x)\hat{r}(1) & \text{if } y > x \end{cases}$$

[A4]

First, consider the case when the individual of age x is older than the individual of age y (top row of **[A4]**). The individual aged y was born y breeding seasons ago, when the individual aged x was $x - y$ years old. At age $x - y$, the proportion of offspring at the local patch after dispersal that are the offspring of an individual aged $x - y$ is defined as $h(x - y)$. Therefore, with probability $h(x - y)$, the individual aged y is the offspring of the individual aged x from $x - y$ breeding seasons ago, and thus the relatedness between the two individuals is one. Then, let $k(x - y)$ define the proportion of offspring at the local patch after dispersal $x - y$ breeding seasons ago that were the offspring of other individuals on the patch. With probability $k(x - y)$, therefore, the individual aged y was born to another individual on the patch. Therefore, the relatedness of the individual aged x to the individual aged y is equal to the relatedness of an individual aged $x - y$ to a random offspring born locally to the patch, which is equal to the relatedness of an individual aged $x - y$ to another random individual on the patch ($\hat{r}(x - y)$). The remaining proportion of offspring at the patch after dispersal $x - y$ breeding seasons ago ($1 - h(x - y) - k(x - y)$) were from elsewhere in the population and thus relatedness is 0.

Second, consider the case when both individuals are the same age (second row of **[A4]**). The probability that both are local to the patch is $(1 - d)^2$. If both individuals are born locally, we then have to consider the probability that both individuals were born to the same mother, and thus are siblings related by 1. If the average proportion across age classes of offspring that are born to an individual is $\bar{h}$, then the probability that two offspring born x breeding seasons ago were born to the same mother is equal to $\bar{h}^2$. One minus $\bar{h}^2$ is then the probability that these two locally born offspring x breeding seasons ago were born to different mothers, in which case the relatedness of an individual aged x to a same aged individual is equal to the relatedness of an individual to a random member of the patch at age 1 when the focal individual established onto the patch ($\hat{r}(1)$). The final scenario (bottom row of **[A4]**) considers the case when the individual aged y is older than the individual aged x. In this case the logic is the opposite to the case when the individual aged x is older than the individual aged y.

To calculate $\hat{r}(x)$, the average relatedness of an individual aged x to another individual on the patch, we need to calculate the average relatedness of individuals aged x to all other age classes. Using each possible relatedness between age classes (**[A4]**), we can do this by weighting each age class specific relatedness term by the asymptotic frequencies of the relevant age classes:

$$\hat{r}(x) = \left(\sum^{y<x} f_y [(h(x - y) + k(x - y)\hat{r}(x - y)] \right) + f_x (1 - d)^2 \left(\bar{h}^2 + \left(1 - \bar{h}^2\right)\hat{r}(1) \right) + \left(\sum_{y=x+1}^{y=\omega} f_y [h(y - x) + k(y - x)\hat{r}(1)] \right)$$

[A5]

Deriving $\hat{r}(1)$

To find a general solution for $\hat{r}(1)$, which is the relatedness of an individual aged 1 to another random breeder on the patch, let us consider a case of a population with 3 age classes ($\omega = 3$). Using the logic that $x = 1$ is the first age class and therefore y cannot be younger than x, $\hat{r}(1)$ with 3 age classes becomes:

$$\hat{r}(1) = f_1(1-d)^2\left[\bar{h}^2 + \hat{r}(1)\left(1-\bar{h}^2\right)\right] + \sum_{y=2}^{3} f_y[h(y-x) + k(y-x)]\hat{r}(1)$$

[A6]

Expanding the summation term:

$$\hat{r}(1) = f_1(1-d)^2\left[\bar{h}^2 + \hat{r}(1)\left(1-\bar{h}^2\right)\right] + f_2[h(1) + k(1)\hat{r}(1)] + f_3[h(2) + k(2)\hat{r}(1)]$$

[A7]

Expanding out each term:

$$\hat{r}(1) = f_1(1-d)^2\bar{h}^2 + f_1(1-d)^2\hat{r}(1)\left(1-\bar{h}^2\right) + f_2\,h(1) + f_2k(1)\hat{r}(1) + f_3\,h(2) + f_3k(2)\hat{r}(1)$$

[A8]

Factoring on the RHS by $\hat{r}(1)$:

$$\hat{r}(1) = \hat{r}(1)\left[f_1(1-d)^2\left(1-\bar{h}^2\right) + f_2k(1) + f_3k(2)\right] + f_1(1-d)^2\bar{h}^2 + f_2\,h(1) + f_3\,h(2)$$

[A9]

Re-arranging, and factoring on the LHS by $\hat{r}(1)$:

$$\hat{r}(1)\left[1 - \left[f_1(1-d)^2\left(1-\bar{h}^2\right) + f_2k(1) + f_3k(2)\right]\right] = f_1(1-d)^2\bar{h}^2 + f_2\,h(1) + f_3\,h(2)$$

[A10]

Dividing both sides by $\left[1 - \left[f_1(1-d)^2\left(1-\bar{h}^2\right) + f_2k(1) + f_3k(2)\right]\right]$:

$$\hat{r}(1) = \frac{f_1(1-d)^2\bar{h}^2 + f_2\,h(1) + f_3\,h(2)}{1 - \left[f_1(1-d)^2\left(1-\bar{h}^2\right) + f_2k(1) + f_3k(2)\right]}$$

[A11]

Finally, to generalise for all possible number of age classes, we can re-write **[A11]** as

$$\hat{r}(1) = \frac{f_1(1-d)^2\bar{h}^2 + \sum_{y=2}^{\omega} f_y h(y-1)}{1 - \left[f_1(1-d)^2\left(1-\bar{h}^2\right) + \sum_{y=2}^{\omega} f_y k(y-1)\right]}$$

[A12]

Once we have $\hat{r}(1)$, $\hat{r}(x)$ for all other age classes can be solved recursively.

Appendix B: Analytical Solutions

The effect of a mutant allele that alters age-specific survival in a social population

Let us first consider how, in a resident population with limited dispersal and social interactions, a mutant allele that affects survival at age x will alter the number of class-y offspring of a focal individual aged x. First, the most obvious effect of this allele is to change the individual's probability of survival to the next breeding season, which is $d\dot{p}(x)$. A change in survival will also alter the contributions a focal individual aged x makes to the offspring class, w_{1x}. For example, if the mutant allele increases survival at age x, then there is a greater chance the focal individual survives to age $x+1$, and this subsequently reduces the probability that an offspring at the focal patch after dispersal will establish onto the patch before the next breeding season. Four classes of offspring will exist at the focal patch after dispersal: 1) the offspring of a focal individual aged x, 2) the offspring of other individuals on the patch that exist due to the genotype of a focal individual aged x, 3) the offspring of other individuals on the patch that don't owe their existence to the genotype of a focal individual aged x, and 4) offspring from elsewhere in the population. As we are interested in the inclusive fitness effect of the mutant allele, we must consider the fates of all the offspring that are impacted by the effect of the allele[24].

We can consider the first two sets of offspring together and ask how a change in survival at age x alters the direct and indirect production of offspring of a focal age x individual (working showed below).

$$\frac{dw_{1x}(1,2)}{d\dot{p}(x)} = \dot{F}(x)[(1-d)g(x) + (1-c)d\bar{g}] - \dot{F}(x)[(1-d)g'(x) + (1-c)d\bar{g}] \quad \textbf{[B1]}$$

with $g'(x)$ displaying that the effect of the allele is to alter the probability that the direct and indirect offspring of the individual aged x establish on to the patch. **[B1]** can be worked through and simplified as:

$$\frac{dw_{1x}(1,2)}{d\dot{p}(x)} = \dot{F}(x)(1-d)g(x) + \dot{F}(x)(1-c)d\bar{g} - \dot{F}(x)(1-d)g'(x) - \dot{F}(x)(1-c)d\bar{g}$$

$$= \dot{F}(x)(1-d)g(x) - \dot{F}(x)(1-d)g'(x)$$

$$= \dot{F}(x)(1-d)[g(x) - g'(x)]$$

$$= \dot{F}(x)(1-d)\left[\frac{1-p(x)+(N-1)(1-\bar{p})}{b(x)(1-d)+(N-1)\bar{b}(1-d)+N\bar{b}(1-c)d} - \frac{1-p'(x)+(N-1)(1-\bar{p})}{b(x)(1-d)+(N-1)\bar{b}(1-d)+N\bar{b}(1-c)d}\right]$$

$$= \dot{F}(x)(1-d)\left[\frac{-d\dot{p}(x)}{b(x)(1-d)+(N-1)\bar{b}(1-d)+N\bar{b}(1-c)d}\right]$$

$$= -d\dot{p}(x)\left[\frac{\dot{F}(x)(1-d)}{b(x)(1-d)+(N-1)\bar{b}(1-d)+\ N\bar{b}(1-c)d}\right]$$

Finally, let $\dot{h}(x) = \frac{\dot{F}(x)(1-d)}{b(x)(1-d)+(N-1)\bar{b}(1-d)+\ N\bar{b}(1-c)d}$ be defined as the proportion of offspring at the focal patch after dispersal that are born due the genotype of a focal individual aged x. Note, $\dot{h}(x)$ is different from $h(x)$ (see **Methods Appendix A**), as $h(x)$ does not partition the offspring with respect to inclusive fitness contributions. The relatedness of the indirect offspring has already been discounted in the calculation of $\dot{F}(x)$, and the relatedness of a focal individual to its own offspring is 1, so we can re-write **[B1]** as

$$\frac{dw_{1x}(1,2)}{dp(x)} = -d\dot{p}(x)\dot{h}(x)$$

[B2]

Let us now consider the third set of offspring and ask how a change in survival of a focal individual at age x impacts the offspring of other individuals on the patch that don't owe their existence to the genotype of a focal individual aged x. In the resident population, this contribution is 0. However, an increase in survival of an individual aged x, for example, will reduce the likelihood that any of these offspring that do not disperse will establish onto the patch before the next breeding season. We can write the average number of offspring of all other individuals on the patch, in the presence of a focal individual aged x, that will establish onto the local patch as

$$(N-1)\bar{F}(1-d)g(x)$$

[B3]

The effect of a mutant allele that alters the survival of a focal individual aged x on this expected number of offspring can then be written as

$$\frac{dw_{1x}(3)}{d\dot{p}(x)} = \ (N-1)\bar{F}(1-d)g(x) - \ (N-1)\bar{F}(1-d)g'(x)$$

[B4]

[B4] can then be worked through and simplified as

$$\frac{dw_{1x}(3)}{d\dot{p}(x)} = \ (N-1)\bar{F}(1-d)[g(x)-g'(x)]$$

$$= (N-1)\bar{F}(1-d)\left[\frac{1-p(x)+(N-1)(1-\bar{p})}{b(x)(1-d)+(N-1)\bar{b}(1-d)+\ N\bar{b}(1-c)d}\right.$$
$$\left. - \frac{1-p'(x)+(N-1)(1-\bar{p})}{b(x)(1-d)+(N-1)\bar{b}(1-d)+\ N\bar{b}(1-c)d}\right]$$

$$= (N-1)\bar{F}(1-d)\left[\frac{-d\dot{p}(x)}{b(x)(1-d)+(N-1)\bar{b}(1-d)+\ N\bar{b}(1-c)d}\right]$$

$$= -d\dot{p}(x)\left[\frac{(N-1)\bar{F}(1-d)}{b(x)(1-d)+(N-1)\bar{b}(1-d)+\ N\bar{b}(1-c)d}\right]$$

Similar to the logic above, let $\dot{k}(x) = \frac{(N-1)\bar{F}(1-d)}{b(x)(1-d)+(N-1)\bar{b}(1-d)+\ N\bar{b}(1-c)d}$ be defined as the proportion of offspring at the focal patch after dispersal that are average direct and indirect offspring of all other individuals bar the focal individual aged x. These offspring are related to the focal individual by $\hat{r}(x)$ and so the above becomes

$$\frac{dw_{1x}(3)}{d\dot{p}(x)} = -d\dot{p}(x)\dot{k}(x)\hat{r}(x)$$

[B5]

Given our assumptions of an infinite population, we can assume that relatedness of any individual on a patch to offspring that have dispersed from elsewhere will be equal to zero. Therefore, the relatedness of a focal individual aged x to the proportion of offspring after dispersal that were not born locally on the patch is zero. Thus, there is an overall balance of the effect of the mutant allele on a focal individual of age x's production of newborns weighted on one side by locally produced offspring (with varying relatedness) and on the other side by dispersed offspring. The total effect of a mutant allele that alters age-specific survival on the production of offspring can then be summed as

$$\frac{dw_{1x}}{d\dot{p}(x)} = -d\dot{p}(x)\dot{h}(x)\ - d\dot{p}(x)\dot{k}(x)r_{1x} = -d\dot{p}(x)[\dot{h}(x) + \dot{k}(x)\hat{r}(x)\ ]$$

[B6]

The overall effect (dw_{yx} for all y) of a mutant allele that alters age-specific survival is then shown in **[6]** in the main text.

The effect of a mutant allele that alters age-specific reproduction in a social population

Let us now consider how a mutant allele that affects reproduction at age x will alter the class-y offspring a focal individual aged x in our social population. First, we assume for simplicity that a change in reproduction of a focal individual aged x does not alter the individual's probability of survival to the next breeding season, or its contributions to the survival of other individuals alive on the patch. These are obvious extensions for future iterations of the model (**see Discussion)**. We therefore limit the effects of a change in reproduction to altering the contributions a focal individual aged x makes to the offspring class, w_{1x}. There are four different types of offspring to consider: 1) the offspring of a focal individual aged x that exist due to its own genotype, 2) the offspring of other individuals on the patch that exist due to the genotype of a focal individual aged x, 3) the offspring of other individuals on the patch that don't owe their existence to the genotype of a focal individual aged x, and 4) offspring from elsewhere in the population. Again, as we are interested in the inclusive fitness effect of the mutant allele, we must consider the fates of all the offspring that are impacted by the effect of the allele[24].

The inclusive fitness effects of a mutant allele that causes a change in the direct rate of reproduction of a focal individual aged x for each class of offspring can be displayed as follows:

$$\frac{dw_{1x}(1)}{d\dot{b}(x)} = \dot{b}(x)[(1-d)g(x) + (1-c)d\bar{g}] - \dot{b}'(x)[(1-d)g'(x) + (1-c)d\bar{g}]$$

[B7]

$$\frac{dw_{1x}(2)}{d\dot{b}(x)} = \sum_z T^x_{1,z}(1-d)g(x) - \sum_z T^x_{1,z}(1-d)g'(x)$$

[B8]

$$\frac{dw_{1x}(3)}{d\dot{b}(x)} = (N-1)\bar{F}(1-d)g(x) - (N-1)\bar{F}(1-d)g'(x)$$

[B9]

with prime notation displaying that the explicit effects of the allele. Above, **[B7]** considers the effect of the allele on the focal individual's direct production of offspring, **[B8]** the effect of the allele on the indirect offspring of focal, and **[B9]** the effect on offspring born to other individuals on the patch not due to the genotype of focal, but whom focal might be related to more than the population average (zero). Again, individuals that disperse from elsewhere in the population to the focal patch are assumed to be related to any individual on the patch by zero, and so the inclusive fitness effect of the allele with regards to the fourth class of offspring is also equal to zero. Furthermore, given our assumption of infinite patches, the effect of the allele on the second and third classes of offspring is limited to those offspring which do not disperse *i.e.* compete for a site at the local patch. The simplification of **[B7 – B9]** follows the same logic as **[B1 – B5]**. The resulting derivations are lengthy and so are available in the Supplementary Information. The overall effect of the mutant allele that causes a change in the rate of reproduction of a focal individual aged x is the sum of the effects **[B7 – B9]** and can be expressed as:

$$\frac{dw_{1x}}{d\dot{b}(x)} = d\dot{b}(x)\left[(1-d)g(x)\left[(1-h(x)) - \dot{I}(x) - \dot{k}(x)\hat{r}(x)\right] + (1-c)d\bar{g}\right]$$

[B10]

The overall effect (dw_{yx} for all y) of a mutant allele that alters age-specific reproduction is then shown in **[8]** in the main text.

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Figures

Figure 1

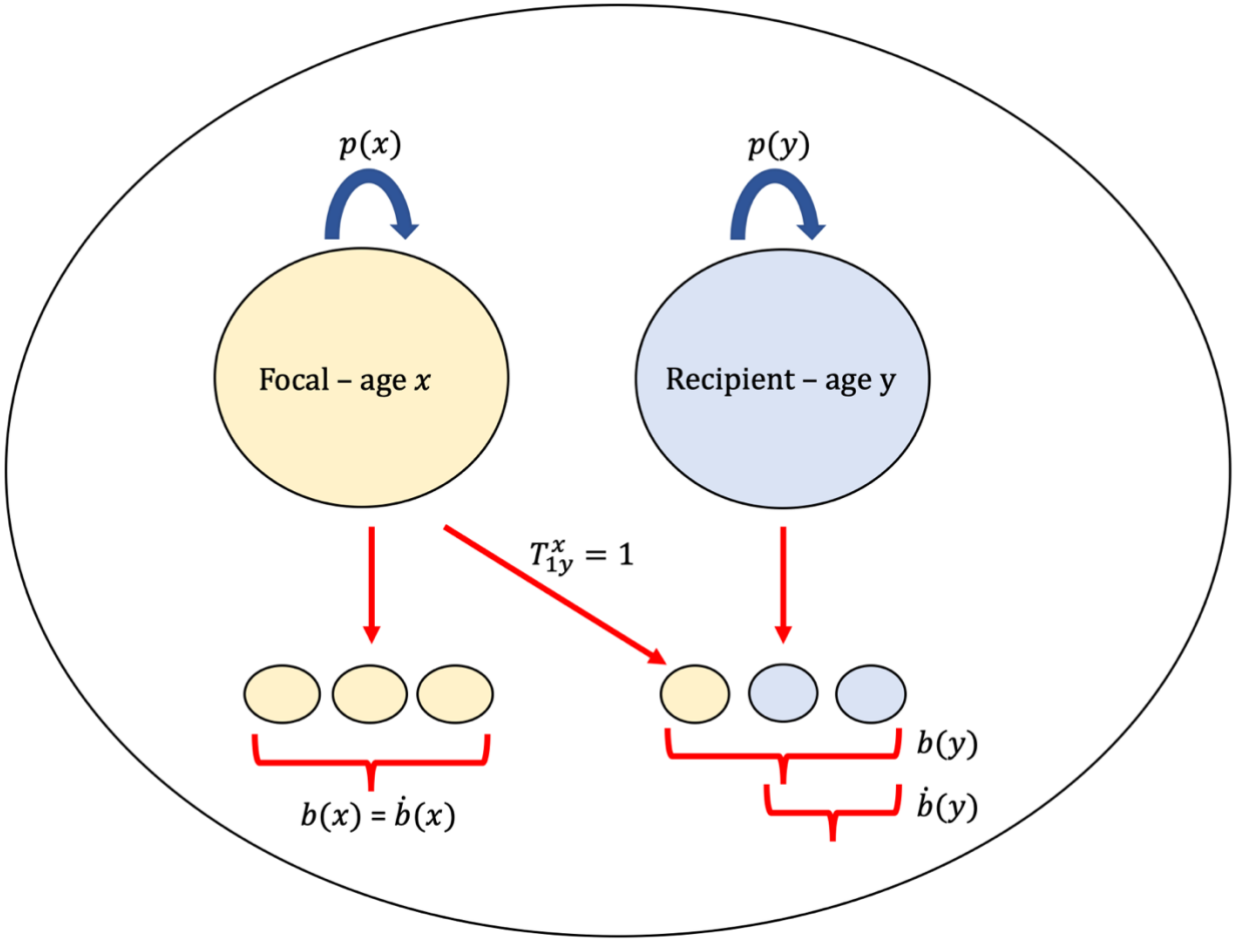
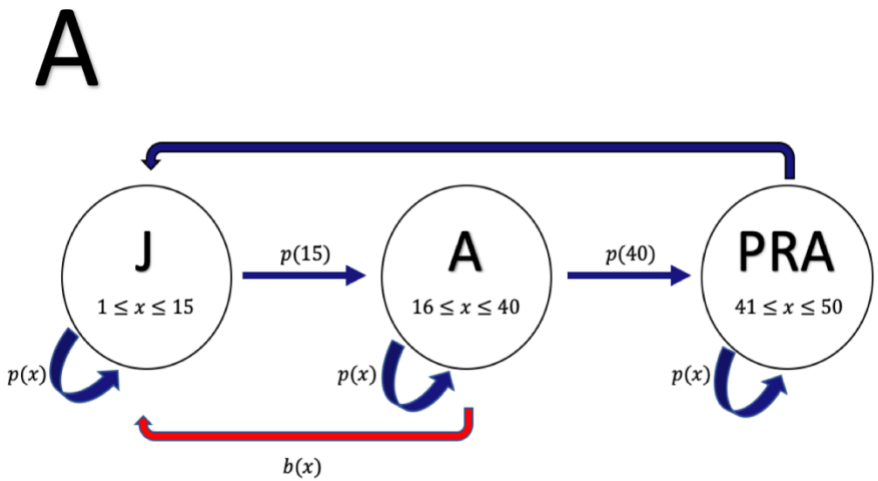
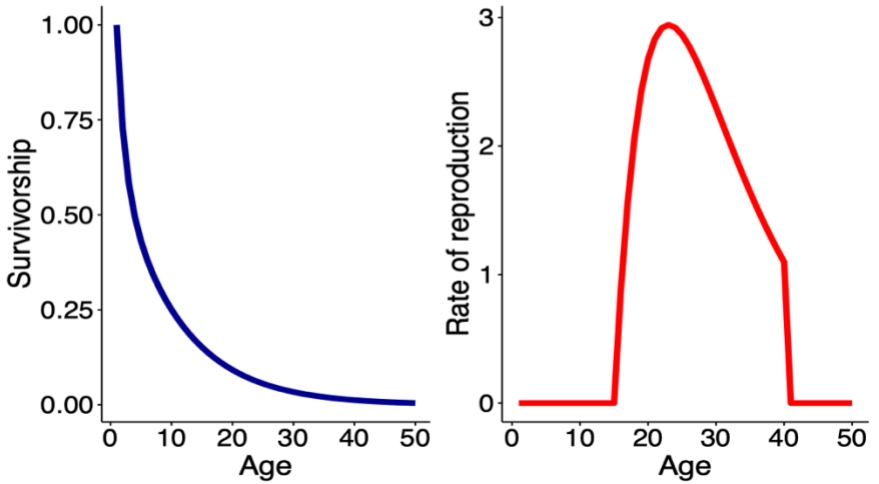
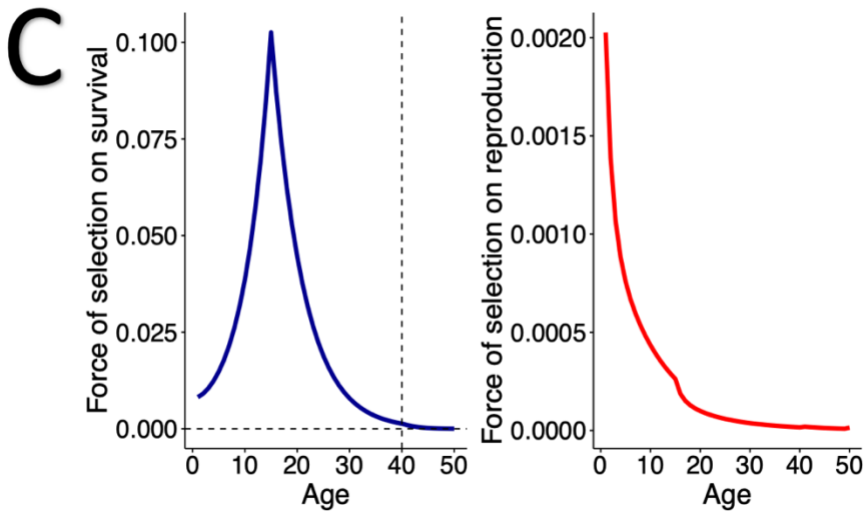


Figure 1. An example of a genetic offspring transfer between two individuals using inclusive fitness. To illustrate transfers, we consider a patch with two individuals, one of age x and the other of age y. The individual aged x has $b(x)$ offspring, survives with probability $p(x)$, and receives no social transfers from other individuals in the population when aged x. We imagine a social behaviour exists whereby the individual aged x contributes to the reproduction of individuals aged y. In this scenario, the individual aged y has $b(y)$ offspring in the current breeding season, but one of these offspring is due to the transfer from the focal individual aged x. Following inclusive fitness logic, the offspring produced due to the social behaviour of the individual aged x is stripped from the inclusive fitness of the individual aged y, leaving $\dot{b}(y)$ as their inclusive fitness contribution to age class 1. The inclusive fitness contribution of the focal individual aged x to age class 1 is $\dot{b}(x) + T_{1y}^{x}\hat{r}(x)$, where $\hat{r}(x)$ represents the relatedness of an individual aged x to the offspring it helped to produce.


A
J
$1 \leq x \leq 15$
$p(15)$
A
$16 \leq x \leq 40$
$p(40)$
PRA
$41 \leq x \leq 50$
$p(x)$
$p(x)$
$p(x)$
$b(x)$


B
Survivorship
1.00
0.75
0.50
0.25
0.00
0 10 20 30 40 50
Age
Rate of reproduction
3
2
1
0
0 10 20 30 40 50
Age


C
Force of selection on survival
0.100
0.075
0.050
0.025
0.000
0 10 20 30 40 50
Age
Force of selection on reproduction
0.0020
0.0015
0.0010
0.0005
0.0000
0 10 20 30 40 50
Age

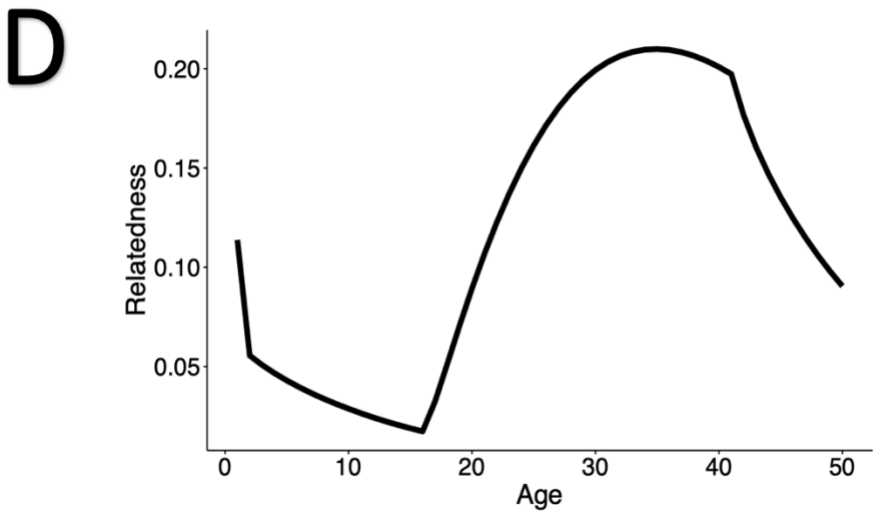

D
Relatedness
0.20
0.15
0.10
0.05
0 10 20 30 40 50
Age

Figure 2. Age specific forces of selection in a social population with post-reproductive help. A) A hypothetical population of iteroparous individuals classified into three life cycle stages: juvenile (J), reproductive adult (A), and post-reproductive adult (PRA). The red arrow from A to J represents the reproduction of adult individuals, whereas the dark blue arrow from PRA to J represents the social contributions from post-reproductive adults to the survival of juveniles. B) The background vital rates of survivorship and reproduction of the model social population. Survival probability at age x is produced from a Siler model (**[11]**) with parameters: $\alpha_1 = 0.4$, $\beta_1 = 0.6$, $\alpha_2 = 0.1$, $\beta_2 = 0$ (See SOM for further details). Reproduction at age x is modelled according to **[13]** with parameters: $\varepsilon = 15$, $\varphi = 0.125$, and $\kappa = 40$ (SOM). C) The forces of selection acting on survival at age x increases during the juvenile period and then decreases but remains above zero in the post-reproductive period. The force of selection acting on reproduction at age x is weaker than the force of selection acting on survival and declines from birth. Other demographic parameters to produce these forces of selection were set to $c = 0$, $d = 0.5$, $N = 4$ and $\omega = 50$. (see **Model** and SOM D) The relatedness of an individual aged x to another random individual on the patch declines throughout the juvenile (pre-reproductive) window, and then increases during adult reproduction before declining again as reproduction ceases.

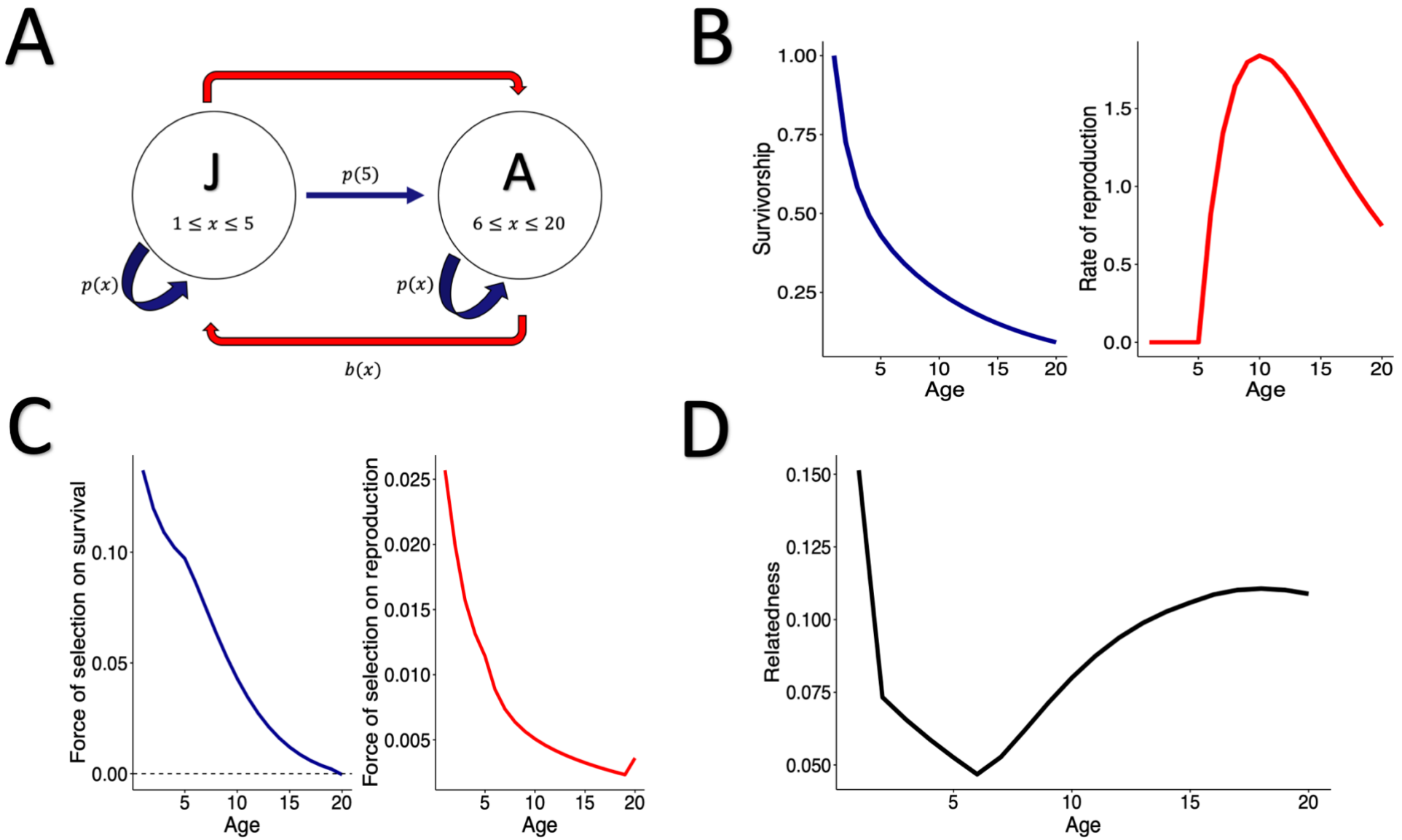

A
J
$1 \leq x \leq 5$
A
$6 \leq x \leq 20$
$p(5)$
$p(x)$
$p(x)$
$b(x)$
B
Survivorship
1.00
0.75
0.50
0.25
5
10
15
20
Age
Rate of reproduction
1.5
1.0
0.5
0.0
5
10
15
20
Age
C
Force of selection on survival
0.10
0.05
0.00
5
10
15
20
Age
Force of selection on reproduction
0.025
0.020
0.015
0.010
0.005
5
10
15
20
Age
D
Relatedness
0.150
0.125
0.100
0.075
0.050
5
10
15
20
Age

Figure 3. Age specific forces of selection in a social population with pre-reproductive help. A) A hypothetical population of iteroparous individuals with two lifecycle stages: juvenile (J) and reproductive adult (A). The red arrow from J to A represents the social contributions from juveniles to the reproduction of adults. Note that here, help is in the currency of reproduction, rather than survival (See Fig. 2A). B) The background vital rates of survivorship and reproduction. Survival at age x is produced from a Siler model (**[11]**) with parameters: $\alpha_1 = 0.4$, $\beta_1 = 0.6$, $\alpha_2 = 0.1$, $\beta_2 = 0$. Rate of reproduction at age x is modelled according to **[13]** with parameters: $\varepsilon = 5$, $\varphi = 0.2$, and $\kappa = 21$. C) The force of selection acting on survival at age x declines from birth. The force of selection acting on reproduction at age x is weaker than the force of selection on survival and also declines from birth but then increases in the final age class. Other demographic parameters to produce these forces of selection were set to $c = 0$, $d = 0.5$ and $N = 4$ and $\omega = 20$. D) The relatedness of an individual aged x to another random individual on the patch declines throughout the juvenile period, and then increases during adult reproduction.

Supplementary Information for

Inclusive fitness forces of selection in an age-structured population

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This PDF file includes:

Supplementary methods for numerical applications and R code for numerical solutions
Further derivations for dw_{1x}
Extended Data Figures 1-4

Other supplementary materials for this manuscript include the following:

R Code for the numerical methods

Supplementary methods for numerical applications

Appendix C: Numerical application methods

To compute the inclusive fitness matrix (**W)**, we first model the propensity of each age class to contribute to the survival and reproduction of each age class, including its own. These genetic offspring transfer propensities can be conceptualised as two different matrices, one for survival and one for reproduction. The entry $\mathbf{T}_{yx}$ represents the relative propensity for age class x to contribute to the survival or reproduction of age class y. This propensity takes the functional form of

$$\mathbf{T}_{yx} = T(x, y, C_x, C_y)$$

[C1]

Where C represents the stage of each age class, which could be, for example, pre-reproductive (juveniles), mature reproductive, mature non-reproductive, or post-reproductive. The form of **[C1]** allows for the relative propensity to be a function of the age and stage of both actor and recipient. Here, for simplicity, for our population with post-reproductive survival (Fig. 1) we input a 1 into each element of the **T** matrix for survival in which the column represents a post-reproductive age class and the row represents a juvenile age class. This means that every post-reproductive age class has the same relative propensity to contribute to the survival of each juvenile age class. Every other element in **T** (survival) is set to 0, and every element in **T** (reproduction) is 0. On the contrary, for our population with pre-reproductive help, we input a 1 into each element of the **T** matrix for reproduction with a column representing a pre-reproductive age class and a column representing a reproductive age class. This means every pre-reproductive age class has the same relative propensity to contribute to the reproduction of each reproductive age class. Every other element in **T** (reproduction) = 0, and every element in **T** (survival) = 0.

The next step is to model the proportions of age-specific survival and reproduction that are due to the social environment. The total social transfers received by age class x is $\sum_z T^z_{x+1,x}$ for survival at age x and $\sum_z T^z_{1,x}$ for reproduction at age class x, which are modelled as proportions of the background demographic rates $p(x)$ and $b(x)$. These proportions may vary by age and stage, and different age classes within the same stage could have different proportions stripped from their survival and reproduction. For example, very young juveniles might owe their survival more to other members of the group than older juveniles. Here, we model cases were 10, 20 and 30% of juvenile survival (Fig.1; Extended Data Fig. 1 and 2) or 10, 20 and 30% of adult reproduction is due to the social environment. These contributions are independent of age within the stage classification *i.e.* the same proportion is stripped from all juvenile or adult stage classes within the same iteration of the model.

We can then quantify indirect fitness contributions by distributing the stripped genetic offspring equivalents that are the result of the social environment. Specifically, a focal individual aged x's genetic offspring contribution to the survival of age class z is:

$$T^x_{z+1,z} = (N-1) f_z t^x_{z+1,z} \hat{r}(x)$$

[C2]

where $(N-1)f_z$ is the expected number of individuals alive on the patch aged z with the focal individual aged x, $\hat{r}(x)$ is the relatedness of a focal individual aged x to a random breeder on the patch, and $t^x_{z+1,z}$ represents the fraction of the total social contributions to the survival of age class z that are due to age class x, and can be written more explicitly as

$$t^x_{z+1,z} = \sum_j T^j_{z+1,z} \frac{f_x \mathbf{T}_{zx}}{\sum_j f_j \mathbf{T}_{zj}}$$

[C3]

The same logic applies to reproductive contributions. For each age class, these indirect genetic contributions are compiled into the inclusive fitness matrix **W**, along with the direct components of survival ($\dot{p}(x)$) and reproduction ($\dot{b}(x)$). The dominant left eigenvector of **W** is equal to the vector of inclusive reproductive value for each age class. The inclusive fitness forces of selection acting on a mutant allele that alters the rate of survival between age x and $x+1$ or rate of reproduction at age x can then be calculated according to **[7]** and **[9]** in the main text. Code for the numerical solutions explored in the main text is available in a separate file.

Further derivations for dw_{1x}

The inclusive fitness effect of a mutant allele that causes a change in the direct rate of reproduction of a focal individual aged age *x* on it's direct fitness can be displayed as

$$\frac{dw_{1x}(1)}{d\dot{b}(x)} = \dot{b}(x)[(1-d)g(x) + (1-c)d\bar{g}] - \dot{b}'(x)[(1-d)g'(x) + (1-c)d\bar{g}]$$

[D1]

The following working displays the simplification of **[D1]:**

$$\frac{dw_{1x}(1)}{d\dot{b}(x)} = \dot{b}(x)(1-d)g(x) + \dot{b}(x)(1-c)d\bar{g} - \dot{b}'(x)(1-d)g'(x) - \dot{b}'(x)(1-c)d\bar{g},$$

$$\frac{dw_{1x}(1)}{d\dot{b}(x)} = \dot{b}(x)(1-d)g(x) - \dot{b}'(x)(1-d)g'(x) + (1-c)d\bar{g}[\dot{b}(x) - \dot{b}'(x)],$$

$$\frac{dw_{1x}(1)}{d\dot{b}(x)} = (1-d)\left[\frac{\dot{b}(x)[(1-p(x)+(N-1)\bar{p}]}{b(x)(1-d)+(N-1)\bar{b}(1-d)+N\bar{b}(1-c)d} - \frac{\dot{b}'(x)[(1-p(x)+(N-1)\bar{p}]}{b'(x)(1-d)+(N-1)\bar{b}(1-d)+N\bar{b}(1-c)d}\right] + (1-c)d\bar{g}[b(x)-b'(x)]$$

The terms inside the brackets are manipulated using algebraic subtraction and, after simplification, results in:

$$\frac{dw_{1x}(1)}{d\dot{b}(x)} = (1-d)\left[\frac{[b(x)-b'(x)]\,[(1-p(x)+(N-1)\bar{p}]\left[(N-1)\bar{b}(1-d)+N\bar{b}(1-c)d\right]}{\left[b(x)(1-d)+(N-1)\bar{b}(1-d)+N\bar{b}(1-c)d\right]\left[b'(x)(1-d)+(N-1)\bar{b}(1-d)+N\bar{b}(1-c)d\right]}\right] + (1-c)d\bar{g}[b(x)-b'(x)]$$

Further simplification results in the expression:

$$\frac{dw_{1x}(1)}{d\dot{b}(x)} = (1-d)[b(x)-b'(x)]\left[\frac{[(1-p(x)+(N-1)\bar{p}]\left[(N-1)\bar{b}(1-d)+N\bar{b}(1-c)d\right]}{\left[b(x)(1-d)+(N-1)\bar{b}(1-d)+N\bar{b}(1-c)d\right]\left[b'(x)(1-d)+(N-1)\bar{b}(1-d)+N\bar{b}(1-c)d\right]}\right] + (1-c)d\bar{g}[b(x)-b'(x)]$$

which using definitions in **Methods Appendix B** is equal to:

$$\frac{dw_{1x}(1)}{d\dot{b}(x)} = (1-d)[b(x)-b'(x)][g(x)(1-h(x)] + (1-c)d\bar{g}[b(x)-b'(x)]$$

which can finally be simplified to:

$$\frac{dw_{1x}(1)}{d\dot{b}(x)} = d\dot{b}(x)[(1-d)g(x)(1-h(x)) + (1-c)d\bar{g}]$$

[D2]

The inclusive fitness effect of a mutant allele that causes a change in the direct rate of reproduction of a focal individual aged age *x* on the fates of offspring it indirectly produced through the reproduction of others can be displayed as

$$\frac{dw_{1x}(2)}{d\dot{b}(x)} = \sum_z T_{1,z}^x (1-d)g(x) - \sum_z T_{1,z}^x (1-d)g'(x)$$

[D3]

The following working displays the simplification of **[D3]:**

$$\frac{dw_{1x}(2)}{d\dot{b}(x)} = \sum_z T_{1,z}^x (1-d)\left[\frac{[(1-p(x)+(N-1)\bar{p}]}{b(x)(1-d)+(N-1)\bar{b}(1-d)+\ N\bar{b}(1-c)d} - \frac{[(1-p(x)+(N-1)\bar{p}]}{b'(x)(1-d)+\ (N-1)\bar{b}(1-d)+\ N\bar{b}(1-c)d}\right]$$

The terms inside the brackets are manipulated using algebraic subtraction and, after simplification, results in:

$$\frac{dw_{1x}(2)}{d\dot{b}(x)} = \sum_z T_{1,z}^x (1-d)\left[\frac{[b(x)-b'(x)]\ [(1-p(x)+(N-1)\bar{p}](1-d)}{[b(x)(1-d)+(N-1)\bar{b}(1-d)+\ N\bar{b}(1-c)d][b'(x)(1-d)+\ (N-1)\bar{b}(1-d)+\ N\bar{b}(1-c)d]}\right]$$

Further simplification and using definitions in **Methods Appendix B** results in the expression:

$$\frac{dw_{1x}(2)}{d\dot{b}(x)} = d\dot{b}(x)(1-d)\left[\frac{[(1-p(x)+(N-1)\bar{p}]\left[\sum_z T_{1,z}^x (1-d)\right]}{[b(x)(1-d)+(N-1)\bar{b}(1-d)+\ N\bar{b}(1-c)d][b'(x)(1-d)+\ (N-1)\bar{b}(1-d)+\ N\bar{b}(1-c)d]}\right]$$

which can finally be simplified to:

$$\frac{dw_{1x}(2)}{d\dot{b}(x)} = -\,d\dot{b}(x)\left[(1-d)g(x)\dot{l}(x)\right]$$

[4]

Finally, the inclusive fitness effect of a mutant allele that causes a change in the direct rate of reproduction of a focal individual aged age *x* on the fates of other offspring born on the patch can be written as

$$\frac{dw_{1x}(3)}{d\dot{b}(x)} = (N-1)\bar{F}(1-d)g(x) - (N-1)\bar{F}(1-d)g'(x)$$

[D5]

The following working displays the simplification of **[D5]:**

$$\frac{dw_{1x}(3)}{d\dot{b}(x)} = (N-1)\bar{F}(1-d)\left[\frac{[(1-p(x)+(N-1)\bar{p}]}{b(x)(1-d)+(N-1)\bar{b}(1-d)+N\bar{b}(1-c)d} - \frac{[(1-p(x)+(N-1)\bar{p}]}{b'(x)(1-d)+(N-1)\bar{b}(1-d)+N\bar{b}(1-c)d}\right]$$

The terms inside the brackets are manipulated using algebraic subtraction and, after simplification, results in:

$$\frac{dw_{1x}(3)}{d\dot{b}(3)} = (N-1)\bar{F}(1-d)\left[\frac{[b(x)-b'(x)]\,[(1-p(x)+(N-1)\bar{p}](1-d)}{[b(x)(1-d)+(N-1)\bar{b}(1-d)+N\bar{b}(1-c)d][b'(x)(1-d)+(N-1)\bar{b}(1-d)+N\bar{b}(1-c)d]}\right]$$

Further simplification and using definitions in **Appendix B** results in the expression:

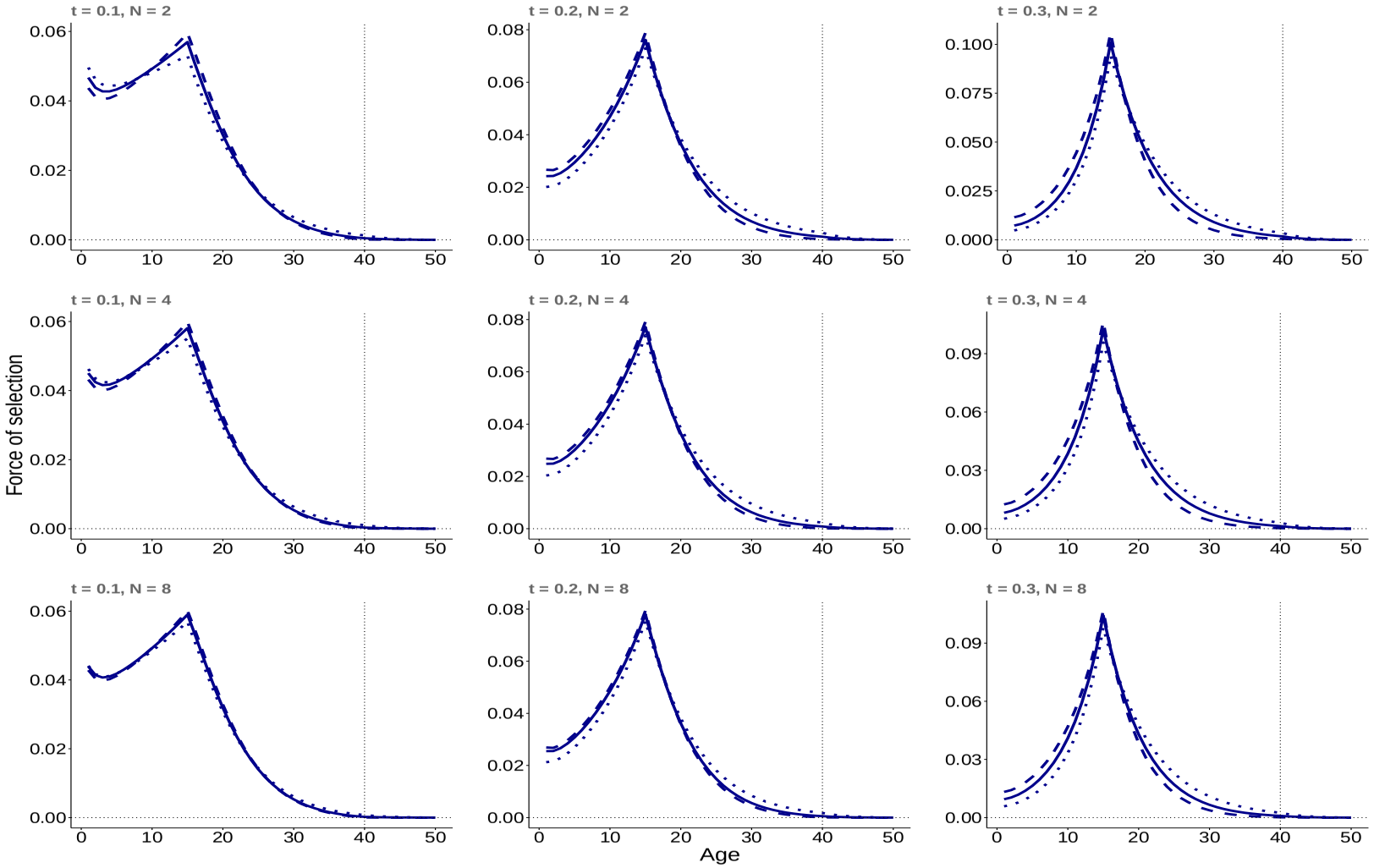
$$\frac{dw_{1x}(3)}{d\dot{b}(x)} = d\dot{b}(x)(1-d)\left[\frac{[(1-p(x)+(N-1)\bar{p}][(N-1)\bar{F}(1-d)]}{[b(x)(1-d)+(N-1)\bar{b}(1-d)+N\bar{b}(1-c)d][b'(x)(1-d)+(N-1)\bar{b}(1-d)+N\bar{b}(1-c)d]}\right]$$

which can finally be simplified to:

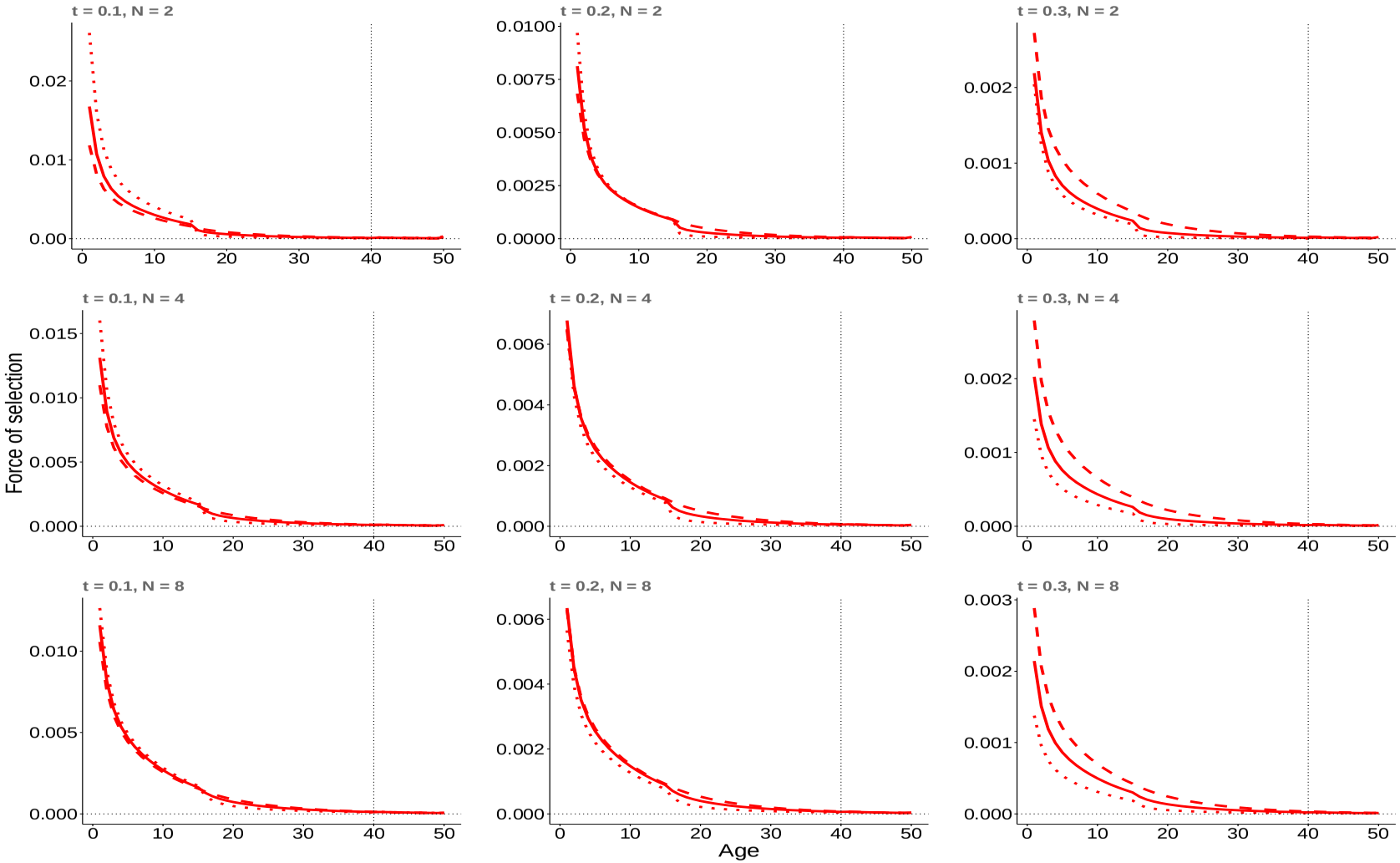
$$\frac{dw_{1x}(3)}{d\dot{b}(x)} = -d\dot{b}(x)[(1-d)g(x)\dot{k}(x)]$$

[D6]

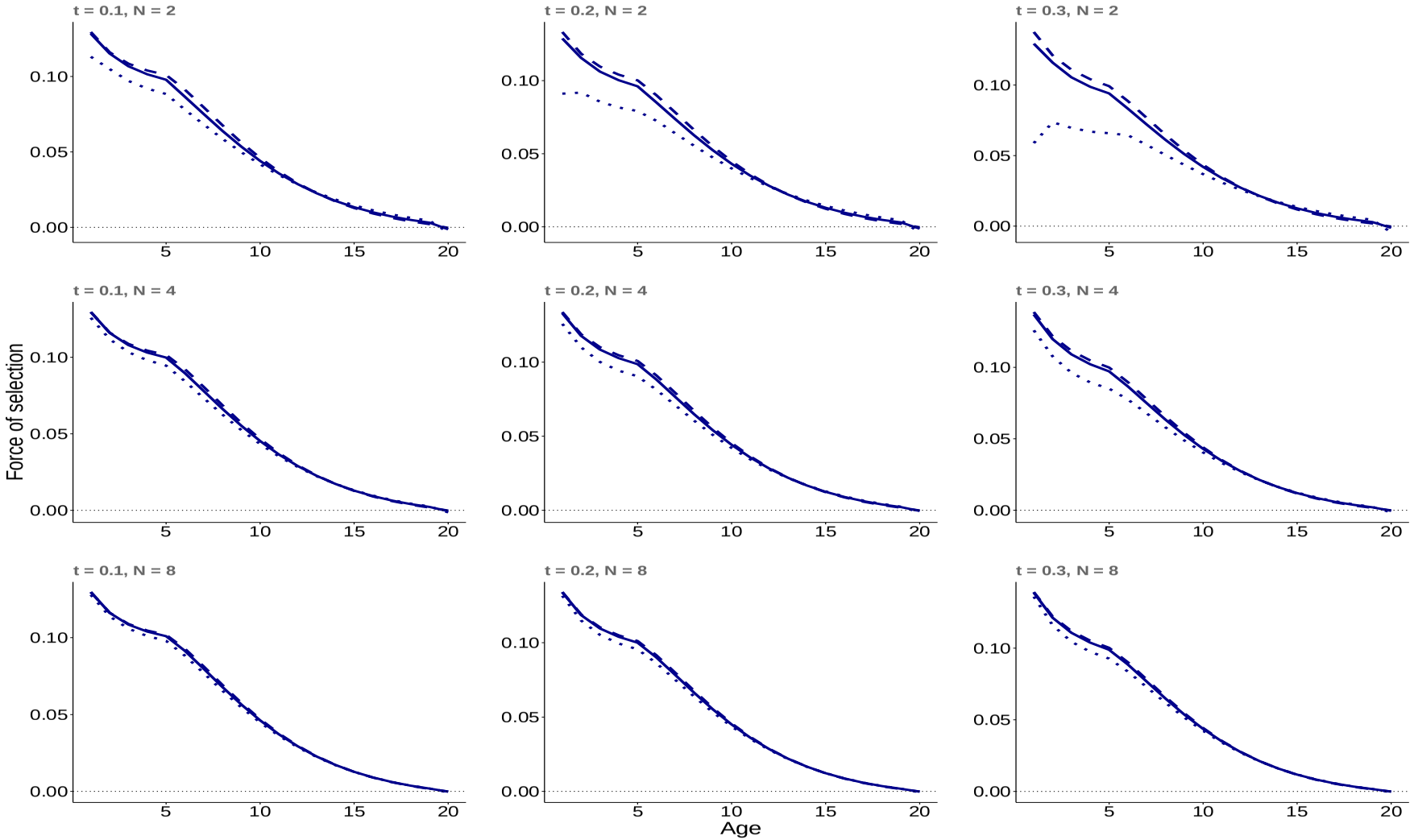
These offspring are related to the focal individual by $\hat{r}(x)$. Equations **[D2]**, **[D4]**, and **[D6]** can then be summed to give **[B10]** in **Appendix B**.



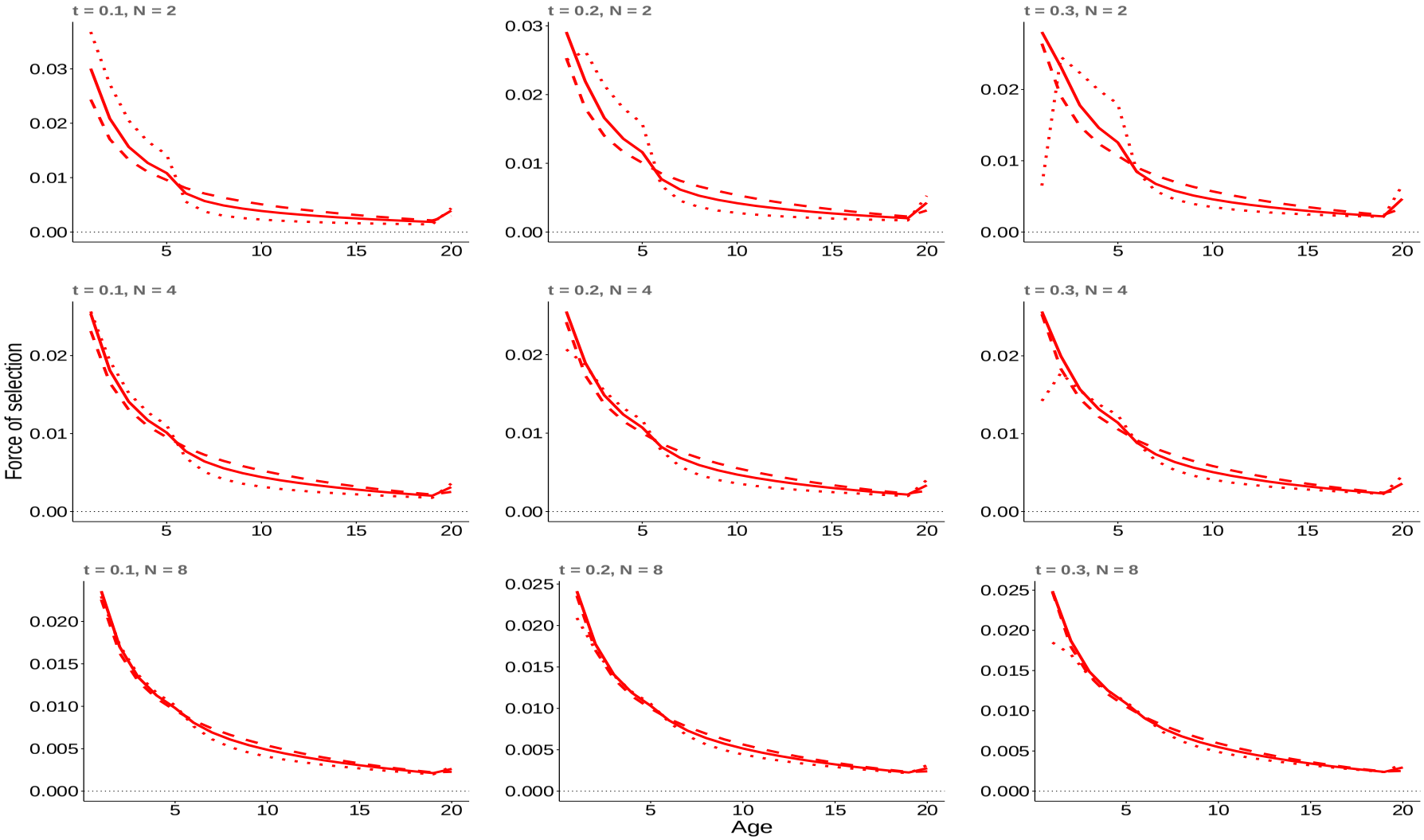
Extended Data Figure 1. The force of selection acting on survival rate at age x in a population with post-reproductive survival (**see Fig. 1**) as a function of the *(i)* the magnitude of the social contribution from post-reproductive individuals to the survival of juveniles, where t = the fraction of juvenile survival that is due to post-reproductive individuals, *(ii)* the number of individuals on the patch (N) and the juvenile dispersal rate in the population: 0.2 (dotted line), 0.5 (continuous line), and 0.8 (broken line). The vertical dotted line at age 40 represents the age at which reproduction ceases.



Extended Data Figure 2. The force of selection acting on reproduction at age x in a population with post-reproductive survival (**see Fig. 1**) as a function of the *(i)* the magnitude of the social contribution from post-reproductive individuals to the survival of juveniles, where t = the fraction of juvenile survival that is due to post-reproductive individuals, *(ii)* the number of individuals on the patch (N) and the juvenile dispersal rate in the population: 0.2 (dotted line), 0.5 (continuous line), and 0.8 (broken line). The vertical dotted line at age 40 represents the age at which reproduction ceases.



Extended Data Figure 3. The force of selection acting on survival at age x in a population with pre-reproductive helpers (**see Fig. 2**) as a function of the *(i)* the magnitude of the social contribution from pre-reproductive individuals to the reproduction of reproductive-aged adults, where t = the fraction of reproduction that is due to pre-reproductive individuals, *(ii)* the number of individuals on the patch (N) and the juvenile dispersal rate in the population: 0.2 (dotted line), 0.5 (continuous line), and 0.8 (broken line).



Extended Data Figure 4. The force of selection acting on reproduction at age *x* in a population with pre-reproductive helpers (**see Fig. 2**) as a function of the *(i)* the magnitude of the social contributions from pre-reproductive individuals to the reproduction of reproductive-aged adults, where t = the fraction of reproduction that is due to pre-reproductive individuals, *(ii)* the number of individuals on the patch (N) and the juvenile dispersal rate in the population: 0.2 (dotted line), 0.5 (continuous line), and 0.8 (broken line)

5 | Higher relatedness is associated with reduced adult lifespan and weaker reproductive senescence in social species

Abstract

In social organisms, natural selection seeks to optimise inclusive fitness. When social interactions between individuals vary across age classes, and group-level characteristics of social species alter the distribution of mortality and reproduction across age classes, total inclusive fitness contributions will vary across age classes. Inclusive fitness forces of selection can then be derived that predict variation in lifespan and rates of senescence. Here, we test for variation in expected adult lifespan and rates of senescence across vertebrates that exhibit alloparental care. In particular, we test for how (a) relatedness between group members, (b) the probability of breeding in the group one is born into, (c) the level of reproductive skew within the group, and (d) the size of the helping effect from alloparents drives variation in these key life history variables. We find that, after accounting for body mass and phylogenetic relationships between species, groups with a higher mean relatedness show lower expected adult lifespan and a distribution of reproduction biased towards older age classes (*i.e.* more negative senescence). Our results indicate that group level characteristics of social organisms explain an important, previously unexplored, source of variation in lifespan and senescence.

Introduction

The ideas of kin selection and inclusive fitness theory have helped to explain the evolution of diverse social behaviours (Bourke 2011), organismal design (West & Gardner 2013), and major evolutionary transitions (Maynard Smith & Szathmary 1995; Bourke 2019). Despite social evolution theory being utilised extensively across evolutionary biology since Hamilton's seminal work (1964a;1964b), it was not until 2007 that the ideas of kin selection and inclusive fitness were integrated into the evolutionary theories of ageing (Bourke 2007). The author suggested that a group-living individual's probability of death and rate of reproduction could be considered to be social traits if these traits have an impact on the fitness of other individuals. Bourke's insight, the importance of social integration for health and well-being in humans (Snyder-Maclker *et al.* 2020), and the late-life changes in social behaviour across animals (Siracusa *et al.* 2022; Albery *et al.* 2022), suggest social interactions may be an important driver of unexplained variation in lifespan and rates of senescence across species.

The majority of studies exploring the relationship between social interactions and ageing have focused on two key areas. The first set of studies have focused on cooperatively breeding species, those that exhibit alloparental care, *i.e.* individuals other than the parents also help to raise offspring (Koenig & Dickinson 2016). Within these species, the phenomena of 'load-lightening' has been revealed (Crick 1992). Load-lightening occurs when helpers in cooperatively-breeding species can reduce the rate of senescence and/or extend the lifespan of breeders by alleviating the energetic costs of raising offspring. There is strong evidence across cooperatively breeding species, in particular birds and mammals, that helpers can indeed reduce the rates of senescence or increase lifespan in breeders (Berger *et al.* 2018; Hammers *et al.* 2019; Downing *et al.* 2021).

The second set of studies have focused more generally on the relationship between sociality and longevity, often comparing cooperative and non-cooperative species. Here, theoretical expectations and empirical evidence are both mixed. A model of cooperation in age-structured populations suggested that longer life, and importantly overlapping generations, can overcome competition between relatives and favour cooperation in populations with limited dispersal (Taylor & Irwin 2000). This result suggests that longer lifespan favours sociality. An analysis that compared the difference in lifespan between cooperative and non-cooperative bird species found that the ancestral condition in cooperative lineages was longer lifespan, thus supporting this directionality (Downing *et al.* 2015). However, a recent study has suggested that age-specific queuing of reproduction could be a reinforcing factor that extends lifespan in cooperative breeders (Kreider *et al.* 2022). This hypothesis is due to strengthened selection for extended lifespan to access reproduction in later age classes. Additionally, there is mixed evidence as to the directionality of the relationship between sociality and longevity in mammals (Healy 2015; Thorley 2020; Lucas & Keller 2020).

A limitation of the studies exploring the interplay of sociality and senescence is that they only compare cooperative *vs* non-cooperative species (Lucas & Keller 2020; Korb & Heinze 2021). These studies do not consider the remarkable variation in the formation and structure of groups, and the types of social interactions that occur within cooperative species. Many groups form by offspring delaying dispersal and remaining with their parents (Koenig & Dickinson 2016); however, others form when unrelated (*e.g.*, groove-billed anis (*Crotophaga sulcirostris*); Bowen *et al.* 1989) or related adults (*e.g.* the edible dormouse (*Glis glis*); Marin & Pilastro 1994) come together and breed communally. Groups can contain multiple breeding individuals of both sexes (*e.g.* the banded mongoose (*Mungos mungo)*; Cant *et al.* 2016); alternatively, reproduction may be monopolised by a single individual of each sex (*e.g.* the meerkat (*Suricata*

suricatta); Thorley *et al.* 2019). In many 'helper-at-the-nest' systems, juveniles can increase the reproductive success of breeders, whilst in plural breeding systems adult individuals may benefit each other when communally breeding, and in some groups it is the oldest individuals that direct help towards juveniles (*e.g.* the killer whale (*Orcinus orca*); Nattrass *et al.* 2019). This variation of group structure and range of social interactions between age classes are yet to be considered when predicting variation in lifespan and rates of senescence across social organisms.

In **Chapter 4**, I built a theoretical framework to quantify inclusive fitness forces of selection. The model does not consider the invasion of a cooperative allele. Instead, social interactions between individuals are modelled as part of the framework in the same currency as survival and reproduction as 'transfers'. Transfers represents harmful or helpful effects from an individual in one age class to the survival or reproduction of an individual in another age class (that could include its own). From the perspective of a focal individual aged x, the transfers the individual receives from others towards it's survival and reproduction are stripped to leave direct fitness, and it's inclusive reproductive value is the sum of this direct fitness and any indirect fitness it gains from transfers to other individual's survival and reproduction. The inclusive fitness forces of selection on survival and reproduction at age x are then a balance of the inclusive reproductive value of the focal individual at age x, and the inclusive reproductive values of the individuals affected by a change to the probability of survival and rate of reproduction of the individual aged x (**Chapter 4)**. Here, we develop and test hypotheses from the model in **Chapter 4** and elsewhere on the rates of senescence and expected lifespan across species that exhibit alloparental care.

Hypothesis 1 – A higher probability of inheritance by philopatric offspring will be associated with a lower adult life expectancy

The first hypothesis considers the indirect force of selection that acts on survival in group-living organisms (Ronce & Promislow 2010; Kreider *et al.* 2022; **Chapter 4**). The inclusive fitness force of selection acting on survival can be written:

$$S_p(x) = \frac{d\dot{p}(x)}{d\delta} \frac{f_x(v_{x+1} - \left[\dot{h}(x) + \dot{k}(x)\hat{r}(x)\right]v_1)}{\mathbf{f}.\mathbf{v}} \tag{1}$$

where the term $\left[\dot{h}(x) + \dot{k}(x)\hat{r}(x)\right]v_1$ measures the indirect force of selection acting against increased individual survival, which is weighted by the inclusive reproductive values of, and relatedness to, newborn offspring on the patch. This effect should be stronger when relatedness is higher, and when philopatric individuals can inherit a breeding position. We therefore predict that species should exhibit reduced lifespans when within-group relatedness and the likelihood of inheritance is high.

Hypothesis 2 – Higher reproductive skew will be associated with a bias of reproduction towards older age classes (*i.e.* more negative senescence)

Reproductive skew measures the distribution of breeding among individuals in a group (Cant 1998). The more biased reproduction is towards fewer individuals, the higher the measure of skew. When there is high reproductive skew in group-living organisms, it is often the oldest individuals of each sex that monopolises breeding opportunities (Shreeves & Field 2002). When this is the case, the distribution of reproduction is shifted towards older age classes, and so the weakening force of selection acting on reproduction should be less pronounced. On the

other hand, in plural breeders, where multiple individuals can co-currently breed, individuals are typically able to reproduce from the age of maturity. Therefore, the weakening force of selection acting on reproduction should be more pronounced. We therefore predict that species in which a single individual of a sex reproduces at any one time should show lower rates of reproductive senescence than groups in which multiple individuals of the same sex can breed at the same time.

Hypothesis 3 – A stronger effect of helpers towards breeder reproductive success will be associated with accelerated actuarial senescence but decreased reproductive senescence later in life

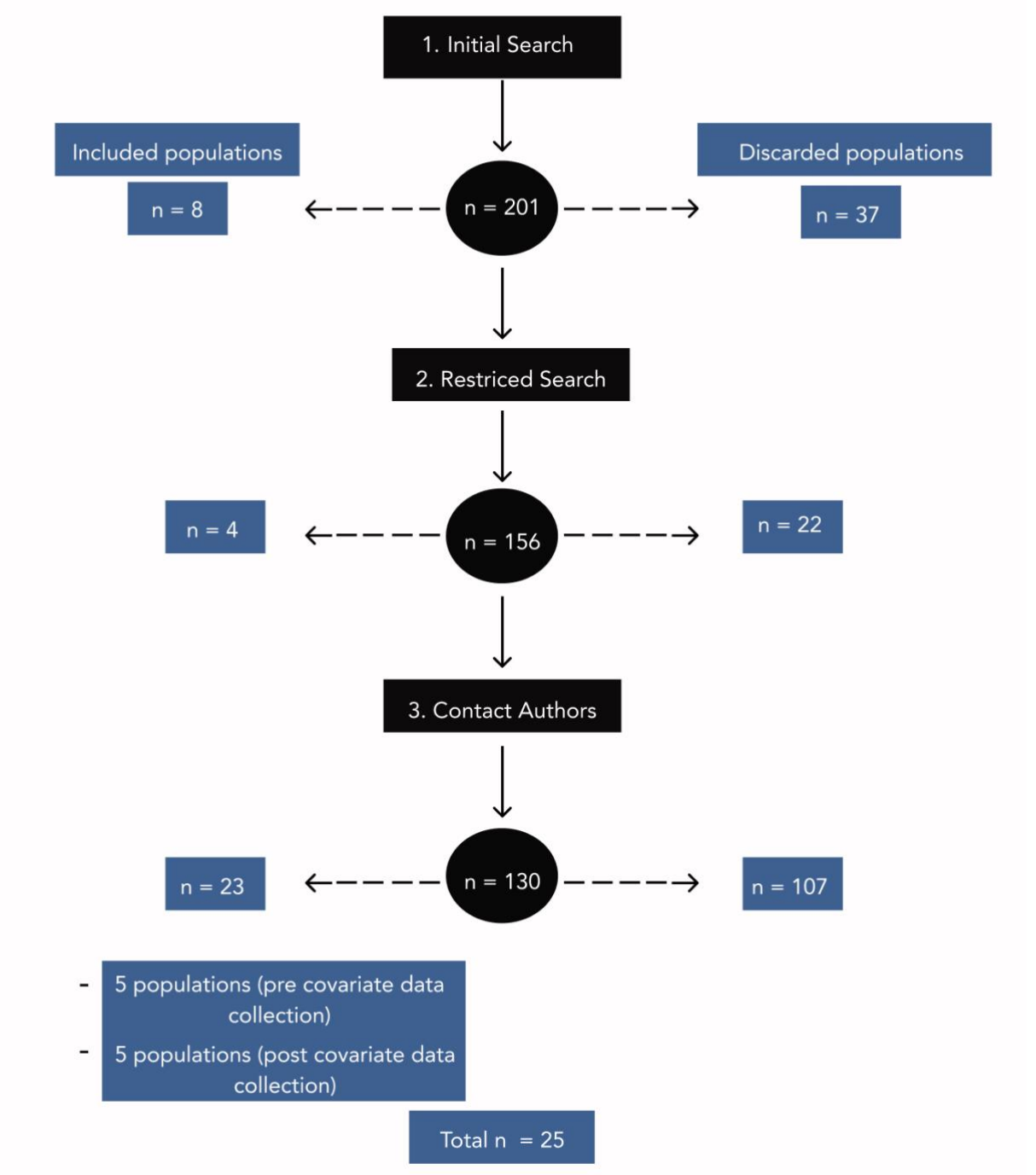
We ran numerical explorations of the model in **Chapter 4** to generate predictions for how helping in early life should alter actuarial and reproductive senescence across our species. We found that the greater the magnitude of help from juveniles to breeder reproduction, the weaker the force of selection on both survival and reproduction at early ages. This is due to the indirect negative consequences of increased individual survival and reproduction, through increasing competition for related offspring the helpers have just contributed to producing via their helping efforts. We therefore predicted that helper-at-the-nest-systems in our dataset should show accelerated rates of actuarial senescence when the magnitude of help *per helper* is higher, and there is higher relatedness between the helper and the helped individual (*i.e.* greater indirect fitness accrued). On the contrary, weaker selection at earlier ages on reproduction should bias reproduction towards older ages. We therefore expected species with a higher magnitude of help to show more pronounced actuarial senescence, but weaker reproductive senescence.

Materials and Methods

Demographic data collection

To test our set of hypotheses, we began by conducting keyword searches using Web of Science with the search term format '*i* AND aging OR *i* AND ageing OR *i* AND senescence OR *i* AND life table OR *i* AND life-table OR *i* AND demography OR *i* AND survival', where *i* = 1) 'alloparental care', 2) 'eusociality', 3) 'communal breeding', 4) 'cooperative breeding and 5) 'cooperatively breeding'. In total, the five search terms produced 2401 results on Web of Science. Of these, 2042 were excluded after reading the abstract, with the remaining 359 being filtered through to a full reading of the paper. These 359 papers contained 201 unique populations (Fig. 1; Table. S1). Thirty-seven populations were removed at this stage because there was no robust evidence for alloparental care in the species or the study did not reference a longitudinal study (Table. S1). The initial search terms recovered eight populations across seven species with both age-specific estimates of actuarial and reproductive life table data: the Seychelles warbler (*Acrocephalus sechellensis*; Cousin Island), the Red wolf (*Canis rufus*; Aligator River National Wildlife Refuge), the African wild dog (*Lyacon pictus*; Selous Game Reserve & Hluhluwe-iMfolozi Park), the Superb fairy-wren (*Malurus cyaneus*; Australian National Botanic Gardens), the Western bluebird (*Siala* Mexicana; Oregon) and the Meerkat (*Suricata suricatta*; Kalahari). Additionally, the search terms recovered the population of long-tailed tits (*Aegithalos caudatus*; Rivelin Valley) for which we had longitudinal estimates of age-specific survival and reproduction (Roper *et al.* 2022).

Fig. 1 | PRISMA flow chart describing the data collection filtering process used to collect life table data for species with alloparental care. An initial set of search terms resulted in 2401 papers from Web of Science. These were filtered down to 25 populations of species that: *(i)* exhibit alloparental care, *(ii)* had age-specific estimates of reproduction and mortality in the wild, and *(iii)* had data available for group-level characteristics for at least one of the models we ran in our analyses. See Methods and Extended Data Table 1 for more information.



After conducting this initial filtering, we split the remaining 156 populations recovered from the search into two categories. These populations either had estimates of actuarial life table data but not reproductive data (and *vice versa*), or did not have data available in the papers read, but the study referenced a longitudinal study of the population from which data were potentially available. These populations were filtered through to a second set of restricted search terms. The first term searched generally for any demography-based studies for the species, and took the form:

"Species Name AND demography OR Common Name AND demography OR Species Name AND demographic OR Common Name AND demographic OR Species Name AND mark-recapture OR Common Name AND mark-recapture"

The second set of restricted search terms looked explicitly for studies on the species at the population location identified in the initial searches, and took the form:

"Species Name AND Population Name AND survival OR Species Name AND Population Name AND reproduction OR Common Name AND Population Name AND survival OR Common Name AND Population Name AND reproduction"

If either of the restrictive searches returned a paper from the initial searches, it was discarded and not re-read. Using this approach, we were able to obtain actuarial and reproductive life table data for two further populations: the Eurasian badger (*Meles meles*; Whytham Woods) and the Giant otter (*Pteronura brasilliensis*; Manu National Park). This approach resulted in a total of 154 remaining populations after the restricted searches. Of these, we excluded 22 populations as there was no clear evidence of a wild longitudinal study. For the remaining

populations, we attempted to contact authors to obtain unpublished data on actuarial and/or reproductive life table estimates.

Overall, the 201 unique populations from the initial search term were filtered down to 35 populations with either actuarial, reproductive, or both life table estimates (Fig. 1; Table S1). For the Cape penduline tit (*Anthoscopus minutus*; Koeberg Nature Reserve) and Campo flicker (*Colaptes campestris*; Fazenda Agua Limpa), repeated sampling of individuals was limited to only some of the age classes across the species' lifespan, and so rates of senescence and expected lifespan could not be reliably estimated. We therefore removed these populations from our analyses. We also removed the Eurasian badger (*Meles meles*: Wytham Woods) due to insufficiently detailed data on social parameters. Finally, for two species, data were available for two distinct populations. For the African wild dog (*Lyacon pictus*), data were available from Hluhluwe-iMfolozi Park and the Selous Game Reserve. Life table estimates from Selous Game Reserve were based on larger sample sizes so we elected to take data from this population. For the Alpine marmot (*Marmota marmota*), data were available from Gran Paradiso National Park and La Grande Sassière Nature Reserve. We chose the La Grande Sassière Nature Reserve population as we were able to collate data for both actuarial and reproductive life table estimates, whereas only actuarial data were available from Gran Paradiso National Park.

Quantifying aging metrics

To quantify aging metrics for each population, we built life tables (Chiang 1984) using the demographic data we collected. Life tables record, for each age (x), the mean probability of survival and rate of reproduction at that age. We took life tables directly from a published source, computed our own from plots using WebPlotDigitizer (https://automeris.io/WebPlotDigitizer/), or

used the raw data of repeated measurements of individuals supplied by authors. The probability of survival at age x ($p(x)$), the hazard of mortality at age x ($\mu(x)$), and survivorship up to age x ($l(x)$), where $l(0) = 1$, could all be calculated if only one was known, using the relationships:

$$l(x) = \prod_{y=0}^{y=x-1} p(y) \tag{1}$$

and

$$\mu(x) = -\ln\left(p(x)\right) \tag{2}$$

The rate of reproduction at age *x* ($m(x)$), when available, was recorded ideally as the number offspring that survived to independence ('weaned' or 'recruited'), but for some bird species only data on fledging success were available. Using $m(x)$ and $l(x)$, we quantified three response variables. First, we calculated the expected adult lifespan for each population as $\sum_{\alpha}^{\omega} l(x)$ (Bongaarts & Feeney 2003), where α is the age of maturity and $l(\alpha)$ was set equal to 1. Second, we used survivorship data to quantify the age-specific distribution (shape) of mortality as:

$$S_{\mu} = \frac{1}{\tau H}\left(\frac{\tau H}{2} - \int_{\alpha}^{\omega} H(x)dx\right) \tag{3}$$

where $H(x)$ captures the cumulate hazard of mortality up to age x, and corresponds to the logarithmic transformation of $l(x)$. The terms $\tau = \omega - \alpha$, with ω denoting the maximum age class, standardises the measure of shape according to adult lifespan. The term $\frac{\tau H}{2}$ represents the value of shape when age-specific mortality is constant (Baudisch & Stott 2019), rendering the

value of S_μ (a) positive if mortality is distributed towards older adult ages classes, (b) around zero if roughly equally distributed between adult age classes, and (c) negative if biased towards younger adult age classes. Finally, we used age-specific reproduction data to quantify the age-specific distribution (shape) of reproduction as:

$$S_R = \frac{1}{\tau B}\left(\int_{\alpha}^{\beta} B(x)dx - \frac{\tau B}{2}\right) \quad (4)$$

where, in this case, $\frac{1}{\tau B}$ provides standardisation of the reproductive lifespan, and $\frac{\tau B}{2}$ represents a constant rate of reproduction across the adult ages. The subtraction in (4) means the value of S_R will be (a) positive if reproduction is biased towards younger adult ages classes, (b) around zero if roughly equally distributed between adult age classes, and (c) negative if biases towards older adult age classes.

Statistical analysis and covariates

To test our four hypotheses regarding the effects of group-level characteristics on lifespan and senescence, we performed four Phylogenetic Generalized Least Squares (PGLS) models using the *nlme* package (Pinheiro *et al.* 2007) in R Studio (R Core Development Team 2015). To account for non-independence in lifespan and rates of senescence that result from common ancestry, we built a phylogeny for all the species in our dataset and then pruned to fit each of the datasets for each model (Hinchliff *et al.* 2015), assuming a Brownian model of evolution. The final set of populations in each model was determined by the availability of covariates (Extended Data Table 2). For five populations (the White-breasted robin (*Eopsaltria georgiana*); the Red-winged fairy-wren (*Malurus elegans*), the Galapagos mockingbird

(*Mimus parvulus*), the Sperm whale (*Phyester microcephalus*), and the Common bottlenose dolphin (*Tursipos truncates*), we were unable to collect information on social parameters, and so they were excluded from all analyses (Fig. 2). Our final sample represented 25 populations of 25 unique species.

Model 1 – Is a higher probability of inheritance by philopatric offspring associated with a lower adult life expectancy?

To test Hypothesis 1, we considered the effects on expected adult lifespan of relatedness and the proportion of individuals that breed in the same group that they were born into. Estimates of relatedness were based on genetic microsatellite data, except for *Glis glis*, where relatedness estimates were based on pedigree relationships between individuals that nested communally (Marin & Pilastro 1994). Estimates of relatedness were set to the relatedness of a philopatric subordinate individual to a dominant breeder of the same sex in groups with dominance hierarchies. When this information was not available, or in cases where multiple individuals within the group co-breed, we used the average relatedness among individuals within the group. Populations were removed from the analysis if no estimates of relatedness were available and/or the proportion of individuals born to a group that became breeders in the same group was unknown. If groups did not have territories or groups were not stable across generations (*e.g.*, long-tailed tits (*Aegithalos caudatus*)) then the proportion of individuals born to a group that became breeders in the same group was set to zero. Finally, due to the known relationship between body size and longevity across animals (Stearns 1983; Healy *et al.* 2019), we included adult body mass (log-transformed) collected from the Amniote Life History Database (Myhrvold *et al.* 2015) as an additional covariate.

Model 2 – Is higher reproductive skew associated with a bias of reproduction towards older age classes (*i.e.* more negative senescence)?

To test Hypothesis 2, we examined how the level of reproductive skew influences the distribution of reproduction across the adult life course. For simplicity, we described skew as a binary variable, set to 1 (high reproductive skew) if a single individual of the sex represented by the life table data reproduced at any given time, and 0 (low reproductive skew) if multiple individuals of the same sex reproduce in the same group at the same time. In most cases, this assignment was simple to allocate as dominance hierarchies were described in the studies for a given species. However, in some cases there were slight nuances. For example, superb-fairy wrens (*Malurus cyanus*) males display an observable dominance hierarchy yet the socially dominant male only sires 39% of offspring produced at the nest (Cockburn *et al.* 2016). Additionally, 44% of subordinate females lay eggs in the nest of a dominant individual in the Seychelles warbler (*Acrocephalus sechellensis*; Komdeur *et al.* 2016). In these cases, we set reproductive skew to 0.

Model 3 – Is a stronger effect of helpers towards breeder reproductive success associated with accelerated actuarial senescence but decreased reproductive senescence later in life?

To test Hypotheses 3, we explored how the magnitude of help and relatedness influence the distribution of mortality and reproduction across the adult life course. We explicitly tested how helping before reproduction later in life impacted an individual's age-specific distribution of mortality and reproduction, and so this analysis just focused on 'helper at the nest' systems. We ran two separate models, with the shape of mortality as the response variable for the first model, and the shape of reproduction as the response variable for the second model. To quantify the effect of the magnitude of help on rates of senescence, we restricted our populations to those in which juvenile individuals provide help to breeders (*i.e.*, helper-at-the-nest systems).

The effect of helpers was recorded as an increase in the number of fledglings or recruits (for birds), an increase in litter size (at birth) or number of pups surviving to independence (mammals). We collected data on relatedness estimates as described in **Model 1**, where possible ensuring that the estimate represented the relatedness of an individual of the helping sex to the helped individual. In species with facultative cooperative breeding, the average number of helpers in a group was weighted by the frequency of groups in which helpers were present to obtain an average group size. To estimate the magnitude of help, we recorded all available data for each population on the effect of helpers on reproductive success. In some cases, this magnitude of help was provided as an effect *per helper*. In cases where the data presented an overall effect of all helpers, we divided this overall effect by the average number of helpers. In species with facultative cooperative breeding, we devalued this magnitude of help *per helper* by the probability that an individual would engage in helping behaviour (*i.e.* the frequency of individuals that act as helpers in their juvenile period for each population). This devaluation was to give a more accurate reflection of the average help an individual would likely provide during its life, rather than assuming the individual has a 100% chance of exhibiting the helping behaviour.

Results

In our first hypothesis, we predicted that higher relatedness and a higher probability of an individual breeding in the group they are born into should favour reduced expected adult lifespan. After accounting for differences in body mass and phylogenetic relationships, a higher within-group relatedness was associated with a lower expected adult lifespan ($\beta \pm SE$ = -3.251 $\pm$ 1.417, p = 0.039; Fig. 1A). There was also a negative trend between an increased probability of philopatric offspring inheriting a breeding position and adult life expectancy, however this trend was non-significant (β = -0.019, $SE = \pm$ 0.010, p = 0.074). The effect of relatedness of expected adult lifespan did not depend on the likelihood of inheritance of a natal breeding

position by non-breeding group members (Relatedness × inheritance: β = 0.031, *SE* = ± 0.025, p = 0.227).

In our second hypothesis, we expected that groups with higher reproductive skew should show a bias in the distribution of reproduction towards older age classes, as it is usually the oldest individuals that gain the dominant breeding position (Shreeves & Field 2002). After accounting for phylogenetic relationships between species, there was in fact no significant relationship between the level of reproductive skew and the distribution of reproduction across the adult life course in our analysis (β = -0.301, *SE* = ± 0.295, p = 0.323; Fig. 2).

Finally, we predicted that a higher magnitude of help in helper-at-the-nest systems would lead to accelerated actuarial senescence, but weaker reproductive senescence. After accounting for phylogenetic relationships between species, there was no relationship between the magnitude of help and the distribution of mortality (Table 1). However, a non-significant trend did emerge for a higher magnitude of help weakening reproductive senescence (β = -1.257, *SE* = ± 0.600, p = 0.063). The estimate of relatedness within a group likewise had no significant impact on the distribution of mortality across the adult lifespan (Fig. 3; Table 1). We did, however, find that higher relatedness was associated with a bias of reproduction towards older age classes, *i.e.* weaker reproductive senescence (β = -2.950, *SE* = ± 0.917, p = 0.009; Fig. 2B).

Figure 1 | Species with a higher estimate of relatedness within the group show a reduced expected adult lifespan, and a bias in the distribution of reproduction towards later age classes. The blue lines represent the estimated linear relationship (±SE; gray) between relatedness within the group for each species (see Methods) and **A)** Expected adult lifespan (log transformed) (n = 18) and **B)** the Shape of reproduction (logit transformed after adding 0.5 to each data point) (n = 13). The more negative the shape of reproduction, the more skewed reproduction is towards later age classes. Data points represent raw values for each population and are scaled according to the estimate of log adult body mass (grams).

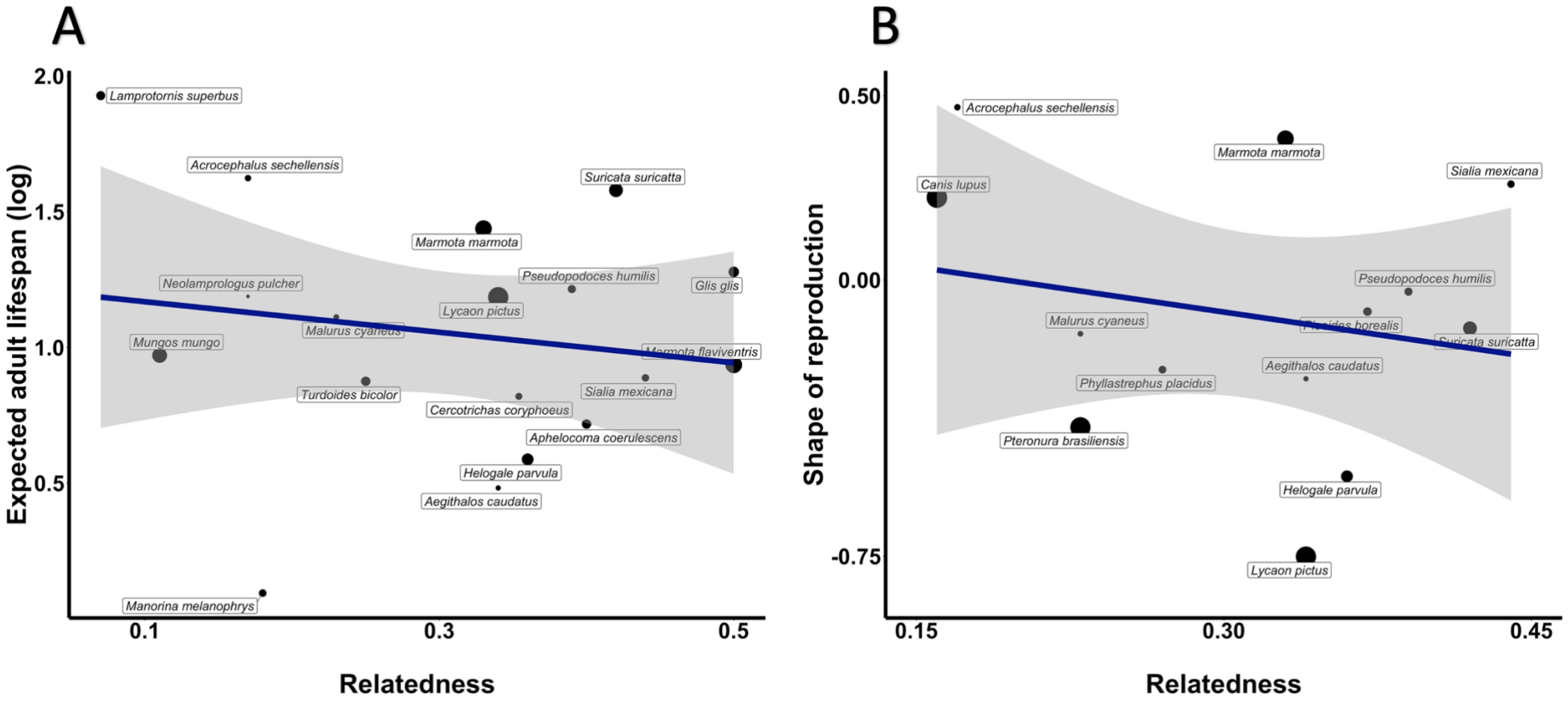


Figure 2 | There was no significant difference in the distribution of reproduction across the life course between species in which multiple individuals of the same sex reproduced at the same time (low) and those in which only a single individual of a sex reproduced at any given time (high). Values of the shape of reproduction are logit transformed after adding 0.5 to each data point, to meet model assumptions of normal error distributions. The gray horizontal line at the point at which the shape of reproduction is equal to zero represents absolute negligible senescence *i.e.* a constant rate of reproduction with age. Values above zero indicate that reproduction is biased towards younger age classes (positive reproductive senescence), whilst values below zero indicate that reproduction is biased towards older age classes (negative reproductive senescence).

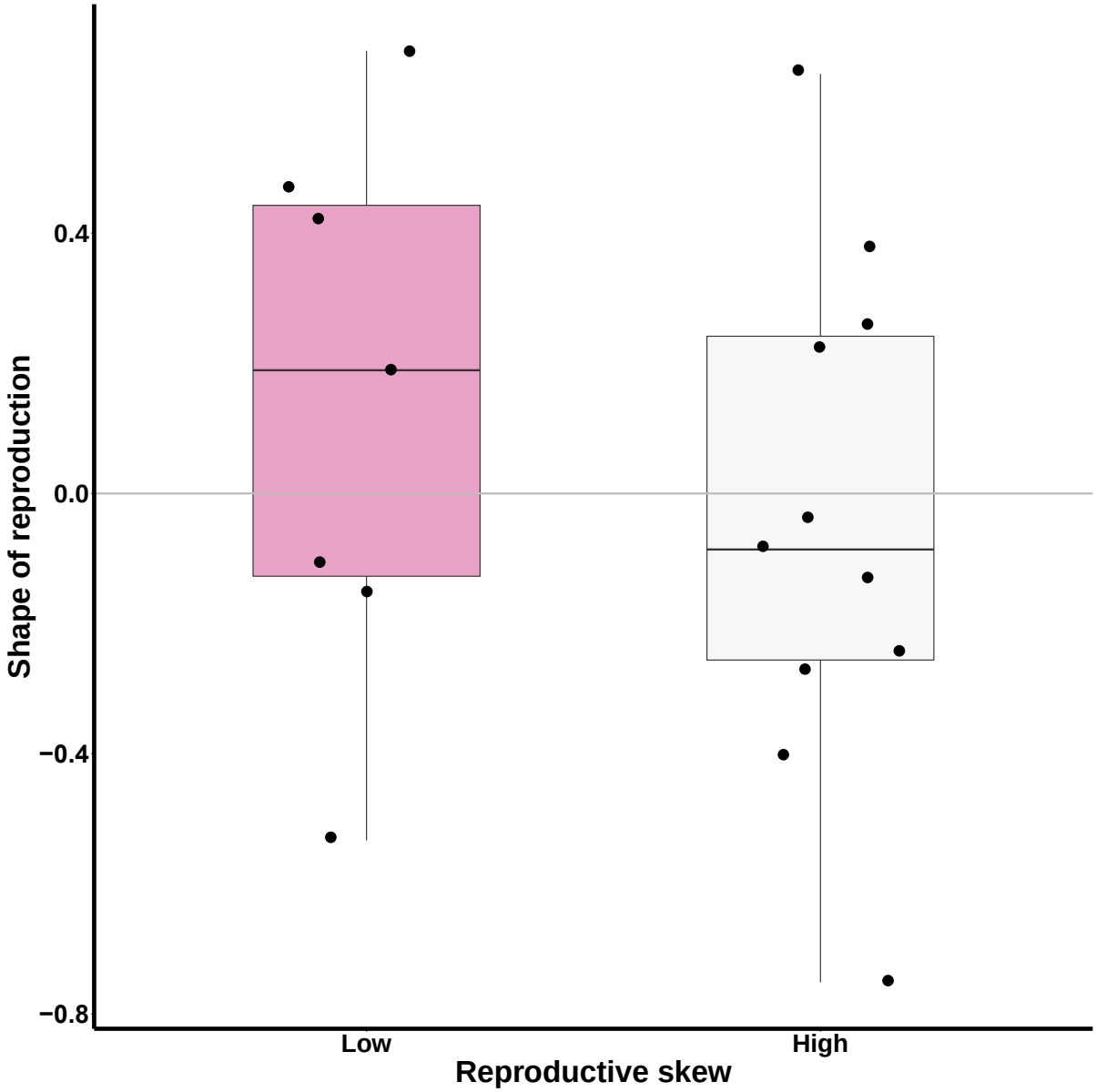


Table 1 | Outputs from PGLS models exploring the relationship between features of species with alloparental care and expected adult lifespan (Model 1), actuarial senescence (Model 3), and reproductive senescence (Model 2 and 4). Relatedness was an estimates of the genetic relatedness of an individual of the modelled sex towards other members of the group. Inheritance recorded the probability an individual born into a group would go on to reproduce in the same group. Body mass was an estimate of adult body mass for the species in question (log transformed). Skew recorded whether a single individual of the modelled sex (high) or multiple (low) reproduced at any given time. Finally, the magnitude of help represented the effect of one helper on the reproductive success of breeders in the group before independent breeding. Statistically significant effects ($p < 0.05$) are presented in bold. All estimates of Pagel's λ, a measure of phylogenetic inertia, were not significantly different from zero for all response variables, indicating a lack of phylogenetic inertia across our study species.

Model	*Response*	*Fixed effect*	*β*	*SE*	*p*
1	Expected adult lifespan (log)	Relatedness	**-3.251**	**1.416**	**0.039**
		Inheritance	-0.019	0.010	0.074
		Body mass	0.054	0.068	0.440
		Relatedness*Inheritance	0.031	0.024	0.227
2	Shape of reproduction	Skew	-0.301	0.295	0.323
3	Shape of mortality	Relatedness	-0.684	1.010	0.511
		Magnitude of help	-0.028	0.562	0.961
4	Shape of reproduction	Relatedness	**-2.950**	**0.917**	**0.009**
		Magnitude of help	-1.257	0.600	0.063

Discussion

Here, we show that group-level characteristics of species that exhibit alloparental care, in particular relatedness, play a role in explaining variation in estimates of expected adult lifespan and the distribution of reproduction across the adult life course. Most investigations of the effect of sociality on lifespan and senescence thus far have focused on comparing species at the level of cooperative *vs.* non-cooperative (Downing *et al.* 2015; Healy 2015; Thorley 2020; Vágási *et al.* 2021). Yet, variation in how groups form, levels of relatedness between individuals, access to reproduction, and social interactions that effect other individuals fitness, all have the potential to alter inclusive fitness forces of selection in social species (**Chapter 4**). We indeed show that these features play a significant role in explaining variation between key life history components among species that exhibit alloparental care.

Expected adult lifespan is lower in groups with higher relatedness

Species that exhibit alloparental care tend to form by individuals remaining on their natal territory, dispersing to a new group as a subordinate or breeder, or joining other adults and breeding communally (Riehl 2013; Downing *et al.* 2020). Offspring can gain direct benefits of philopatry before dispersing from a 'safe haven' (Stacey & Ligon 1991), and in some situations may also provide help and increase the reproductive success of relatives, gaining indirect fitness benefits (Brown 1974; Emlen 1982a; Emlen 1982b; Hamilton 1964a; Hamilton 1964b; Hatchwell 2009). Individuals can also gain further direct fitness benefits by helping when group size is correlated with reproductive success, benefitting from group augmentation after inheriting a breeding position (Kokko *et al.* 2001; Kingma 2017). If individuals remain in the same group they are born into, access to reproduction when initially mature is often limited due to the high likelihood of inbreeding (Nichols 2017; Jennions & Macdonald 1994), or the presence of more dominant individuals that monopolise reproduction (Leadbeater *et al.* 2011). When there are limited breeding opportunities within a group, there may be additional selection

on mortality in older individuals with lower residual inclusive reproductive value to 'turnover' their breeding position to a younger, related, individual (Bourke 2007; Kreider *et al.* 2022; **Chapter 4**). This additional selection pressure should, all else being equal, be more pronounced when the older individual is more closely related to the potential recipient, which is more likely in species where offspring exhibit stronger philopatry and ultimately attempt to breed within their natal group. We report a trend towards groups with higher within group breeder inheritance showing reduced expected adult lifespan, but a significantly negative relationship between within-group relatedness and expected adult lifespan across species. This finding suggests that, across the species in our study, a higher mean relatedness within the group favours a faster turnover of breeders, providing some support for hypothesis. Despite this apparent relationship, there may be some caveats to this result. We attempted to account for confounding differences in expected lifespan between species using the assumption that increasing body mass correlates strongly with reduced mortality (Stearns 1983; Healy *et al.* 2019). One additional possibility, however, is that the species in our dataset that experience particularly harsh environments may have stronger philopatry, due to the 'safe-haven' effect (Stacey & Ligon 1991), which raises relatedness. This in turn may lead to a correlation between higher relatedness and reduced expected adult lifespan, that is independent of the additional selection pressure on breeders to turnover their breeding position to philopatric offspring.

Reproductive skew and reproductive senescence

When access to reproduction is predominantly driven by age, the age-specific distribution of reproduction should, logically, be biased towards older age classes. This bias of reproduction to older age classes should in turn result in weaker rates of reproductive senescence (Vaupel *et al.* 2004). Here, we did not find a statistically significant difference in the distribution of age-specific reproduction between those species in which a single individual of a given sex reproduced at one time *vs* those species in which multiple species of a given sex reproduced at

any given time. There are limitations in the way we define skew in this analysis as "High" *vs.* "Low", as most populations fall somewhere between the complete monopoly of reproduction by a single individual, and completely plural breeding, rather than at the extremes. For examples from well-studied systems, 39% of offspring are sired by the dominant male in the superb fairy-wren (*Malurus cyaneus*; Cockburn *et al.* 2016), and 44% of subordinate females lay eggs in the nest of a dominant individual in the Seychelles warbler (*Acrocephalus sechellensis*; Komdeur *et al.* 2016). There is debate to whether reproductive skew can be defined on a continuous range (see e.g. Rubenstein *et al.* 2016), but, here, it might a provide a higher resolution answer to this question.

There is an additional hypothesis for how reproductive skew might impact expected adult lifespan, which we did not consider here. Indeed, in their simulation model of ageing in cooperative breeders, Kreider *et al.* (2022) suggested that species with age-based queues for reproduction should have a longer expected lifespan than species with no age-related queue. All else being equal, if reproduction is distributed to older age classes, then this should favour extended life. However, their model does not consider the ability for individuals to achieve indirect fitness whilst waiting in the queue. Fitness is primarily determined by waiting in a queue to become the breeder, and achieving the reproductive success of a breeder. Cooperative breeders that have tend to have age-specific queuing, or at least reduced shared parentage, tend to be family based (Downing *et al.* 2020), meaning that relatedness is likely higher. It is therefore a reasonable assumption that individuals in age-based queuing systems do stand to gain from indirect fitness benefits when queuing. If individuals have a low probability of ultimately achieving the breeding position (*e.g.* due to low mortality of breeders, or high group size), then this might favour greater helping effort expenditure to maximise indirect fitness returns, which if costly may reduce the average expected adult lifespan. A future test that can

quantify a species' level of age reproductive queuing (*e.g.* weakly age-dependent vs strongly age-dependent; Kreider *et al.* 2022), along with estimated indirect *vs.* direct fitness returns per age class when queuing, could illuminate an answer to this probing question.

Magnitude of help and rates of senescence

Individuals that delay dispersal and then engage in helping behaviour to boost the reproductive success of related individuals can gain indirect fitness benefits. The higher the magnitude of help *per helper*, and the higher the relatedness between the helper and the related offspring, the higher the indirect fitness reward reaped by the helper (Koenig & Dickinson 2016). In an extension to **Chapter 4**, we showed that, when this indirect fitness benefit reward is higher, there is reduced selection on survival and rate of reproduction in juvenile age classes. We therefore interpreted this result by suggesting that species with helpers that exhibit a higher magnitude of help should display faster actuarial senescence, and weaker reproductive senescence. Our analyses showed no relationship between the magnitude of help, or relatedness, and the distribution of mortality across the adult lifespan. A weaker force of selection on the survival of juvenile individuals after providing a higher amount of help arises in the model due to the competitive effects directed towards the offspring they just helped to produce. By surviving, the individual is reducing the probability the offspring establishes into the local group (Ronce & Promislow 2010; **Chapter 4**). However, rather than selecting for reduced individual survival, in reality, this increased selection pressure to reduce competition may instead favour dispersal after helping (which the model does not currently allow). This would suggest that juvenile individuals that provide more help to closely related individuals, accruing more indirect fitness, should be more inclined to disperse after helping – dependent on their continued presence being costly to the helped offspring (*e.g.* through group size

constraints). This may explain the disconnect between our theoretical prediction and the result we found.

Our analysis did reveal a bias in reproduction towards older age classes in species with a higher level of relatedness, and a trend towards biased reproduction in older age classes in species with a higher magnitude of help. A bias in reproduction towards older age classes in groups with higher relatedness may link to the points discussed earlier, in that family groups tend to show higher reproductive skew (Downing *et al.* 2020). This is often due to inbreeding avoidance, and a higher tolerance of related individuals breeding instead of oneself. In turn, this may reduce the breeding opportunities of younger individuals, biasing reproduction to older ages. Repeated analysis with a larger sample size may confirm whether the trend towards a higher magnitude of help from juveniles being associated with weaker reproductive senescence is aligned with our theoretical predictions.

One significant limitation of the model in **Chapter 4**, and prior models on the forces of selection (Hamilton 1966; Baudisch 2005), is that the effect of perturbations to the rate of survival or reproduction on fitness are modelled independently. For example, a higher magnitude of help suggests alternative hypotheses for the force of selection on survival and reproduction, which suggests asynchrony between the two (**Chapter 4**). Yet, selection for reproduction to be biased towards older age classes, could in turn increase the force of selection on survival to older age classes. The interaction between perturbations to mortality/survival and reproduction in force of selection models is an important future step that will improve our understanding of how senescence should evolve generally. In social species, this should also be modulated by social transfers between age classes: for example, a reduced rate of mortality in older age classes that provide help to younger individuals might not increase the force of

selection on reproduction as much as in a system were older individuals can only achieve direct fitness.

The evolution of lifespan and senescence in cooperative breeders

Here, we performed an analysis of variation in adult lifespan and the distributions of mortality and reproduction across species that exhibit alloparental care. The social insects are an obvious missing group from our analysis in which extreme lifespans (Keller & Genoud 1997) and queen-worker differences in rates of senescence (Kramer & Schaible 2013; Heinze & Giehr 2021) have been explored. We excluded social insects from our analysis due to limited individual-level estimates of survival and reproduction in wild populations, and difficulty in defining the individual in 'superorganism' systems (Boomsma & Gawne 2017). Yet our results, and modelling (**Chapter 4**), should extend to those systems. It would be interesting to explore whether particularities of social insects, e.g. queen-worker conflict (Bourke 2007), could be integrated into the theory. In particular, it has been suggested that the optimal longevity for a queen from the perspective of a worker and a queen may differ, and a period of potential conflict may arise when the queen is at an age when workers would maximise fitness by resource turnover (Almond *et al.* 2019). Using inclusive reproductive values developed in **Chapter 4**, game theoretical approaches may be utilised to investigate how this conflict is resolved.

Although we attempted to include species in our study across a diverse range of taxa, our analysis was restricted by the need for studies in which longitudinal monitoring of individuals had been conducted (Clutton-Brock & Sheldon 2010). This requirement often biases the types of species included in comparative analyses, creating data gaps (Conde *et al.* 2019), mostly due to limited feasibility of monitoring wild populations of certain systems, *e.g.* fossorial

species. An obvious caveat to this work is the restricted sample of species, and whether the results would hold with data from a more diverse range of systems. Nonetheless, we showed with the species studied here that key group-level features, in particular relatedness, may represent an important source of variation in the evolution of mortality, reproduction, and lifespan among social species. Ultimately, our results show that modelling higher resolution features of social groups are important to understanding how long they live, and how they age.

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6 | Individual variation explains ageing patterns in a cooperatively breeding bird, the long-tailed tit *Aegithalos caudatus*

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RESEARCH ARTICLE

Individual variation explains ageing patterns in a cooperatively breeding bird, the long-tailed tit *Aegithalos caudatus*

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Abstract

1. Alloparental care in cooperatively breeding species may alter breeder age-specific survival and reproduction and subsequently senescence. The helping behaviour itself might also undergo age-related change, and decisions to help in facultative cooperative breeders are likely to be affected by individual condition.
2. Helpers in long-tailed tits *Aegithalos caudatus* assist relatives after failing to raise their own brood, with offspring from helped nests being more likely to recruit into the breeding population.
3. Using data collected over 25 years, we examined the age trajectories of survival and reproduction in adult long-tailed tits to determine how these were affected by the presence or absence of helpers and how helper behaviour changed with age.
4. There was evidence for increased reproductive performance with breeder age, but no effect of age on the probability of survival. We found no evidence of significant senescent decline in survival or reproductive performance, although individuals accrued less inclusive fitness in their last year of life. Lifetime reproductive success was positively related to both reproductive life span and body mass. Within a season, breeders that were assisted by helpers enjoyed greater reproductive success through enhanced offspring recruitment in the following year. We found no evidence that age affected an individual's propensity to help, or the amount of indirect fitness accrued through helping.
5. We found a positive correlation between life span and multiple components of reproductive success, suggesting that individual variation in quality underpins age-related variation in fitness in this species. Helping decisions are driven by condition, and lifetime inclusive fitness of immigrants was predicted by body mass. These findings further support individual heterogeneity in quality being a major driver for fitness gains across the life course of long-tailed tits.

KEYWORDS
ageing, cooperative breeding, demography, helping, inclusive fitness, life history, senescence

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1 | INTRODUCTION

Senescence is the decline in physiological condition with age that leads to an increased risk of mortality and/or reduced reproductive success after reaching maturity (Medawar, 1952). Although once thought unlikely to be observed in nature, there is now widespread evidence for senescence in wild populations (Gaillard & Lemaître, 2020; Nussey et al., 2013). Studies have also unearthed a diversity of ageing patterns (Jones et al., 2014) that includes negligible (Finch, 1990) or even negative (Vaupel et al., 2004) senescent life histories, where risks of mortality remain constant or decline with age, and reproductive success remains constant or increases with age. Senescence has classically been explained as a result of the declining strength of natural selection with age (Hamilton, 1966; Medawar, 1952; Williams, 1957) and the subsequent trade-offs between early and late-life performance that will often favour the former (Kirkwood, 1977). Given the diversity of ageing patterns observed in wild populations, however, the key question to address now is why the decline of the strength of selection with age seems more prominent in some species, and less so or even absent in others. Critically, there is growing theoretical (e.g. Lee, 2003) and empirical (e.g. Hammers et al., 2019) evidence that sociality may contribute significantly to explaining this variation in ageing patterns.

The general relationship between sociality, longevity and ageing appears complex and not unidirectional (Lucas & Keller, 2020). On the one hand, a species' propensity to be social is likely to be driven somewhat by age-related patterns of survival and reproduction. Indeed, long life with high adult survival and overlapping generations, for example, can provide greater opportunities for individuals to interact, and thus facilitate the evolution of cooperation (Arnold & Owens, 1998; Downing et al., 2015; Ross et al., 2015; Taylor & Irwin, 2000). On the other hand, sociality itself may alter selection gradients on age-specific survival and reproduction, and therefore the pattern of senescence (Bourke, 2007; Lee, 2003). Most current empirical evidence that sociality can impact senescence comes from studies of cooperative breeders. In cooperatively breeding species, helpers are individuals that aid the rearing of offspring that are not their own. This alloparental care may allow breeders to reduce their current reproductive investment, reproduce at higher rates for longer proportions of their life span and enjoy increased survival ('load lightening' – Crick, 1992). This load lightening in turn may delay or even mask senescence (Lucas & Keller, 2020).

Recent evidence supports the idea that load lightening can delay senescence in cooperative breeders (Downing et al., 2021). Studies in facultatively social species such as the Seychelles warbler *Acrocephalus sechellensis* and the Alpine marmot *Marmota marmota* have demonstrated that breeders that receive help experience slower mortality (i.e. actuarial) senescence (Berger et al., 2018; Hammers et al., 2019). Such species-specific studies have, however, tended to focus on reproductive (Russell et al., 2007; Sharp & Clutton-Brock, 2010) or actuarial senescence (Berger et al., 2018; Hammers et al., 2019) in isolation, but not both in an integrative manner (but see Cooper et al., 2021). Given that these two components of fitness can follow different ageing trajectories (Jones et al., 2014), and that costs of reproduction, and alleviation by helpers, may be important for survival (Kirkwood, 1977), a full picture of how cooperative breeding can shape age-specific fitness acquisition requires a detailed understanding of both reproductive and survival components.

Intuitively, the focus of load-lightening research has been on the effect of the helping behaviour on breeder fitness at different ages. In theory, however, if the costs and benefits associated with helping are (st)age dependent, we may then expect (st)age-dependent expression and fitness consequences of the helping behaviour from the helper's perspective (McNamara & Houston, 1996; Rodrigues, 2018). There are plentiful examples of such age polyethism of helper behaviour in nature (Field et al., 2006; Zöttl et al., 2016). Workers of the termite *Neocapritermes tracua*, for example, switch helping behaviour from a feeding role to self-sacrificing colony defence as they age and their feeding efficiency declines (SobotnÍk et al., 2012). Despite this evidence, however, we are not aware of any study that has explicitly tested for senescence in helping behaviour.

Here, we investigate ageing patterns in the long-tailed tit *Aegithalos caudatus*, a facultative cooperative breeder with redirected helping. In this species, all individuals attempt to breed in every year of their life. If nests fail early in the season, individuals may attempt to breed again, but later in the seasons they may instead choose to help other breeding pairs in raising their offspring by feeding nestlings and fledglings (MacColl & Hatchwell, 2002; Hatchwell, 2016; Figure 1). The helping behaviour is altruistic, favoured by the indirect fitness benefits individuals gain when increasing the productivity of broods to which they are related (Hatchwell et al., 2014). Given that individuals can switch between breeding and helping roles both within and across breeding seasons, it is important to investigate age-related patterns of both breeding and helping behaviour to fully understand individual ageing patterns in this species (Figure 1; Table 1).

This study species provides many benefits for senescence research in social animals. First, given the short adult life spans of individuals (mean = 1.67 years), lifetime reproductive success (LRS) can be straightforwardly quantified for many individuals. Second, as helping is performed only by failed breeders, and there are no direct fitness benefits of helping (Meade & Hatchwell, 2010), we can clearly separate direct and indirect components of inclusive fitness. This distinction allows us to investigate ageing patterns in these components of fitness independently, and then subsequently how helpers may alter age trajectories of components of breeder fitness. Finally, these fitness components can further be broken down into moments of the reproductive schedule for each year of a bird's life. This breakdown scales from clutch sizes through to the number of fledglings, to the number of recruited offspring for breeders, and from the decision to help to the probability that an offspring

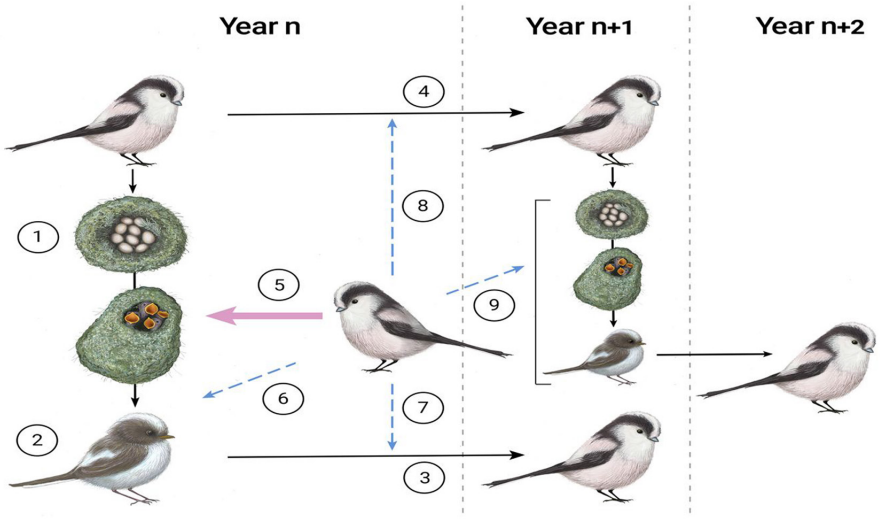


FIGURE 1 The life cycle of long-tailed tits *Aegithalos caudatus* and key demographic events. Numbers indicate demographic events we investigated, the models for which are displayed in Table 1. In year *n*, adult long-tailed tits produce clutches of around 8–12 eggs (1) in socially monogamous pairs which develop via a nestling and fledgling (2) stage ultimately to recruit into the population in year $n+1$ (3). Adults may also survive (4) to year $n+1$, with a mean life expectancy of 1.67 years. At the nestling stage, breeders may receive assistance rearing their brood from individuals in the population who have failed to breed—redirected helpers (5). Helpers tend to be related individuals, whose effect is not to increase the number of fledglings at a nest (6), but instead the probability a given fledgling recruits into the population (7). Helpers have been shown to have limited effects on breeder survival (8), while their effects on breeder fitness in year $n+1$, that is production of recruits in year $n+2$ (9), is unknown.

receiving help successfully recruits into the breeding population. In this species, therefore, we can investigate senescence from the level of inclusive fitness (Hamilton, 1964), and down through multiple fitness components that contribute to this metric, rendering the long-tailed tit an excellent study system for investigating senescence.

In this study, we undertook a comprehensive analysis of ageing in long-tailed tits that includes (a) ageing patterns of breeder survival and reproduction [including age-related changes in extra-pair paternity (EPP)], (b) the effects of helpers on these patterns and (c) ageing patterns of helper behaviour (see Table 1). First, for breeders, we expected survival and reproduction to follow an increase from maturity to a peak followed by a senescent decline, as is evident in other passerines and in general across animals (e.g. Bouwhuis et al., 2009; Clutton-Brock, 1984). Given that the long-tailed tit is a short-lived species, we expected a trade-off between reproductive output and survival, which we tested for by including direct fitness and reproductive effort (see Materials and Methods) in models of survival. In our study population, females exhibit a degree of promiscuity, with an estimated 11% of offspring in 30% of nests being the product of extra-pair mating (Green & Hatchwell, 2018). To fully understand the ageing patterns of reproduction in this species, we therefore also explored the relationship between (a) female age and production of extra-pair offspring and (b) male age and the probability of both gaining EPP and losing paternity to extra-pair sires. We had no clear a priori prediction for how EPP should vary with female age, given the diversity of results obtained for other species (Jennions & Petrie, 2000; Raj Pant et al., 2020). In the case of males, for which we have some evidence for variation in quality (Meade & Hatchwell, 2010), we expected a male's life span (which we assume is a predictor of quality) to be positively associated with the probability of gaining EPP and negatively associated with the probability of being cuckolded. Second, we tested for load-lightening effects of helpers on breeder current reproduction, survival and future reproduction. Previous work in this species has shown that helpers boost long-term (recruit number) but not short-term (clutch size and brood size) productivity of breeders (Hatchwell et al., 2004). We therefore expected the presence of helpers to reduce age-related decline in measures of recruit production but not short-term productivity. Previous research has also found that helpers boost survival of male breeders caring for large broods, but found no significant effects of being helped in year *n* on an individual's clutch size or timing of breeding in year $n+1$ (Meade et al., 2010). We extend these analyses to ask whether being helped in year *n* influences the probability of fledging offspring and accruing direct fitness in year $n+1$. Finally, given that previous work in this species has suggested that helpers are relatively high-quality individuals (McGowan et al., 2003; Meade et al., 2010), we expected to find limited age-related decrease in helping performance, measured through (a) indirect fitness accrual and (b) the probability a helped fledgling recruited into the population. If senescence occurs in the study population, however, this may influence an individual's decision to help or not given that helping is a costly behaviour (Hatchwell et al., 2014).

2 | MATERIALS AND METHODS

2.1 | Study site and collection of data

Since 1994 (data here up until 2019), a population of long-tailed tits has been studied within a 2.5 km^2 study site in the Rivelin Valley, Sheffield, UK (52°23′N, 1°34′W). The animal study was

TABLE 1 Summary of models investigated in this study. Terms: age at last reproduction (ALR), reproductive effort (RE), presence or absence of helpers (helpers), relative lay date (RLD), relative fledge date (RFD).

	Figure 1	Response variable of demographic events investigated	Fixed effects	Random effects
Ageing patterns of breeder survival and reproduction	1, 2, 3, 4	Clutch size	Age, Age^2, ALR	Identity, Year
		Fledglings	Age, Age^2, ALR, Sex	Identity, Breeding Pair Identity, Year
		Direct fitness	Age, Age^2, ALR, Sex	Identity, Breeding Pair Identity, Year
		Survival	Age, Age^2, Sex, Direct Fitness, RE	Identity, Year
Effects of helpers on breeders' ageing patterns	6,7,8,9	Current reproduction: fledgling production and recruit production	Age, Age^2, ALR, Sex, Helpers	Identity, Breeding Pair Identity, Year
		Survival	Age, Age^2, ALR, Sex, Helpers	Identity, Year
		Future reproduction: RLD, clutch size, fledgling production and direct fitness (all in year $n+1$)	Age ($n+1$), Age^2 ($n+1$), Helpers (n), ALR, Brood size (n), Recruits (n), RFD (n), Spring temp, Winter rainfall	Identity, Year
Ageing patterns of helping behaviour	5, 7	Decision to help	Age, Age^2, ALR, RLD, RE	Identity, Year
		Probability a fledgling recruit	Helper mean age, fledgling sex, RFD, Helpers (number), relatedness	Identity, Year
		Indirect fitness	Age, Age^2, ALR, Sex	Identity, Year

reviewed and approved by the University of Sheffield Ethical Review Committee (Project Applications and Amendments Sub-Committee). Every year, nestlings are ringed with a unique colour-ring combination, and blood samples are taken under Home Office Licence (PPL7007834) for genetic analysis. Over 95% of unringed adults that appear in the study population are also colour-ringed and blood-sampled; these unringed individuals are assumed to be immigrants born outside the study site. Individuals from whom blood samples are taken are genotyped at 19 microsatellite loci (Adams et al., 2015); sharing of alleles at these loci provides an estimate of pairwise relatedness between individuals using the method of Queller and Goodnight (1989). Body mass (±0.1 g) and tarsus length (±0.1 mm) are recorded for offspring on day 11 after hatching, and upon first capture for new immigrants in the population. Immigrants are assumed to be aged one in the year of their first sighting, based on the observations that individuals tend to move relatively short distances following their first breeding season (Hatchwell, 2016). The probability of resighting individuals in this system is high (92% and 83% for males and females respectively; Gullett et al., 2014), and so individuals are assigned a year of death equivalent to the year in which they were last sighted.

In each breeding season, clutch size, number of fledglings and the presence of helpers were recorded for each nest following the methods of Hatchwell et al. (2004). The following year ($n+1$), surviving fledglings are recorded as recruits for breeders in year n. Recruits were converted to genetic offspring equivalents following Green and Hatchwell (2018) to give measures of direct fitness (for breeders) and indirect fitness (for helpers) that are directly comparable and can be summed to obtain inclusive fitness (Hamilton, 1964). To calculate indirect fitness, we first estimated the average effect of a given number of helpers on the probability of offspring recruitment. Using this information, we then estimated the average fraction of a recruit that is attributable to a helper, for a given number of helpers at a nest. A helper's indirect fitness was then obtained by multiplying this fraction by the relatedness between the helper and any offspring that successfully recruited. Where multiple offspring recruited, the average relatedness between these and the helper was calculated. Helpers received no indirect fitness if no recruits were produced from the nest they helped, nor if their relatedness to recruits was ≤ 0. The direct fitness of an individual in a given year of their life was the number of recruits produced in a year minus the fraction of recruits attributable to any helpers. The remaining fraction was then halved twice, first to account for the contribution of the other breeding partner to the recruit produced, and second to account for the average relatedness of 0.5 between parents and offspring. The inclusive fitness accrued by a bird in a year of their life is simply the sum of the indirect and direct fitness they accrue in the year. Note that the measures of fitness we calculated correct for parentage lost and gained through extra-pair matings. Fitness metrics were calculated for a total of 813 individuals, for which complete life-history information was available.

We also used information on an individual's total reproductive investment across all nesting attempts in a given year to

quantify a measure of reproductive effort (RE), following Tarwater and Arcese (2017). In long-tailed tits, incubation is performed exclusively by females, who are fed by their partners, while provisioning of nestlings is carried out by both sexes at approximately equal rates (Hatchwell, 2016). For females (n = 339), we calculated RE as the sum of an incubating (number of eggs × days incubating) and a provisioning (number of chicks × days provisioning) term. For example, an individual that lays 10 eggs, incubates for 13 days and then provisions these 10 chicks for 13 days would have a reproductive effort of 260. For males (n = 287), RE was calculated as the number of days their breeding partner spent incubating plus the same provisioning term used for females.

2.2 | Analyses of age-related components of fitness

Linear and quadratic effects of age were included in mixed-effects models to test for age-related patterns in the different components of fitness (Table 1). A significant quadratic effect of age may be due to improvement early in life rather than senescence, however, and therefore we performed 'post-peak analysis' to test for a significant negative linear relationship between age and the respective fitness component. This methodology excludes the quadratic effect in analyses of individuals of peak age and older with respect to a particular fitness component (Bouwhuis et al., 2009), where peak age represents the age with the highest mean value of relevant fitness components. A significant negative term of linear age in such an analysis would, if present, provide more reliable evidence of senescence.

When investigating population-level patterns of ageing, it is imperative to control for changes in the phenotypic composition of the population due to between-individual variation in quality (Van de Pol & Verhulst, 2006; Vaupel & Yashin, 1985). Age at last reproduction (ALR) was therefore included in all models testing for reproductive senescence to control for selective disappearance of 'poor-quality' individuals (Van de Pol & Verhulst, 2006; Table 1). Long-tailed tits always attempt to reproduce in their first year (Hatchwell, 2016), and so there is no need to control for selective appearance in our study population. In addition, we included the year (1994–2019) in which the data were collected and individual identity as random effects (Table 1). This step allowed us to account for both environmental variation and non-independence caused by repeated sampling of individuals across years. For models of reproductive senescence in which the two sexes were analysed together, we included a Pair ID term (mother ID plus social father ID) to control for non-independence caused by contributions of both parents to the production of fledglings and recruits.

Low sample sizes at late age classes can be an issue in detecting senescence (Nussey et al., 2008). Therefore, we reran our models with birds aged 4 and older classified as 4+ to boost sampling power. Also, through preliminary analysis, we identified that one bird (ID 1978) had a particularly successful year in its sixth year of life (inclusive fitness = 0.61, mean across population for a single year = 0.06) and so we reran our models with this bird excluded from the dataset. Throughout the results section, we report results of these alternative analyses only where they generate qualitatively different results. All analyses were conducted in R (R Core Development Team, 2015) and in RStudio (v 4.0.2). Mixed-effects models were run using the LME4 package (Bates et al., 2015). Finally, access to the study site was heavily restricted in 2001 following an outbreak of foot-and-mouth disease, resulting in uncertainty about (a) recruitment of offspring born in 2000 and (b) the survival of adult birds last observed in 2000. Thus, we removed these data from survival and direct fitness analyses.

2.2.1 | Ageing patterns of breeder survival and reproduction

Reproduction

We modelled clutch size with a Poisson error structure and for females only, given they are likely to have principal control over the number of eggs laid. For individuals that laid multiple clutches in the same year, we chose to analyse the size of the first clutch only to standardise measures across individuals and reflect condition at the start of the breeding season. As a result of the high level of whole brood mortality in long-tailed tits (>70%; Hatchwell, 2016), the number of fledglings produced and the amount of direct fitness accrued are heavily zero-inflated, so we modelled them with a compound Poisson error structure. Extra-pair paternity is corrected for in these analyses through paternity assignment (see Supplementary Information). We modelled both sexes together including sex as a covariate and a Pair ID term to account for non-independence. We also tested for an interaction between sex and age to test for sex-specific age trajectories of reproduction. For reproductive components of fitness, we were also able to account for terminal effects (see Supplementary Information; Clutton-Brock, 1984; Coulson & Fairweather, 2001). Finally, we used biometric information (see Supplementary Information) to test for correlations between lifetime inclusive fitness and life span with body mass.

To test for ageing patterns of extra-pair paternity, we investigated females and males separately using information from the genetic pedigree of the study population (see Supplementary Information). For females (n = 225), we modelled the probability they produced offspring from multiple males in a given year of their life as a function of their age, ALR and the difference in age between them and their social male partner using a binomial error distribution. The rates of conspecific brood parasitism in long-tailed tits are negligible (Green & Hatchwell, 2018; Hatchwell et al., 2002), so the social mother was assumed to have maternity in all cases. For social fathers (n = 213), we first modelled the probability they raised broods including at least one extra-pair offspring using the same fixed effects. In both models, we also

included the number of nestlings as a fixed effect to control for the increased likelihood of extra-pair paternity in larger groups (Raj Pant et al., 2019). Of the 429 males for which we had accurate lifetime recruit data, 46 were known to have sired at least one extra-pair chick. We modelled the probability males gained EPP with age (linear and quadratic) and ALR as fixed effects, and individual identity as a random effect. Due to the small number of males gaining EPP, attempts to account for Year as a random effect led to the model failing to converge.

Survival

We used the probability of individual survival from 1 year to the next to investigate actuarial senescence with a binomial error distribution. We included age (linear and quadratic) and sex as fixed effects, and an interaction between the two to test for differences in age trajectories of survival between the sexes. We also included the mean daily rainfall (mm) from 1 October in year n to 1 March in year $n+1$, previously shown to have a negative effect on survival in long-tailed tits (Meade et al., 2010). Weather data were provided by Weston Park Weather Station, Museums Sheffield, 5 km from the Rivelin Valley study site. Finally, we included direct fitness in year n as a further fixed effect to test for trade-offs between current reproduction and future survival. We performed an additional survival model for individuals that we had RE data for including both RE and direct fitness as fixed effects.

2.2.2 | Effects of helpers on breeders' ageing patterns

As helpers only help at nests that have survived beyond the incubation period, they are inevitably associated with more productive nests (Hatchwell, 2016). Helpers can arrive at any point between hatching and the fledgling stage and therefore, when testing for an effect of helpers on breeder fitness, we restricted our analyses to just those nests that produced fledglings and therefore could have received help, resulting in 249 breeding attempts for 390 individuals (199 females and 191 males).

Current reproduction

To test for the effect of helpers on the productivity of successful breeders, fledgling number was modelled with a Poisson error structure. Since our fitness metrics account for the contribution of helpers to recruit production, we model the effect of helpers on absolute recruit number rather than direct fitness with a negative binomial error structure. We included interactions between breeder age (linear and quadratic) and whether helpers were present or not at their nest to test for differences in age trajectories of reproduction between breeders that did and did not receive help.

Survival

To test for the effects of helpers on breeder survival, we first attempted to recapture the results of Meade et al. (2010) with 10 more years of data. We therefore included the breeder's brood size, relative fledge date and whether they produced recruits or not as fixed effects, alongside breeder age (linear and quadratic) and whether helpers were present or not. In their analysis, Meade et al. (2010) found that helping confers survival benefits to breeders, but that this effect was restricted to male breeders with large broods. We therefore fitted pairwise interactions between helping and sex and brood size to test whether survival benefits of help depended on breeder sex and brood size. Finally, we also included the mean daily rainfall (mm) from 1 October in year n to 1 March in year $n+1$, as this has previously been shown to have a negative effect on the survival of females (Meade et al., 2010).

Future reproduction

We extended the work of Meade et al. (2010) to investigate helper effects on future reproduction of breeders. For individuals that produced fledglings in year n and therefore could have received help, we tested for an effect of whether they received help or not on clutch sizes and relative lay dates in year $n+1$. We then tested whether being helped in year n influenced the probability of fledging offspring or accruing direct fitness through recruit production in year $n+1$. Fixed effects included whether the individual was helped, their brood size, and whether they produced recruits, in year n, and their age (linear and quadratic) in year $n+1$. We also included the mean daily rainfall throughout winter and the mean minimum daily temperature in degrees from 15 February to 15 March in year $n+1$ (spring temperature) as further fixed effects.

2.2.3 | Ageing patterns of helper behaviour

To investigate senescence of helping behaviour, we first tested for age patterns of indirect fitness accrual across the population. We then modelled the probability a fledgling recruited into the population as a function of its helpers' mean age (764 fledglings across 99 nests between 1995 and 2018). We modelled success or failure of recruitment by each fledgling using a binomial error distribution. We included relative fledge date (date of fledging relative to the 1 May), the number of helpers and the sex of the fledgling (to account for female-biased dispersal) as fixed effects (Green & Hatchwell, 2018). We also modelled the mean relatedness of the fledgling to its helpers as a fixed effect, given helpers have been shown to adjust their provisioning rates in accordance with their relatedness to nestlings (Leedale et al., 2018; Leedale et al., 2020; Nam et al., 2010).

We also tested whether the age of an individual influenced the decision to help. To do so, we restricted our analysis to birds that had the opportunity to help (98 helping decisions by 90 females and 163 helping decisions by 144 males). For this analysis, we only included birds that had failed to fledge offspring in a particular year and had at least one viable helping option (i.e. a nest of at least one first-order relative containing nestlings) in the study site on the day that its final nesting attempt failed. We also restricted the analysis to individuals for which we had information on RE from failed breeding attempts in the year in question. In these analyses, fixed effects were thus age, ALR, RE and relative lay date.

3 | RESULTS

3.1 | Ageing patterns of breeder reproduction and survival

3.1.1 | Reproduction

We found variable relationships between age and different moments of the reproductive schedule. First, we found no relationship between maternal age and clutch size ($\chi^2 = 0.76$, $p = 0.38$; Figure 2a; Table S1a). Analysing both sexes together, we found a significant quadratic relationship between breeder age and number of fledglings produced when older individuals were clustered as 4+ and individual 1978 was removed ($\chi^2 = 4.28$, $p = 0.04$; Figure 2b; Table S1b). Post-peak analysis revealed this was likely due to early life improvement, with no significant negative linear effect of age on fledgling production for individuals older than 2 (Age 3+: $\beta = -0.20 \pm 0.20$, $\chi^2 = 1.04$, $p = 0.31$). A similar quadratic effect was observed for direct fitness ($\chi^2 = 3.90$, $p = 0.05$; Figure 2c;

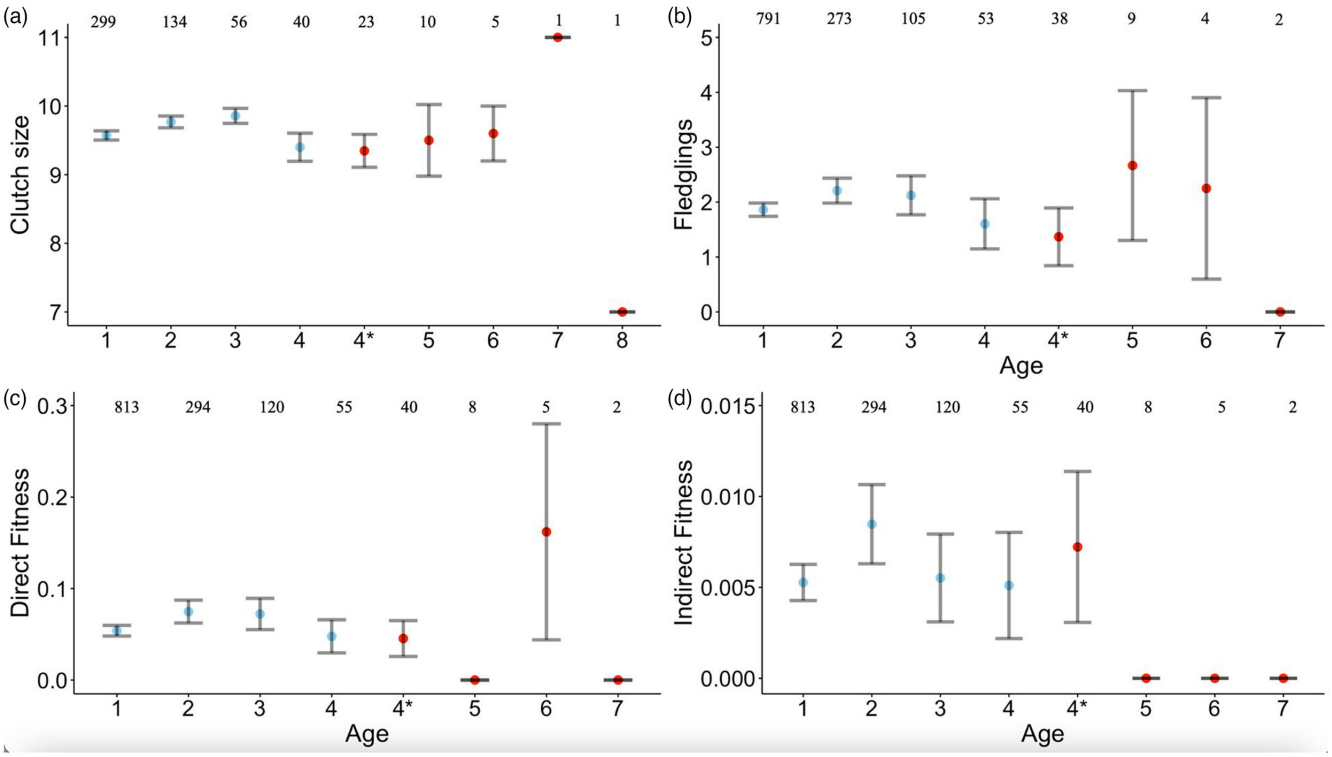


FIGURE 2 Age patterns of reproduction. Mean ± *SE* (a) clutch sizes of females, (b) number of fledglings, (c) direct fitness and (d) indirect fitness in relation to individual age. Results are presented both for when individuals aged 4 and older are grouped as 4+ (blue), and as the raw means for age classes 4 to 7/8 (red). Sample sizes for each age class are displayed above each data point.

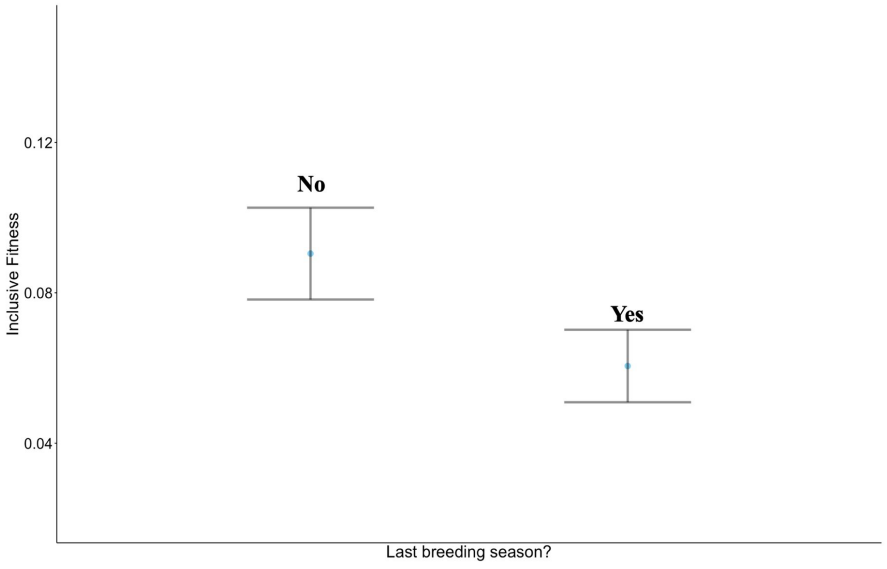


FIGURE 3 Inclusive fitness in an individual's last breeding season. Mean ± *SE* inclusive fitness for individuals in breeding seasons throughout their life span (No) compared to their final breeding season (Yes).

Table S1c) and inclusive fitness ($\chi^2 = 4.28$, $p = 0.04$; Figure S1; Table S1d). These were again due to early life improvements, with no significant negative linear effects of age in post-peak analyses (direct fitness: Age 2+: $\beta = -0.37 \pm 0.27$, $\chi^2 = 2.29$, $p = 0.13$; inclusive fitness: Age 2+: $\beta = -0.28 \pm 0.25$, $\chi^2 = 1.29$, $p = 0.25$). However, we found that individuals gained significantly less fitness in their last year of life. In the case of direct fitness, this effect was marginally significant when individuals were grouped as 4+ ($\beta = -0.53 \pm 0.30$, $\chi^2 = 3.78$, $p = 0.05$) but not when assigned their true age (Table S1c). For inclusive fitness, a significant terminal effect was observed even when individuals were assigned their true age ($\beta = -0.55 \pm 0.27$, $\chi^2 = 4.59$, $p = 0.03$; Figure 3; Table S1d).

In addition to these age effects, we found significant effects of ALR on fledgling production ($\chi^2 = 4.40$, $p = 0.04$) and accrual of direct ($\chi^2 = 4.36$, $p = 0.04$) and inclusive fitness ($\chi^2 = 10.93$, $p < 0.001$; Table S1b–d), suggesting selective disappearance of poorer quality individuals. ALR was not correlated with nestling mass among philopatric individuals or with adult body mass among immigrants ($p > 0.30$); however, among immigrants, adult body mass was significantly positively correlated with lifetime inclusive fitness (Figure S2; $\beta = 0.42 \pm 0.19$, $\chi^2 = 12.36$ $p = 0.001$). For philopatric recruits, we found no evidence that receiving help as a nestling had a significant impact on life span or lifetime accrual of inclusive fitness ($\beta = 0.45 \pm 0.29$, $\chi^2 = 2.42$ $p = 0.12$). Finally, we found no evidence of age effects on a female's propensity to produce extra-pair offspring, nor a male's likelihood of gaining, or losing out to, EPP (Table 2). Furthermore, there were no effects of ALR in either the male or female analysis, indicating that birds with different reproductive life spans did not have significantly different rates of extra-pair paternity.

TABLE 2 Models investigating the effect of age on extra pair paternity (EPP) in females and males. For females, we modelled the probability that a female sired at least one nestling that was not the genetic offspring of the social father. For males, we modelled the probability that the social father was cuckolded. Age at last reproduction (ALR) and brood size (*N*) were also included as fixed effects. Year and bird IDs were included as random effects.

Fixed effects	β	*SE*	χ^2	*p*
Females				
Age	0.65	0.55	1.47	0.22
Age^2	−0.13	0.10	1.82	0.18
ALR	0.004	0.14	0.001	0.97
N	0.002	0.06	0.001	0.97
Males: probability of losing EPP				
Age	−0.43	0.45	1.02	0.31
Age^2	0.10	0.07	2.26	0.13
ALR	−0.07	0.14	0.24	0.63
N	0.01	0.07	0.03	0.86
Males: probability of gaining EPP				
Age	−0.90	1.03	0.85	0.36
Age^2	0.24	0.20	1.90	0.17
ALR	−0.09	0.52	0.03	0.86

Note: Models were fitted with a binomial error structure.

3.1.2 | Survival

Our overall mean rate of survival across the population was lower (0.38) than previously recorded (0.50; Hatchwell, 2016). However, this is likely an artefact of our study sample of individuals. The individuals included in our survival analyses were only those for which we could calculate accurate values of fitness across their whole life span. This therefore biases our sample to birds for which no breeding seasons were missed, and in particular birds observed breeding for a single year (i.e. shorter lived birds). There was no significant effect of age on the probability of adult survival across the whole population ($\chi^2 = 0.05$, $p = 0.81$; Figure 4a; mean survival between years = 0.38), and no difference between the sexes in their age trajectories of survival (Age × Sex: $\chi^2 = 0.34$, $p = 0.56$; Age^2 × Sex: $\chi^2 = 0.08$, $p = 0.78$). We found no significant effect of RE on the probability of survival for females ($\chi^2 = 0.60$, $p = 0.44$) or males ($\chi^2 = 0.23$, $p = 0.63$; Table S1f). Rather, individuals that gained direct fitness in a given year through the production of offspring that successfully fledged and recruited were then more likely to survive to the following year ($\beta = 0.78 \pm 0.34$, $\chi^2 = 5.51$, $p = 0.02$; Figure 4b). When included in models testing for the effects of RE, there was a significant effect of direct fitness on the survival of females ($\beta = 1.45 \pm 0.51$, $\chi^2 = 8.56$, $p = 0.003$) but not males ($\chi^2 = 0.03$, $p = 0.87$; Table S1f). Winter rainfall was also negatively associated with female (-0.73 ± 0.28, $\chi^2 = 5.54$, $p = 0.02$) but not male survival ($\chi^2 = 0.48$, $p = 0.49$; Table S1f).

3.2 | Effects of helpers on breeders' ageing patterns

In line with previous work, we found no effect of helping on fledgling production ($\beta = 0.007 \pm 0.04$, $\chi^2 = 0.04$, $p = 0.85$) but a significant positive effect of the number of helpers on the probability that fledglings recruited into the breeding population ($\beta = 0.16 \pm 0.06$, $\chi^2 = 7.02$, $p = 0.01$). Greater breeder survival to year $n+1$ was not predicted by the presence of helpers for males ($\beta = -0.46 \pm 0.28$, $\chi^2 = 2.83$, $p = 0.09$), nor females ($\beta = -0.10 \pm 0.27$, $\chi^2 = 0.14$, $p = 0.71$). Instead, for males, higher survival to year $n+1$ was associated with a later relative fledge date of offspring in year n ($\beta = 0.04 \pm 0.02$, $\chi^2 = 6.24$, $p = 0.01$), while for females, survival to the following breeding season was negatively related to winter rainfall levels ($\beta = -1.01 \pm 0.29$, $\chi^2 = 11.15$, $p = 0.001$) and positively related to the number of offspring surviving to recruit ($\beta = 0.69 \pm 0.28$, $\chi^2 = 5.95$, $p = 0.01$). We found no significant interactions between helper presence and age (linear nor quadratic) for fledgling production (Age × Helpers: $\chi^2 = 0.08$, $p = 0.77$; Age^2 × Helpers: $\chi^2 = 0.01$, $p = 0.92$) or recruit production (Age × Helpers: $\chi^2 = 0.04$, $p = 0.85$; Age^2 × Helpers: $\chi^2 = 0.08$, $p = 0.77$) nor survival (Age × Helpers: $\chi^2 = 0.59$, $p = 0.44$; Age^2 × Helpers: $\chi^2 = 0.60$, $p = 0.44$). Thus, although helpers boost breeders' production of recruits, they appear to do so in an age-independent manner and do not alter the slope of the age trajectories of breeder fitness. Finally, there were no effects of helpers on breeder future reproduction: receiving help in year *n* did not influence clutch size, relative lay

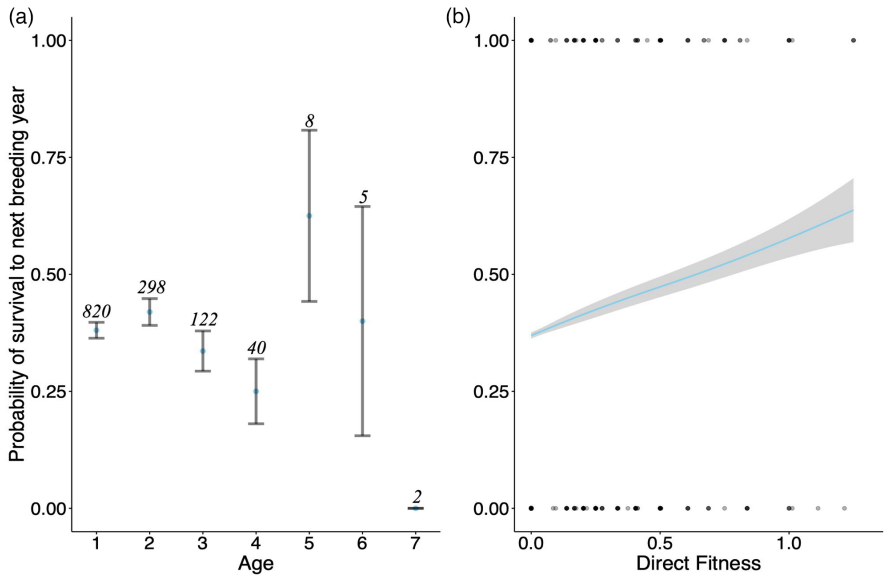


FIGURE 4 Age patterns of survival. (a) Mean ± *SE* probability of adult survival from one breeding season to the next with respect to age. (b) The predicted probability ±*SE* of survival to the next breeding season as a function of an individual's direct fitness in the current breeding season.

date, the probability of fledgling offspring or the probability of accruing direct fitness in year $n+1$ (all $p > 0.57$; Table S2).

3.3 | Ageing patterns of helper behaviour

There was no significant effect of age on the amount of indirect fitness accrued by helpers (Figure 2d), but birds with longer life spans were likely to gain more indirect fitness (Table 3). After finding a positive effect of direct fitness on breeder survival, we tested whether greater nest productivity conferred survival benefits to helpers as well. At the nests where helpers provided help, however, there were no effects of the number of fledglings ($n = 159$: $\beta = -0.02 \pm 0.10$, $\chi^2 = 0.05$, $p = 0.83$), nor the number of recruits ($n = 161$: $\beta = 0.11 \pm 0.11$, $\chi^2 = 0.97$, $p = 0.32$) on the probability that a helper survived to the next year. These results reaffirm the lack of direct fitness benefits of helping in this system (Meade & Hatchwell, 2010).

There was no effect of mean helper age on the probability that a fledgling at a helped nest recruited into the breeding population (Table 3). Greater recruitment was detected among males, due to female-biased dispersal in this study population (Hatchwell, 2016). Furthermore, a higher mean relatedness between a fledgling and its helpers significantly increased the fledgling's probability of recruitment (Table 3). This result is consistent with the finding that helpers increase their provisioning efforts when they have higher relatedness to a brood (Leedale et al., 2018; Leedale et al., 2020; Nam et al., 2010).

Finally, there were no effects of age on an individual's decision to help or not (Table 3). RE was significantly positively associated with the decision to help in females, that is individuals with higher RE in a breeding season were more likely to help ($\beta = 0.78 \pm 0.35$, $\chi^2 = 5.55$, $p = 0.02$). We did not find this effect in males, but were unable to test for a difference between the slopes of RE on the decision to help or not, since RE was calculated differently for the two sexes. In males, but not females, ALR had a significant positive effect on the decision to help (Table 3), indicating that birds with longer life spans were more likely to help in a given year of their life.

4 | DISCUSSION

In this study, we investigated age patterns of fitness in a wild population of long-tailed tits *Aegithalos caudatus*. We found evidence of increased reproductive performance with age from maturity, but a lack of evidence for significant senescent decline. However, breeders gained significantly less fitness in their final year of life. We detected no significant effects of age on survival, which was instead positively correlated with the amount of direct fitness accrued. Helpers had no impact on clutch or brood size, but did have a positive effect on recruit production for breeders in the year that they received help. This effect of helpers on breeder reproduction was (breeder) age-independent, and, furthermore, helper presence did not affect breeder survival. Considering ageing patterns of helpers themselves, we found no effects of age on (a) the decision to help, (b) the probability that a helped fledgling would recruit and (c) the indirect fitness they accrue. Long-tailed tits are a short-lived, small passerine species, threatened by a high rate of extrinsic mortality. The individuals who manage to live substantially longer than average appear to be of higher quality, and our results reflect that this variation in individual quality across the population is of paramount importance for all aspects of this cooperative breeding bird's life history.

4.1 | Breeder ageing patterns and effects of helpers

The extent to which alloparental care can influence the patterns of senescence, and how age in general may contribute to explaining variation in social behaviour remain key questions in evolutionary ecology (Berger et al., 2018). Across species, the effect of helpers on different components of breeder fitness are variable (positive: Khan & Walters, 2002; Berger et al., 2018; Hammers et al., 2019; negative: Brouwer et al., 2006; no effect: Sharp & Clutton-Brock, 2010). Long-tailed tits exhibit an unusual helping system relative to most other

TABLE 3 Models investigating the effect of age on helper behaviour. We investigated the effect of age on (i) indirect fitness accrued, (ii) the probability a helped fledgling recruit into the population and (iii) the decision to engage in helping behaviour. See Supplementary Information for modelling of Terminal Investment (TI).

Fixed effects	β	SE	χ^2	p
Indirect fitness				
Age	0.37	0.72	0.36	0.55
Age^2	−0.14	0.16	1.19	0.28
Sex	2.49	0.80	20.86	**<0.001**
ALR	0.50	0.26	5.18	**0.02**
TI	−0.38	0.63	0.56	0.46
Prob. fledgling recruits				
Helper age[a]	0.11	0.11	0.94	0.33
Sex of fledgling	0.87	0.21	18.07	**<0.001**
Relative fledge date	−0.01	0.02	0.60	0.44
Number of helpers	0.41	0.20	4.23	**0.04**
Relatedness of fledglings to helpers[a]	1.60	0.78	4.28	**0.04**
Decision to help by males				
Age	−0.87	0.84	1.14	0.29
Age^2	0.12	0.18	0.51	0.47
Relative lay date	−0.02	0.03	0.36	0.55
Reproductive effort	0.10	0.20	0.24	0.62
ALR	0.54	0.20	9.08	***0.002***
Decision to help by females				
Age	−1.79	1.44	1.40	0.24
Age^2	0.44	0.28	2.23	0.14
Relative lay date	−0.05	0.06	0.72	0.40
Reproductive effort	0.78	0.35	5.55	**0.02**
ALR	−0.29	0.31	0.96	0.33

Note: The indirect fitness model was fitted with a compound Poisson error structure, while the other two models were fitted with a binomial error structure. Significant ($p < 0.05$) terms are given in bold.

[a]Where breeders were assisted by multiple helpers, average values across all helpers were calculated.

cooperative breeders, in which individuals may redirect care to related broods after failing to breed themselves. In this study, helpers were found to boost breeder productivity in terms of number of recruits, in line with previous work (Hatchwell et al., 2014), but had no effect on fledgling number or breeder survival. When comparing nests that received and did not receive help there were no significant differences between the slopes of age trajectories of recruit production. This suggests that although helpers boost the magnitude of breeder recruit production, the effect is independent of breeder age. While previous work on this system had suggested that male breeders benefited from the presence of helpers when raising large broods (Meade et al., 2010), such an effect was not evident in our analysis, which included an additional 11 years of data (Figure S3). Indirect fitness returns from helping thus appear to be generated primarily through the enhancement of offspring recruitment in this species. This conclusion is consistent with the negligible contribution (2.5%) that load-lightening makes to the indirect fitness benefits that helpers receive from their cooperative behaviour (Hatchwell et al., 2014).

We found consistent evidence of increasing breeder reproductive performance in early life. This is a common pattern among animals (Bouwhuis et al., 2009; Clutton-Brock, 1984; Sharp & Clutton-Brock, 2010) that is typically attributed to differential mortality with regard to individual quality (Balbontin et al., 2007; Reid et al., 2003). In our results, this was supported by significant effects of ALR on fledgling production, and direct and indirect fitness accrual, suggesting selective disappearance of poor-quality individuals. We did not, however, find any effects of age or ALR on the probability of a female engaging in extra-pair mating, or a male gaining or losing offspring to EPP, adding to the variable results for age effects on EPP among passerines (Cooper et al., 2021; Hsu et al., 2017; Raj Pant et al., 2020). The increase in breeder reproductive performance we found was not followed by significant late-life decline, which can be difficult to detect in wild populations due to low sampling of older age classes (Nussey et al., 2013). We addressed this issue by using a longitudinal dataset spanning 25 years of individual-level data and by grouping individuals aged 4 and older into a 4+ category. The quadratic relationships for fitness components (Figure 2) suggest a senescent pattern, but in all components of reproduction, the mean estimate for individuals aged 4 is lower than those of age 4+, meaning that the oldest individuals in the population are driving up the average. We suggest therefore that the absence of significant quadratic effects may be explained by the high performance of the longest-lived individuals masking weak senescence in both reproduction and survival, rather than by a complete absence of senescence in long-tailed tits. Although there are growing theoretical (Baudisch, 2011) and empirical (Péron et al., 2019) arguments that senescence and life span are independent, it may be more difficult, statistically, to detect senescence in species that are short lived where estimates of survival and reproductive performance are based on annual measures. Reproduction in long-tailed tits is annual, but more fine-grained (e.g. seasonal) estimates of survival within years, combined with other proxies of senescence such as body condition, might provide greater power for detecting senescence in this and other short-lived species.

4.2 | Variation in individual quality

In long-tailed tits, ALR is consistently associated with higher fitness (Table 1). Evidence of differences in reproductive life spans among individuals determining age-specific fitness trajectories is widespread across species, from fishes (Suzuki et al., 2010) to baboons

(McLean et al., 2019). As we have done here, studies usually consider ALR a reasonable indicator of individual quality, but ALR itself does not describe a causal mechanism that determines 'quality'. In reality, 'quality' will be determined from a plethora of variables, including developmental conditions, body mass, acquisition of resources, luck and better nest locations with reduced risks of predation (Lim et al., 2014). Can we delve deeper into what determines such quality?

In birds, metrics such as relative lay date (Verhulst & Nilsson, 2008) and body mass may offer finer resolution indices of individual quality. Indeed, Meade and Hatchwell (2010) reported that among those long-tailed tits that failed to breed successfully and had relatives available to help, helpers had earlier relative lay dates compared to non-helpers. Body mass is known to correlate with age-specific reproduction and survival both within and across species (e.g. Péron et al., 2019), and for long-tailed tits is important in determining hierarchies in non-breeding winter flocks (Napper et al., 2013). We found no evidence that body mass at maturity (immigrant breeders) or as a nestling (philopatric breeders) was associated with life span (ALR), but immigrant body mass was associated with higher lifetime inclusive fitness (Figure S2). Furthermore, some variation in individual 'quality' may not be attributable to morphological traits, but may instead be determined by environmental factors. Long-tailed tits are not territorial (Hatchwell, 2016), but spatial autocorrelation of reproductive performance (e.g. Marrot et al., 2015) has not yet been investigated. Individuals that are able to survive a year may gain information on what particular areas are likely to give them the best chance at successful reproduction.

4.3 | Positive covariation of individual reproduction and survival

Rather than an expected trade-off between survival and reproduction, we instead found a positive correlation between breeder reproduction and survival throughout this study. The most likely explanation for the apparent absence of trade-off between survival and reproduction is differences among individuals in both resource allocation and acquisition (Van Noordwijk & De Jong, 1986). Life-history trade-offs between current reproduction and future survival and reproduction are often revealed only when experiments are used to break the link between investment in different functions, caused by these individual quality effects (Zhang & Hood, 2016). There are other, non-mutually exclusive, explanations for why survival and reproduction in long-tailed tits may positively covary. As a corollary to an individual's direct fitness accrual being significantly correlated with their chance of survival, we also found that individuals suffered significant decreases in fitness in their final year of life (Figure 1d). This 'terminal decline' (Coulson & Fairweather, 2001) could be due to rapid physiological change, with individuals of different quality senescing at varying rates. Countering this, however, we found no evidence of terminal effect in fledgling production (Table S1), though we cannot rule out the possibility that other indicators of fledgling quality, such as body mass, may reflect parental terminal decline.

An alternative explanation, however, could be that higher rates of reproduction really do boost breeder survival through social benefits of producing offspring. In red-cockaded woodpeckers *Picoides borealis*, each juvenile reduces breeder mortality rate by 16% for males and 26% for females after accounting for helping and territory quality (Khan & Walters, 2002; Walters & Garcia, 2016). This finding was attributed to larger group sizes providing better protection against predation (Walters & Garcia, 2016). Unlike red-cockaded woodpeckers, long-tailed tits are not territorial and live in large flocks with relatively fluid membership outside the short breeding season. These non-breeding flocks are typically composed of all birds associated with a successful nest (breeders, offspring and any helpers), augmented by failed breeders who are sometimes relatives of the breeders (Napper & Hatchwell, 2016). Switches of individuals between flocks occur regularly, as do flock mergers, and there is no evidence that helping is a strategy to buy membership of a winter flock by birds that have failed to produce their own brood (McGowan et al., 2006; Napper & Hatchwell, 2016). Flock members forage together and gain thermoregulatory benefits at night from roosting in linear huddles (Hatchwell et al., 2009). Previous research suggests that dominance (predicted by sex and body size), rather than kinship, determines the position of individuals in linear huddles and thus the thermoregulatory benefits of roosting (McGowan et al., 2006; Napper et al., 2013). Napper et al. (2013) also showed that kinship fails to predict dominance hierarchy within flocks. Increased offspring production, and an attendant increase in kinship to the non-breeding flock, is thus unlikely to benefit a breeder in terms of enhancing their position within a roost. There could, however, be survival benefits not yet uncovered of entering a flock with more individuals from your successful nest who are all likely to be relatives. Kinship, for example, positively affects the strength of social associations between individuals in non-breeding flocks (Napper & Hatchwell, 2016). Reduced competition over resources, reduced predation risk or other benefits due to these stronger associations may therefore provide survival benefits to offspring and parents leading to co-dependent survival. If producing more offspring does indeed bring survival benefits for breeders, then despite the lack of direct evidence of the presence of helpers boosting breeder survival, helpers may actually augment breeder survival indirectly through the increased production of recruits (Walters & Garcia, 2016).

4.4 | Ageing patterns of helping behaviour

Age is a principal cause of variation in helping behaviour across cooperatively breeding species. In the majority of cooperative bird species, offspring delay dispersal to remain in their natal territory and help their parents. In these systems, helping is therefore biased towards younger age classes, while older individuals specialise on reproduction (Koenig & Dickinson, 2004). A similar bias is seen in other taxa; in stenogastrine wasps *Liostenogaster flavolineata*, for example, Field et al. (2006) showed that older individuals work less hard in an age-based queue of inheritance. However, the reverse situation is also seen: in both apostlebirds *Struthidea cinereal* and Damaraland mole rats *Fukomys damarensis*, investment in cooperation increases, rather than decreases, with age (Woxvold et al., 2006; Zöttl et al., 2016). An individual's incentive to help will be driven by their possible returns

from the help and the effects on their future fitness. As individuals age, direct fitness opportunities may be reduced due to reproductive competition with younger individuals, favouring menopause and female abstention from reproduction in species such as humans or killer whales *Orcinus orca* where intra-group relatedness is high and indirect fitness can be gained (Cant & Johnstone, 2008; Johnstone & Cant, 2010). Alternatively, direct fitness opportunities may increase with age, for example when dominant breeders are usually older individuals. In these systems, such as meerkats *Suricata suricatta* or stenogastrine wasps (Field et al., 2006; Sharp & Clutton-Brock, 2010), we may expect helping behaviour to decline as individuals age.

In the redirected helping system of long-tailed tits, individuals only help if they fail in their own breeding attempts (Hatchwell, 2016). No direct benefits of helping have been detected (Meade & Hatchwell, 2010; this study), and therefore helping behaviour can be fully understood with respect to returns from indirect fitness (Hatchwell et al., 2014). We found no evidence of age effects on helping. Rather, indirect fitness accrual and the decision to help appear to be better predicted by differences in individual quality. ALR was significantly correlated with accrual of indirect fitness and a male's decision to help, supporting evidence that helping is condition dependent (McGowan et al., 2003). Although ALR was not a significant predictor for whether females will help, we found evidence that a female's decision was significantly correlated with her RE in that year. Given that all individuals in the analysis had the opportunity to help, that is a first-order relative with a brood was available in the population, it raises the question why individuals that expended little RE would not choose to help. This may point to evidence of condition-dependent helping in females. The females that had a larger RE and then decided to help could do so because they were of intrinsically higher quality.

Although we found no evidence here, a prospective helper's decision to help may also be influenced by the age of recipients. Where helping yields significant indirect fitness benefits to helpers, as it does in long-tailed tits, Hamilton's (1964) rule suggests, all else being equal, that greater relatedness between actor and recipient will lead to greater returns for the actor. Potential recipients can vary in other ways, however, that may determine the benefit they will receive from being helped. Having helpers when young may aid inexperienced breeders (Magrath, 2001), while receiving help when old may alleviate greater costs of reproduction due to senescence (Hammers et al., 2019). Helpers may not, therefore, value all relatives equally if their phenotypic differences affect the benefits a helper can gain (Schulman & Rubenstein, 1983).

5 | CONCLUSIONS - INDIVIDUAL HETEROGENEITY EXPLAINS AGEING PATTERNS IN A COOPERATIVE BREEDER

Studies are accumulating that suggest sociality may play a role in explaining variation in life span across animals (Lucas & Keller, 2020). Although rare across species, the nature of the facultative redirected helping system in long-tailed tits allowed us to separate age-related changes in direct and indirect fitness and also facilitated a specific test for consequences of age on the decision of individuals to help. Our findings especially highlight that differences in individual quality within populations, especially in those where the risk of extrinsic mortality is extremely high, are of paramount importance when investigating ageing patterns of survival, reproduction and helping. In wild populations, the occurrence of senescence can be hard to detect (Nussey et al., 2008) and requires longitudinal datasets following many individuals. Often, senescence is revealed only when the number of sampled individuals who live substantially longer accumulates. In long-tailed tits, these individuals seem to be of intrinsically higher quality, allowing them to escape the trade-offs between survival and reproduction, and not fall victim to their old age.

AUTHORS' CONTRIBUTIONS

B.J.H. managed the field study and collected data on which analyses are based over 25 years; M.R., N.J.S. and J.P.G. compiled and analysed data; M.R. wrote the manuscript along with N.J.S., B.J.H. and J.P.G. revised and edited the manuscript. All authors gave final approval for publication.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

Data available from the Dryad Digital Repository https://doi.org/10.5061/dryad.g79cnp5rz (Roper et al., 2022).

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SUPPORTING INFORMATION

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This file contains Supporting Information for "**Individual variation explains aging patterns in a cooperatively breeding bird, the long-tailed tit (*Aegithalos caudatus*)"**

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Supplementary Online Methods

Biometrics

Although we did not have biometric information for every year of an individual's life, we had information on body size from at least one year for the majority of the 867 individuals for whom we had lifetime recruit production data. We tested for correlations between body mass on lifespan and lifetime inclusive fitness, accounting for sex and cohort. We performed

separate models for philopatric and immigrant individuals, as body mass is measured at maturity for immigrants and the nestling stage for philopatric individuals. Since hatch date was determined through daily checks of nests, there is a 24-hour error around the estimate of nestling age. Because body mass changes rapidly during the nestling period, we chose to model body mass with tarsus length (which provides a relatively inflexible measure of age) as a covariate in order to obtain an estimate of relative body mass.

Modelling terminal age effects

As well as considering effects of age, sex and ALR on individual reproduction, there is the potential for terminal effects of age to occur, either positive (Clutton-Brock 1984) or negative (Coulson & Fairweather 2001). We were able to account for terminal investment (TI) in fledgling production and direct fitness accrual. We added a two-level class variable which categorised breeding attempts as being last (1) or not (0), and then quantified TI by comparing the difference between a birds penultimate and ultimate year of life (two data points per individual for individuals that lived for more than one year). Given that long-tailed tits attempt to breed in every year of their life, the last breeding attempt is equivalent to their final year of life. Due to this feature of long-tailed tit breeding, ALR and TI are directly correlated. This issue leads to co-linearity within models in which both variables are included, and so we analysed TI in separate models without ALR.

Extrapair paternity

We used genetic information for the social parental pair and all other ringed males present in the population to assign paternity for each nestling that was successfully genotyped between 1994 and 2019. Nestlings were excluded when we didn't have the genotype of either the mother or social father. Paternity was checked using the likelihood-based approach implemented in

CERVUS 3.0.7 (Marshall *et al.* 1998; Kalinowski *et al.* 2007), and whole broods of nestlings were excluded when paternity assignment was ambiguous for at least one nestling. By this we mean the parentage assignment was inconclusive in failing to rule out that a nestling was sired by a male other than the social male.

Figure S1 Mean ± SE Inclusive Fitness in relation to individual age. Results are presented both for when individuals aged 4 and older are grouped as 4+ (blue), and as the raw means for age classes 4 to 7 (red). Sample sizes for each age class are displayed above each data point.

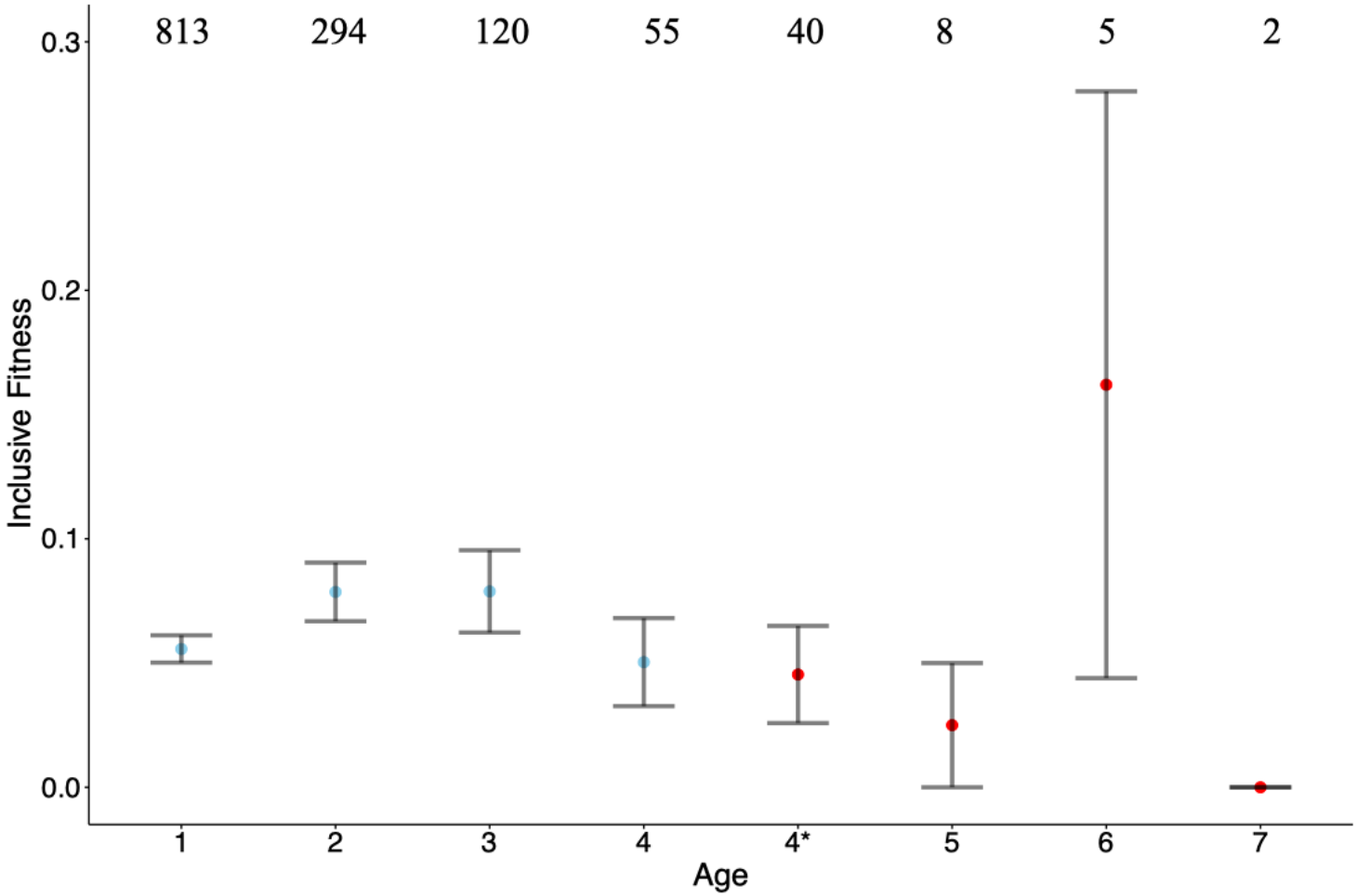


Figure S2 Predicted mean ± SE probability a female helped (1) or not (0) in a breeding year when the opportunity was available, as a function of her reproductive effort in the current season.

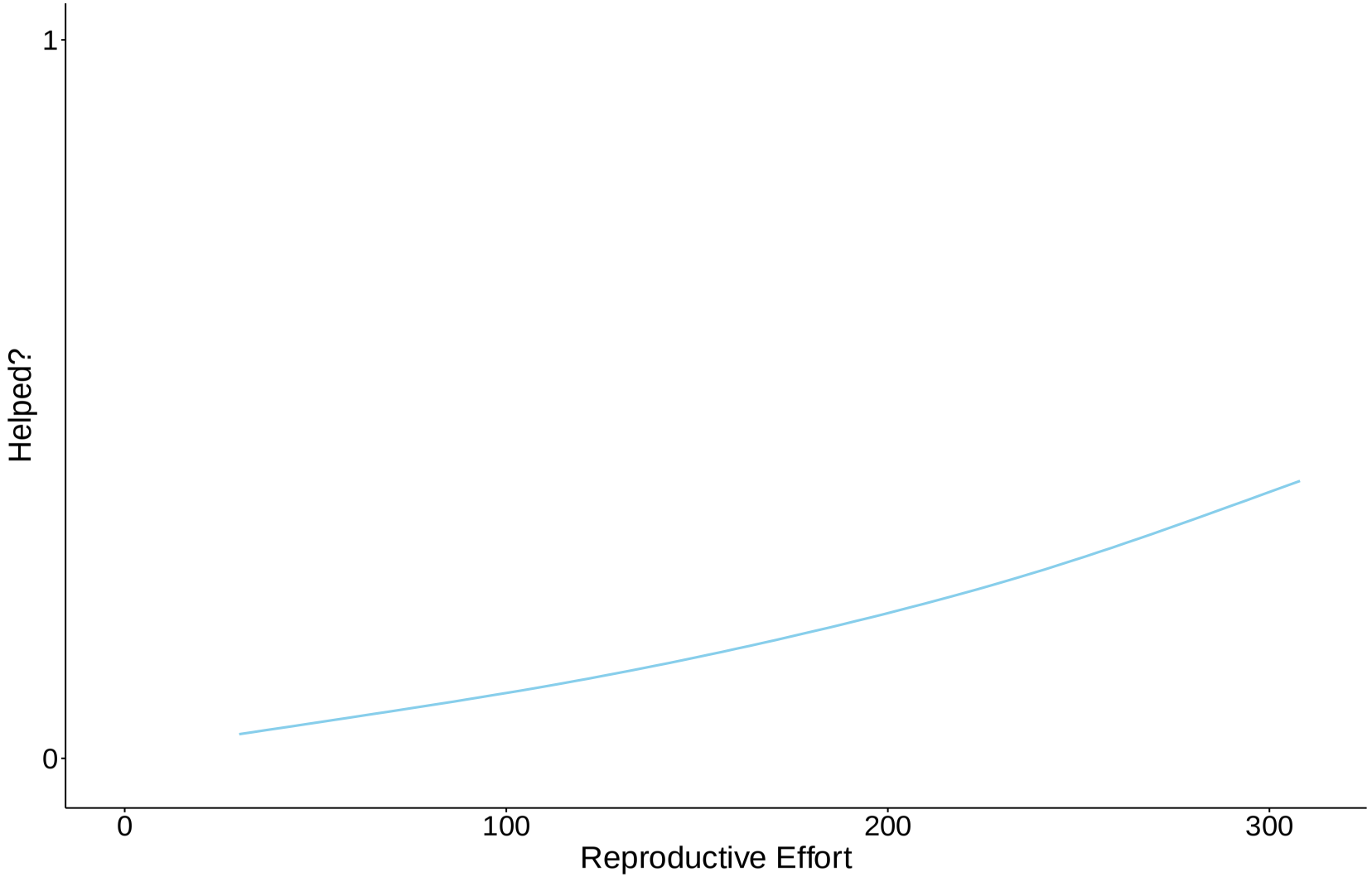


Figure S3 Predicted mean ± SE relationship between immigrant mass at maturity and lifetime inclusive fitness accrued. Lifetime inclusive fitness is measured as genetic offspring equivalents, stripped of the effects of helpers (See Methods for full details).

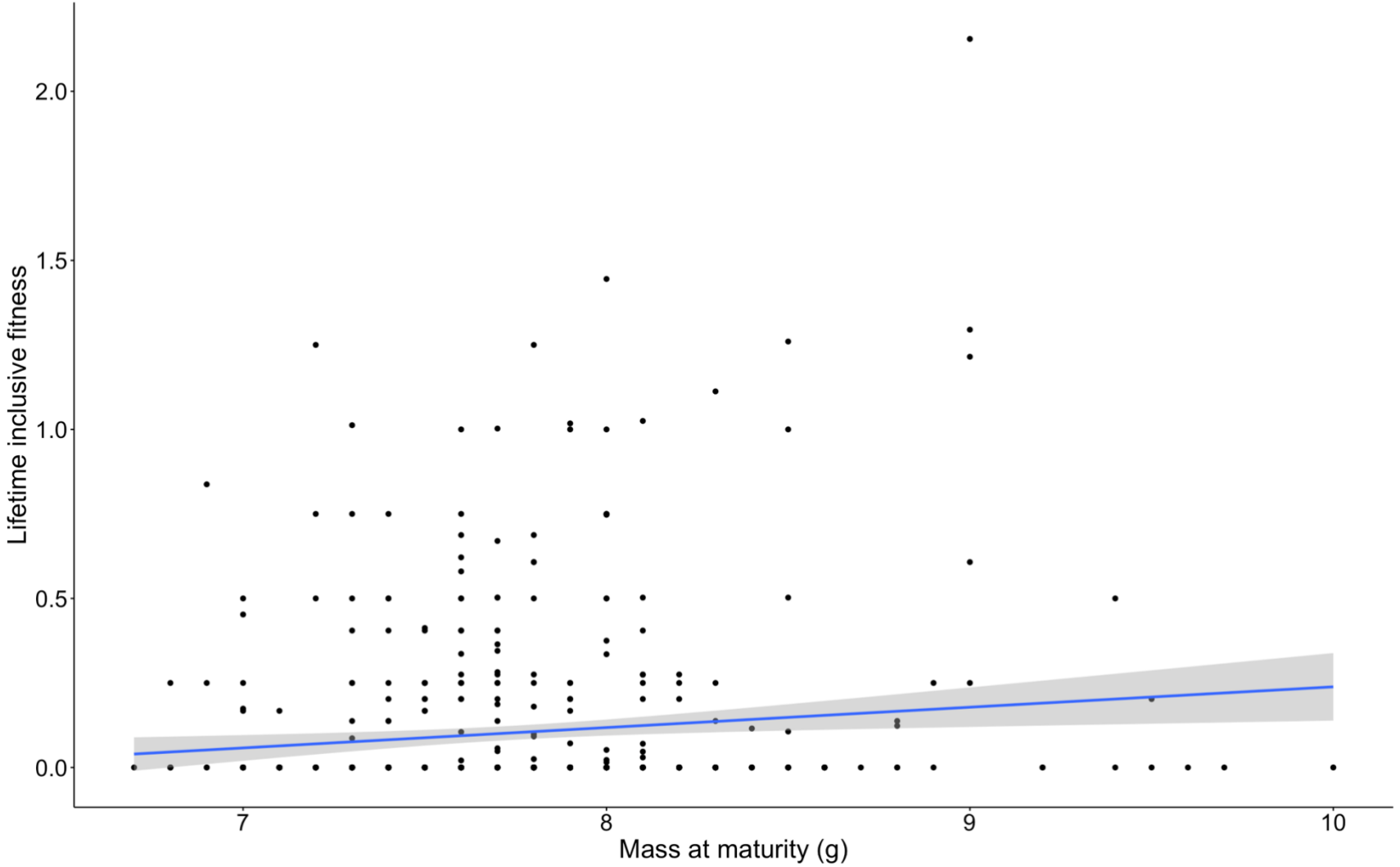


Figure S4. Mean ± SE predicted relationship between the presence of helpers and breeder survival as a function of brood size for male and female breeders.

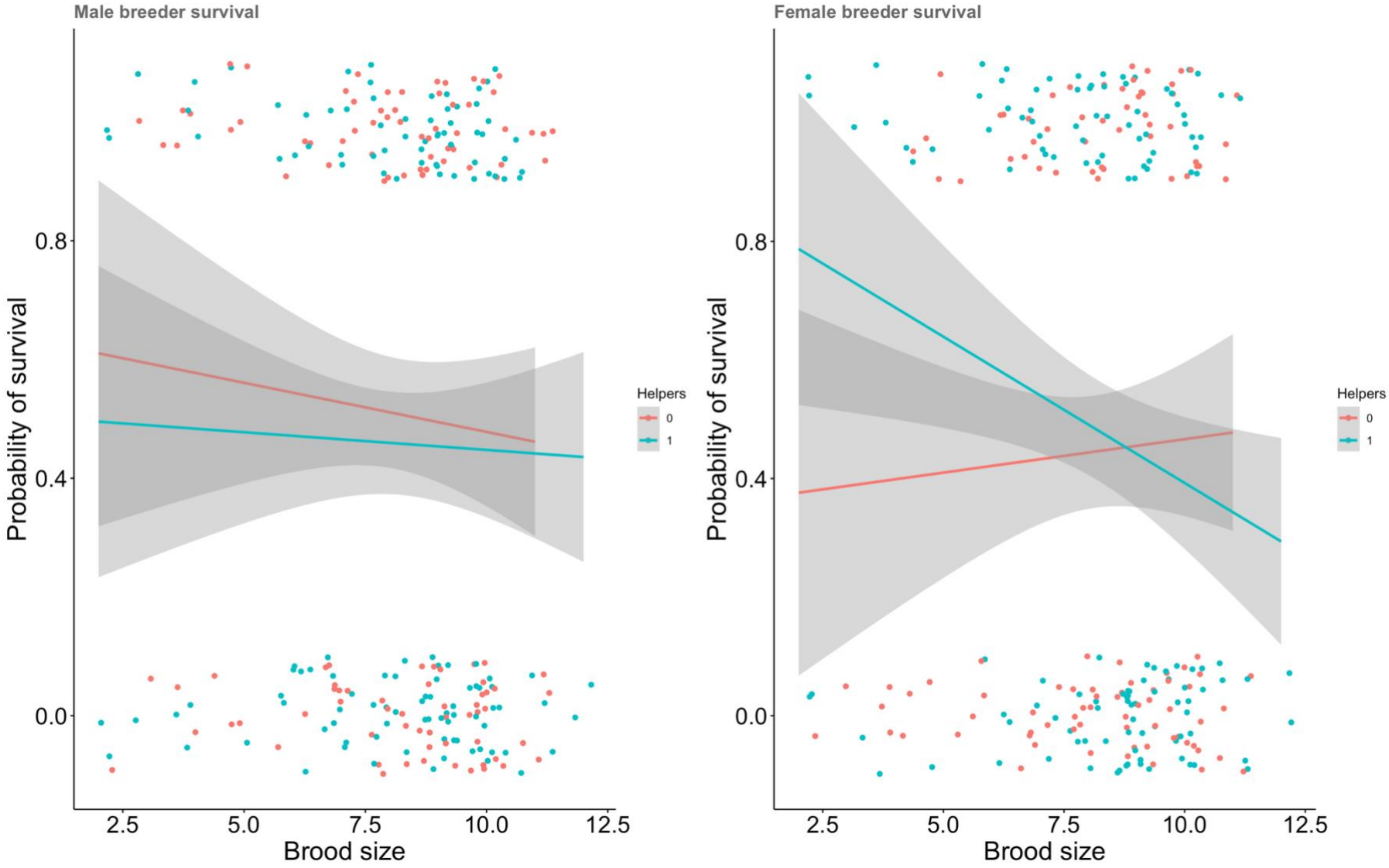


Table S2a. Models predicting the clutch size of a female according to its age, age at last reproduction (ALR), and whether the nest received help later in the breeding season. Random effects included were the year of study (1994-2019) and the IDs of both parents of the clutch.

Fixed effects	β	SE	χ^2	P
Age	0.04	0.05	0.76	0.38
Age^2	-0.006	0.007	0.72	0.39
ALR	-0.004	0.01	0.10	0.75
Helpers	<0.001	0.03	<0.001	0.99

Models were fitted with a poisson error structure. Significant terms are given in bold. See Methods for full details.

Table S2b. Models predicting the number of fledglings produced by an individual according to their age, age at last reproduction (ALR), and terminal investment (TI). Random effects included were the year of study (1994-2019) and the ID of the breeder. Results are shown for all individuals assigned their true ages, and for additional analyses in which individuals aged 4 years or more are grouped as 4+ and/or individual 1978 is removed (for full details, see Methods).

Fixed effects	β	SE	χ^2	P
True age				
Age	0.21	0.29	0.62	0.43
Age^2	-0.06	0.06	1.49	0.22
ALR	0.15	0.07	4.40	**0.04**
Sex: Male	-0.18	0.13	2.26	0.13
Age:Sex	-0.54	0.50	1.20	0.27
Age^2:Sex	0.13	0.10	1.62	0.20
Grouped as 4+				
Age	0.54	0.41	1.92	0.17
Age^2	-0.15	0.09	2.76	0.10
ALR	0.15	0.07	4.46	**0.03**
Sex: Male	-0.18	0.13	2.23	0.14
Grouped as 4+; without 1978				
Age	0.70	0.42	2.98	0.08
Age^2	-0.19	0.10	4.28	**0.04**
ALR	0.15	0.07	4.40	**0.04**
Sex: Male	-0.19	0.13	2.48	0.12
Terminal investment				
Age	-0.05	0.10	0.27	0.60
Sex: Male	-0.20	0.17	1.37	0.24
TI	-0.03	0.20	0.03	0.87

Models were fitted with a compound Poisson error structure. Significant ($p < 0.05$) terms are given in bold.

Table S2c. Models predicting direct (DF) fitness accrued by an individual according to their age, age at last reproduction (ALR), and terminal investment (TI). Random effects included were the year of study (1994-2019) and the ID of the breeder. Results are shown for all individuals assigned their true ages, and for additional analyses in which individuals aged 4 years or more are grouped as 4+ and/or individual 1978 is removed (for full details, see Methods).

Fixed effects	β	SE	χ^2	P
True age				
Age	0.27	0.32	0.82	0.36
Age^2	-0.07	0.06	1.72	0.19
ALR	0.22	0.11	4.36	**0.04**
Sex: Male	-0.12	0.14	0.87	0.35
Age:Sex	-0.81	0.76	1.28	0.26
Age^2:Sex	0.23	0.17	2.10	0.15
Grouped as 4+	0.79	0.49	2.98	0.08
Age	-0.20	0.11	3.85	**0.05**
Age^2				
ALR	0.21	0.11	4.30	**0.04**
Sex: Male	-0.13	0.14	1.07	0.30
Grouped as 4+; without 1978				
Age	0.79	0.52	2.66	0.10
Age^2	-0.21	0.12	3.46	**0.05**
ALR	0.21	0.12	3.90	**0.04**
Sex: Male	-0.15	0.14	1.23	0.27
Terminal investment				
Age	0.09	0.16	0.43	0.50
Sex	-0.04	0.23	0.04	0.84
TI	-0.46	0.30	3.00	0.08

Models were fitted with a compound Poisson error structure. Significant ($p < 0.05$) terms are given in bold..

Table S2d. Models predicting Inclusive fitness (IF) accrued by an individual according to their age, age at last reproduction (ALR), and terminal investment (TI). Random effects included were the year of study (1994-2019) and the ID of the breeder. Results are shown for all individuals assigned their true ages, and for additional analyses in which individuals aged 4 years or more are grouped as 4+ and/or individual 1978 is removed (for full details, see Methods).

Fixed effects	β	SE	χ^2	P
True age				
Age	0.25	0.31	0.75	0.39
Age^2	-0.08	0.06	2.02	0.16
ALR	0.30	0.10	10.93	**<0.001**
Sex: Male	0.10	0.14	0.54	0.46
Age:Sex	-0.06	0.66	0.01	0.92
Age^2:Sex	0.04	0.14	0.12	0.73
Grouped as 4+				
Age	0.70	0.47	2.45	0.12
Age^2	-0.19	0.11	3.56	0.06
ALR	0.29	0.09	10.54	**0.001**
Sex: Male	0.09	0.14	0.44	0.51
Grouped as 4+; without 1978				
Age	0.79	0.49	2.91	0.09
Age^2	-0.22	0.11	4.28	**0.04**
ALR	0.29	0.10	10.05	**0.002**
Sex: Male	0.08	0.14	0.39	0.53
Terminal investment				
Age	0.11	0.14	0.75	0.39
Sex	0.04	0.23	0.04	0.84
TI	-0.55	0.27	4.59	**0.03**

Models were fitted with a compound Poisson error structure. Significant ($p < 0.05$) terms are given in bold.

Table S2e. Models investigating the effect of age on extrapair paternity (EPP) in females and males. For females, we modelled the probability that a female sired at least one nestling that was not the genetic offspring of the social father. For males, we modelled the probability that the social father was cuckolded. Age at last reproduction (ALR) and brood size (N) were also included as fixed effects. Year and bird IDs were included as random effects.

Fixed effects	β	SE	χ^2	P
Females				
Age	0.65	0.55	1.47	0.22
Age^2	-0.13	0.10	1.82	0.18
ALR	0.004	0.14	0.001	0.97
N	0.002	0.06	0.001	0.97
Males: probability of losing EPP				
Age	-0.43	0.45	1.02	0.31
Age^2	0.10	0.07	2.26	0.13
ALR	-0.07	0.14	0.24	0.63
N	0.01	0.07	0.03	0.86
Males: probability of gaining EPP				
Age	-0.90	1.03	0.85	0.36
Age^2	0.24	0.20	1.90	0.17
ALR	-0.09	0.52	0.03	0.86

Models were fitted with a binomial error structure. Significant ($p < 0.05$) terms are given in bold.

Table S2f. Models predicting the probability of survival of adult long tailed tits according to the individual's age, sex, direct fitness and reproductive effort (RE) in a given year. Mean daily rainfall (mm) over winter was also included as a fixed effect. Random effects were the year of study (1995-2018) and the ID of the individual. Results are presented for the whole population (without RE as a covariate) and separately for females and males for whom RE measures were available (for full details, see Methods).

Model and fixed effects	β	SE	χ^2	P
Whole population				
Age	0.05	0.22	0.05	0.81
Age^2	-0.02	0.04	0.29	0.59
Sex	0.20	0.12	2.99	0.08
Direct fitness	0.78	0.34	5.51	**0.02**
Winter rainfall	0.23	0.41	0.34	0.56
Age:Sex	-0.27	0.44	0.36	0.55
Age^2:Sex	0.02	0.09	0.07	0.79
Grouped as 4+				
Age	0.36	0.36	0.99	0.32
Age^2	-0.11	0.08	1.71	0.19
Sex	0.20	0.12	2.85	0.09
Direct fitness	0.91	0.32	8.12	0.004
Winter rainfall	-0.22	0.14	2.45	0.12
Females				
Age	0.69	0.49	2.35	0.13
Age^2	-0.15	0.11	2.58	0.11
RE	0.09	0.11	0.60	0.44
Direct fitness	1.45	0.51	8.56	**0.003**
Winter rainfall	-0.73	0.28	5.54	**0.02**
Males				
Age	-0.01	0.47	0.001	0.98
Age^2	-0.006	0.10	0.003	0.96
RE	0.06	0.12	0.23	0.63
Direct fitness	-0.10	0.58	0.027	0.87
Winter rainfall	-0.23	0.33	0.48	0.49

Models were fitted with a binomial error structure. Significant terms are given in bold.

Table S3. Models investigating the effect of helpers on breeder age-trajectories of future reproduction in year n+1. We included fixed effects of age (linear and quadratic), brood size, relative fledge date (to 1st May), winter rainfall and spring temperature, whether the individual produced recruits or not and the presence of helpers. Random effects included in all models were individual ID and Year.

Fixed effects	β	SE	χ^2	P
Clutch Size				
Age in year n+1	0.03	0.28	0.02	0.93
Age^2 in year n+1	-0.005	0.04	0.02	0.90
Helpers in year n	-0.01	0.09	0.02	0.89
ALR	-0.003	0.04	0.07	0.93
Brood size year n	0.001	0.02	<0.001	0.99
Recruits in year n	0.06	0.10	0.34	0.56
Relative fledge date year n	0.001	0.005	0.05	0.83
Spring temperature	-0.001	0.03	0.021	0.96
Winter rainfall	0.01	0.09	0.02	0.89
Lay Date				
Age in year n+1	3.70	2.54	2.13	0.14
Age^2 in year n+1	-0.55	0.37	2.33	0.13
Helpers in year n	-0.56	1.00	0.31	0.58
ALR	-0.13	0.50	0.07	0.79
Brood size year n	-0.51	0.23	4.83	**0.03**
Recruits in year n	-0.11	1.13	0.002	0.96
Relative fledge date year n	0.15	0.06	6.14	**0.01**
Spring temperature	-2.27	0.63	11.98	**0.001**
Winter rainfall	3.19	2.26	2.22	0.14
Fledglings				
Age in year n+1	-0.93	0.92	1.06	0.30
Age^2 in year n+1	0.17	0.13	1.70	0.19
Helpers in year n	-0.05	0.31	0.03	0.87
ALR	-0.03	0.15	0.05	0.82
Brood size year n	0.01	0.08	0.03	0.87
Recruits in year n	0.36	0.33	1.21	0.27
Relative fledge date year n	-0.01	0.02	0.33	0.56
Spring temperature	0.08	0.10	0.61	0.44
Winter rainfall	0.13	0.37	0.12	0.72
Direct Fitness				
Age in year n+1	-1.76	1.55	1.81	0.18
Age^2 in year n+1	0.27	0.22	2.29	0.13
Helpers in year n	0.18	0.47	0.14	0.71
ALR	0.08	0.24	0.10	0.75
Brood size year n	0.02	0.11	0.02	0.88
Recruits in year n	-0.28	0.54	0.31	0.58
Relative fledge date year n	0.001	0.03	0.08	0.78
Spring temperature	0.29	0.19	3.54	0.06
Winter rainfall	0.59	0.56	1.26	0.26

Clutch size and relative lay date were modelled with a gaussian error structure, while survival, fledgling production and direct fitness were modelled with binomial error structure. Significant ($p < 0.05$) terms are given in bold.

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7 | Discussion

In this thesis, I have studied the variability of ageing and senescence in wild populations of multicellular organisms. I have focused both on how long individuals live (ageing), and the distribution of mortality and reproduction across their life course that determines rates of senescence. In particular, I have focused on social species, in which the model (**Chapter 4**) and comparative analysis (**Chapter 5**) I have presented suggest that sociality contributes to previously unexplained variation in how species age (Jones *et al.* 2014; Baudisch & Vaupel 2012). Each chapter I have presented throughout this thesis has its own discussion, which I shall not repeat. Instead, I will focus on general themes and future directions. I will begin by discussing the concept of the 'force of selection', that is central to the evolutionary theory for senescence and all nuances. Then, I will focus on future directions for the study of senescence in social species, both theoretical and empirical, and suggest new areas for investigation.

7.1 The evolution of senescence

7.1.1 The force of selection

As discussed throughout this thesis, Hamilton's (1966) model provided the first mathematically grounded argument for why senescence could evolve. The concept of the 'force of selection' has since been ingrained in the minds of evolutionary biologists who study senescence. A weakening force of selection in older age classes, meaning that alleles that invade with detrimental effects are less likely to be eliminated by natural selection, facilitates the evolution of senescence. Using the same logic, I argued in **Chapter 2** that if the force of selection does not decline in later age classes, or the decline is less pronounced, then senescence should be more strongly selected against. The diversity exhibited across species distributions of mortality and reproduction across their life course are not at odds with the force of selection argument. Hamilton was not incorrect, but perhaps his slightly pre-emptive statement that "senescence cannot be avoided by any conceivable organism" (Hamilton 1996) has fuelled a desire to treat model assumptions as deficiencies (Vapuel *et al.* 2004), rather than simply assumptions. The aphorism usually attributed to the statistician George Box that "All models are wrong, but some are useful" applies here. Hamilton's model was exceptionally useful, in

weaving together verbal arguments of mutation accumulation (Medawar 1952) and antagonistic pleiotropy (Williams 1957), and providing logic for senescence. Breaking assumptions of his model of senescence, however, is an important exercise that many authors have conducted (Lee 2003; Vaupel *et al.* 2004; Baudisch 2005; Caswell & Salguero-Gómez 2013; **Chapter** 4). These exercises have illuminated why Hamilton's model cannot predict the full scale of variation in senescence, but have also shown that the force of selection is, nonetheless, the central concept to the evolution of senescence.

There are several breakable assumptions in the structure and methodology of Hamilton's (1966) model. The first is the assumption that the Malthusian parameter defines Darwinian fitness. Taken from Fisher (1930), this assumption built on mathematical treatments of selection in age-structured populations from Haldane (1927) and Norton (1928), and has since been extended by work from Charlesworth (1994) to show that the effect of an allele on the Malthusian parameter is a reliable guide for the evolutionary fate of the allele (Rose *et al.* 2007). As discussed extensively in **Chapters 4** and **5**, this assumption ignores species with social interactions between individuals, where inclusive fitness is a more appropriate measure of Darwinian fitness. I have shown how extending Hamilton's logic to inclusive fitness forces of selection can allow the post-reproductive force of selection on survival to remain above zero, and generally that age-specific forces of selection will be modulated by age-class variations in social behaviour. Still, however, the logic that a declining force of selection (in this case an inclusive fitness force of selection) will facilitate the evolution of senescence holds.

A second set of major assumptions Hamilton made were the chosen 'indicators of selection', *i.e.* the particular parameters he chose to represent the effect of a mutation. For example, for the force of selection on survival, Hamilton (1966) took the derivative of the Euler-Lotka equation with respect to ln $p(x)$, where an increase in ln $p(x)$ is equivalent to a reduction in mortality between age x and $x+1$. Baudisch (2005) performed an exercise in which she used alternative indicators of selection, *i.e.* taking the derivate of the Euler-Lotka equation with respect to alternative parameters representing survival and mortality. The results showed that the forces of selection in these cases need not always decline with age. The argument of 'which indicators are better' is still an open question, which relies on a deeper empirical understanding of the genetics underlying age-specific mutations. This need for understanding includes the rate at which mutations occur, how this rate varies by age, and the effect (*e.g.* additive *vs.*

proportional) of mutations on age-specific survival and reproduction (Baudisch 2005). However, despite these other indicators being perhaps equally valid and predicting alternative patterns of senescence, the underpinning logic that a declining force of selection is associated with the evolution of senescence holds.

There have been a suite of other exercises that break assumptions of Hamilton's model but retain the logic of a declining force of selection as central to the evolution of senescence. One particular exercise that I have discussed throughout this thesis has concerned breaking the assumption that a species demography can best be described by age. Instead, when modelling size-related mortality and reproduction (Vaupel *et al*. 2004), or stage-based mortality and reproduction (Caswell & Salguero-Gómez 2013), the force of selection might not decline with age. An important extension to the modelling work I conducted in **Chapter 4** could be to consider stage-based demography within social groups, *e.g.* subordinate vs dominant phenotypes. From this extension, predictions for the plasticity of ageing in cooperative breeding systems could be generated (see Future Research).

One outstanding assumption of Hamilton's model that, to my knowledge, is yet to be broken and formally tested, is that mutations affect either mortality or reproduction, but not both. This assumption led to contrasting predictions in **Chapter 5**, where, in one analysis, I predicted accelerated actuarial (mortality) senescence but weaker reproductive senescence. I have shown in **Chapter 3**, supporting work elsewhere (Jones *et al*. 2008, Fay *et al*. 2022), that although there can sometimes be asynchrony between age-specific distributions of mortality and reproduction within individuals, there are significant signals of positive covariation between mortality and reproduction across taxa. Modelling this covariation, understanding how the effect of a mutation on mortality affects the force of selection on reproduction, and *vice versa*, is an important future step for force of selection models.

7.1.2 Senescence in the wild

A solid set of theory is required to generate predictions for the evolution of ageing in different populations. Ideally, this theory should be developed through the various expansions on the force of selection described above. However, the need to develop theory beyond Hamilton's work was only revealed from estimates of ageing and rates of senescence in the wild. This need was most notably shown by the identification of

species in which mortality and reproduction remains constant, or mortality declines with age and the rate of reproduction increases with age (Finch 1990; Jones *et al*. 2014; Shefferson *et al*. 2017). These findings have been revealed through long-term monitoring of populations from which demographic data, in particular estimates of age-specific survival and reproduction, can be recorded. Indeed, the majority of this thesis would not have been possible without longitudinal monitoring of wild populations. For studies to investigate senescence, repeated sampling of the same individuals is essential (Nussey *et al*. 2008; Sheldon & Clutton-Brock 2010; Nussey *et al*. 2013; Gaillard & Lemaître 2020). Only with the repeated sampling of individuals throughout their lifespan can we separate changes in the mortality risk and the rate of reproduction within individuals from population level patterns, *e.g.* selective disappearance (van de Pol & Verhulst 2006), a form of analysis that was conducted in **Chapter 6** (Roper *et al*. 2022). Yet, this form of analysis is not without its drawbacks, which I detail and then present possible solutions.

With high-resolution demographic data also comes bias. Intuitively, data are usually only available for species that are easier to monitor, but there is also biome and taxonomic bias which can lead to data gaps and bias in our understanding of how ageing has evolved (Jones *et al*. 2014; Conde *et al.* 2019). The difficulties of available data were an issue that became clear in **Chapter 5**, in which I was limited to a small sample of studies. Often, it may be exceptionally difficult or impossible to study a species longitudinally in a wild setting, for example if the species is fossorial or aquatic based. For species in which wild study is exceptionally difficult, mescocosms reflecting natural populations, for example those employed in in the Trinidadian guppy system (*Poecilia reticulata*; Reznick *et al*. 2004), could be used. Semi-artificial environments in which more of a species wild ecology can be maintained may offer closer estimates of wild rates of mortality and reproduction than lab-based studies can offer. Additionally, accurate age-specific estimates of mortality and reproduction across the whole lifespan of the species may be impossible if the species outlives human research careers, or due to limitations of funding (Salguero-Gómez *et.al* 2021). The ability for population monitoring to be passed down through generations (e.g. red deer (*Cervus elaphus*) on the Isle of Rum; Clutton-Brock *et al*. 1982), is one way to overcome this limitation, but is clearly not a widespread solution. Techniques such as probabilistic dendrochronology (Ricker *et al.* 2020), and the Forest Global Earth Observatory at the Smithsonian Conservation Biology Institute (Gonzalez-

Akre *et al.* 2016), have shown innovative solutions of obtaining demographic estimates of mortality and reproduction in long lived plants, overcoming the difficulties detailed above.

Even within those species for which detailed demographic data are available, there remain issues that invite caution in our interpretation of the ageing rates from wild populations. For example, in many systems, we cannot say with complete certainty that an individual not re-sighted in a given year has died, rather than moving out of the study site. Some individuals may also have unknown ages of birth or death, due to left or right truncation (Colchero *et al*. 2012), which can reduce sample size. Applications to cope with these challenges (Colchero *et al*. 2012; Koons *et al*. 2014) provide a probabilistic approach to estimate survival and detection probabilities, accounting for low recaptures probability and unknown ages. Although these applications are innovative tools that have drastically improved our ability to generate more reliable ageing estimates, they are still probabilistic estimates that may be influenced by unavoidable selective sampling. A potential, yet probably not cost-effective, solution would be to track mobile organisms that can move out of the study site with non-invasive GPS signalling (Gunner *et al.* 2021). A period of non-movement over a set duration of time could give a more reliable indication of actual death, rather than assuming all individuals that leave the study site have died.

Ultimately, understanding the evolution of senescence in a wild population requires a robust interface between theory, data, and statistical methods. As senescence researchers, we should apply the appropriate model of the force of selection to predict ageing patterns in a given system. For example, considering whether the species is social and its rates of survival and reproduction are best predicted by age, stage, or their combination. We can then be guided by the appropriate theoretical model to collect relevant demographic data from repeated samples of individuals across their life course, ideally in the settings described above. It is unlikely that there is one encompassing force of selection model for all species, and that the same empirical system can be applied across a vast number of species. Accounting for the distinguishing features between both theory and data collection across systems is essential to better understanding senescence in the wild.

7.2 Senescence in social species

In **Chapter 4**, I modelled inclusive fitness forces of selection to show that the evolutionary fate of alleles in social species depends in part due to age-dependent social interactions. In **Chapter 5**, I revealed signals that group-level characteristics in species that exhibit alloparental care do indeed modulate how long species live and their distribution of reproduction across the life course (but see **Chapter 5 Discussion** for potential caveats). In **Chapter 6**, I investigated ageing in a wild population of the long-tailed tit (*Aegithalos caudatus*), revealing limited effects of load-lightening in this system, and that individual heterogeneity was most important for population level ageing patterns. These chapters focused on the interaction between sociality and ageing. I begin this part of the discussion by detailing possible extensions to the model developed in **Chapter 4**. Then, I consider how the model could lead to new insights in the same vein as **Chapter 5**, where I identified an association between group relatedness, shorter expected adult lifespan, and weaker reproductive senescence. I then consider potential empirical tests, based off the previous chapters, that could be investigated in systems like the long-tailed tit in **Chapter 6**. I finish this section by considering a new avenue of research, 'social ageing', which reverses the line of questioning that has been conducted in this thesis. Rather than asking how sociality might alter patterns of ageing, instead, considering how patterns of ageing might alter sociality.

7.2.1 Future research

The model employed in **Chapter 4** has several limitations that fall into roughly two kinds of assumptions with respect to the species: (1) demography and (2) sociality. The first category considers common demographic modelling assumptions such as population stability, and invariance of mortality/survival and reproduction over time (Caswell 2019). These assumptions have been broken, and have seen great progress in various avenues of demographic research (see *e.g.* Tuljapurkar *et al.* 2009), but I did not consider them in this thesis due to both a need for mathematical simplicity and time constraints. These additions are obvious extensions to the model in **Chapter 4**. Extending the model to a two-sex systems falls somewhere in between these two categories. Haploid, asexual genetics are commonly assumed in the infinite island modelling framework I utilised in **Chapter 4** for kin selection models (see *e.g.* Taylor

1992; Pen 2000; Ronce *et al*. 2000; Ronce & Promislow 2010). As I model dispersal between patches, there is still variation in relatedness that is required to inform inclusive fitness forces of selection despite clonal reproduction. However, there is a biological case for adding two sexes to the model. In many cooperative breeding systems there is often sex-biased dispersal, *e.g.* female biased in the long-tailed tit (*Aegithalos caudatus*; Hatchwell 2016). This sex-biased dispersal can lead to variation between the sexes in age-specific relatedness and age-specific prospects of direct and indirect fitness. Given empirical evidence of sex-specific differences in lifespan and rates of senescence (Lemaître *et al*. 2020), a two-sex model may provide higher resolution predictions for senescence in social species. Sex-specific inclusive fitness forces of selection could then predict variation in ageing metrics based on the dispersing sex, differential relatedness between the sexes towards recipients, and differential access to reproduction between the sexes. The second category consists of modelling assumptions of the social system, that could improve the biological applicability of the model. Ultimately, there is a trade-off when modelling between simplicity for generality and biological relevance. However, I believe the two features I describe below, delayed dispersal and stage structure, could add biological relevance to the model without detracting from its general applicability, and provide further testable comparative predictions across social species.

It is common in infinite island modelling that juveniles can disperse at the beginning of life or stay in the patch that they were born into for life (see *e.g.* Ronce & Promislow 2010). This assumption was used in **Chapter 4**, and is modelled through a rate of dispersal parameter, d, that varies between 0 (no dispersal) and 1 (all individuals disperse). Yet, an important feature of many cooperative breeding systems is delayed dispersal, in which individuals may remain in their natal patch for some time before dispersing to breed in a different location (Koenig & Dickinson 2016). Dispersal is an important component of driving local relatedness between current members of a group, which is captured in some respects by the current model format. In this respect, dispersal, as is currently modelled, does well at generating variation in relatedness to inform predictions for inclusive fitness forces of selection. However, the inclusive fitness force of selection at each age class may differ if dispersal probability, and ultimately breeding success, varies with age (*e.g.* the dwarf mongoose (*Helogale parvula*); Creel & Waser 1992). When individuals have the prospect of dispersing to a new patch, this probability will alter the distribution of age classes in a group, and the associated age-specific costs and benefits of dispersing relative

to staying on the same patch will alter age-specific inclusive reproductive value.

A further extension is the addition of stage structure to the model. Perhaps rather than age, the stage, or a combination of both, will be a better predictor of mortality risk and rate of reproduction in social species. For example, meerkats (*Suricata suricatta* show distinct mortality trajectories between dominant and subordinate individuals (Thorley *et al.* 2020). In the current format of the model, stages are modelled in some respect as juveniles, reproductive and post-reproductive. An additional stage-structuring of the reproductive age classes (*i.e.* individuals with the physiological capacity to reproduce) could be incorporated by designating individuals with a probability of being a subordinate or a dominant based on age. Subsequently, assigning (st)age specific estimates of mortality risk, rate of reproduction, and transfers to other (st)age classes could facilitate stage-by-age computation of inclusive fitness forces of selection (see Caswell *et al.* 2018 for proposed methodology but with inclusive fitness, rather than *r*, as Darwinian fitness). This modelling exercise could allow for predictions to be made as to the extent of plasticity of ageing rates within social systems that exhibit subordinate and dominants phenotypes (Heinze & Giehr 2021).

In this thesis, I tested predictions from the model in **Chapter 4** and elsewhere (Ronce & Promislow 2010; Kreider *et al.* 2022) comparatively on a set of mammals, birds, and a fish that exhibit alloparental care. Limitations of this analysis are discussed in detail in **Chapter 5**, so I will not discuss them here. Instead, lastly, I want to consider how the model of **Chapter 4** could be applied to an empirical system. The fundamental concept of the model is the idea of 'transfers' between age classes. The transfers are contributions of one individual of a given age class to the survival and/or reproduction of another individual of a given age class (which could be the same age class). I conceptualised these transfers in **Chapter 5** as the effects of helpers on the reproductive success of breeders, that were documented across studies. However, from my knowledge of the exhaustive literature review conducted in **Chapter 5**, only a few cooperative species have documented age-specific estimates of helping effort and relatedness of the helper to recipients. For a full empirical test of the model, one would need to record age-specific effects of individuals on the survival and reproduction of all other individuals, and have genetic estimates of relatedness to model how relatedness changes with age.

Overall, the findings I have presented in this thesis indicate an important role of social

interactions in the variation of lifespan and senescence across social species. I hope that this work will provide stimulation for future theoretical, comparative, and experimental analyses. To conclude the thesis, I briefly switch the direction of thought from how sociality might modulate ageing patterns, to how ageing patterns might alter individual sociality.

7.2.2 Social ageing

Social ageing is defined as the change in individual sociality throughout the adult life stage (Siracusa *et al.* 2022). For example, humans tend to limit the number of social interactions with increasing age to those relationships they deem more 'meaningful' (Charles & Carstensen 2010). However, those individuals who are more socially integrated display improved health (Synder-Mackler *et al.* 2020). Important questions remain around the causality of social ageing. Is increased social connectedness a driver of improved health, or can individuals remain more social because of individual variation in ageing rates? Albery *et al.* (2022) tested for changes in social connectedness in red deer (*Cervus elaphus*), whilst accounting for potential selective disappearance: those individuals that are more social may engage in more competitive interactions and as a result are more likely to die, resulting in a demographic filtering leaving 'less social' individuals to survive to older age classes. However, the authors did find, after accounting for this potential effect, that more socially connected individuals were likely to live longer. The review by Siracusa *et al.* (2022), focusing on mammals, set out to explore verbal theoretical predictions that might drive changes in social behaviour with age. They suggested that social ageing could occur as a result of senescence, be adaptive to ameliorate senescence, or result from positive effects of age and demographic changes. This new avenue of research is an interesting flip of the directionality of the relationship between sociality and ageing explored in this thesis.

From an adaptive perspective, I suggest that social ageing could be integrated into thoughts around the evolution of age-specific social behaviour (Rodrigues 2018). For example, there is the prediction that if an individual decides between directing help towards two individuals of equal relatedness, it should choose the individual with the higher reproductive value to maximise their indirect fitness. The model I presented in **Chapter 4** instead highlights the importance of inclusive reproductive value, rather than conventional reproductive value. Individuals should base helping decisions based on

their own inclusive reproductive value, the recipient's inclusive reproductive value, and relatedness to the recipient. It would be valuable to attempt to quantify the level of variation in social ageing that can simply be explained by evolutionary theory surrounding age-dependent social behaviour. Understanding the level of variation explained by standard social evolution theory might provide a greater indicator for the requirement of novel theory for the apparent phenomena of 'social ageing'.

7.3 Concluding Remark

The work in this doctoral thesis was driven by two primary directions of investigation: developing a general understanding of the variation in ageing patterns across multicellular organisms, and exploring the interplay between ageing and sociality. The evolutionary theories of senescence are mature, yet still can be extended to encompass a broader range of organismal features, and our ability to test theory with empirical data is ever expanding. The available empirical data suggest that these mature theories require updated thinking, as exemplified in this thesis. In the medical field, ageing is now considered a disease (de Grey & Rae 2007), and ageing populations are an impending workforce and healthcare crisis that humanity faces (Storey *et al.* 2019). At an application level, this thesis posits that the variable rates of ageing across species can be understood by extensions to central evolutionary theory. My work points towards directions of avenues for further exploration, in which we may identify new model systems to understand proximate mechanisms of variable ageing, from which potential mechanisms of intervention may be revealed. The evolution of senescence is an important, never-ageing question, but has come a long way since the mid 20th century.

7.4 Discussion Bibliography

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8 | Appendix

The following Appendix Items are included:

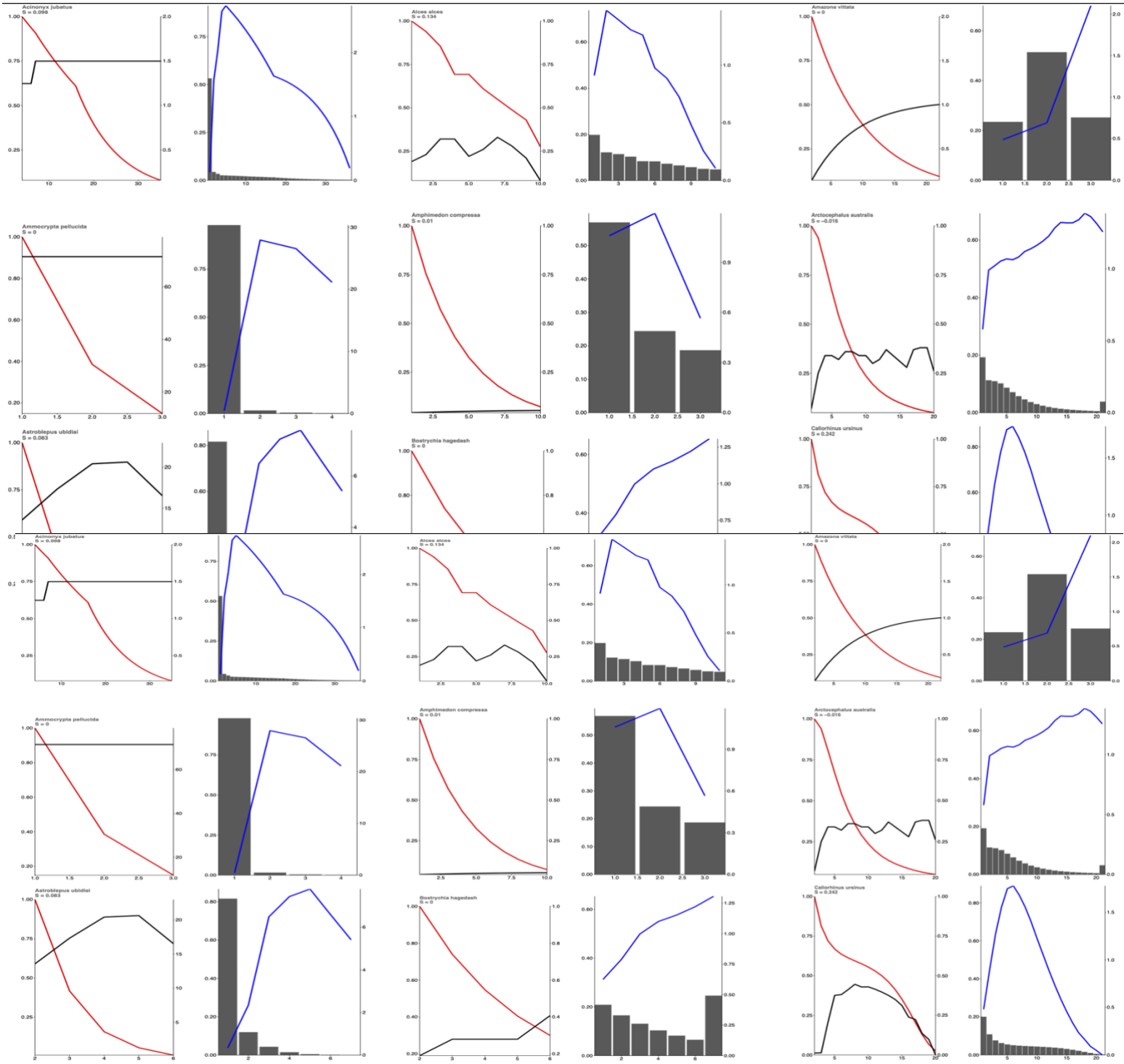
- **Appendix A** includes the extended supplementary information for **Chapter 2**, that is included here due to space limitations.
- **Appendix B** includes a published reply that I co-authored with Roberto Salugero-Gómez addressing the differences between Hamilton's (1966) and Caswell's (1978; 2010) forces of selection for age and stage structured populations.
- **Appendix C** includes a published bibliography piece I was asked to co-author along with Roberto Salguero-Gómez, Connor Bernard, and Jacques Deere, which discusses the historical study of senescence, and thoughts on future study.

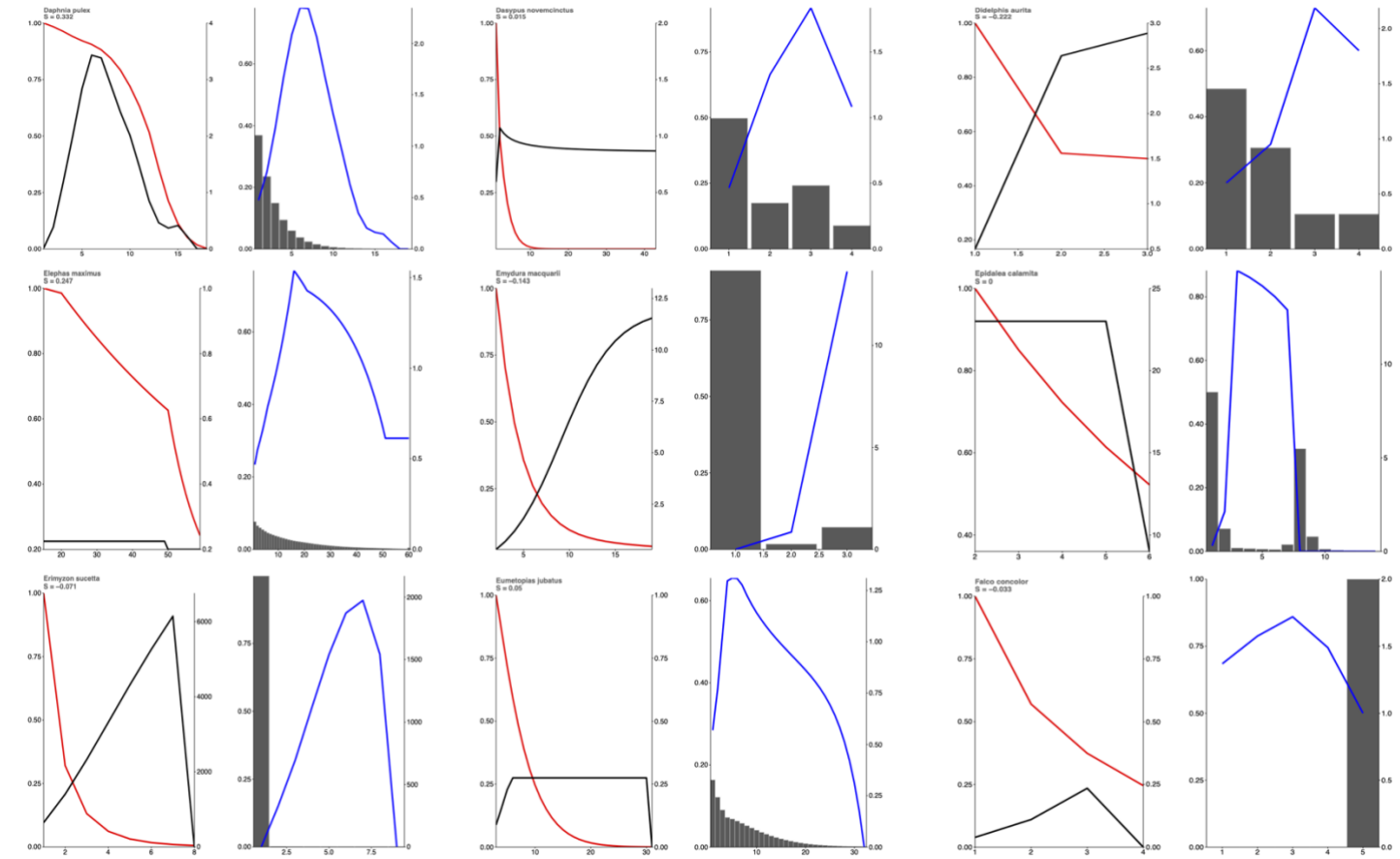
Appendix A

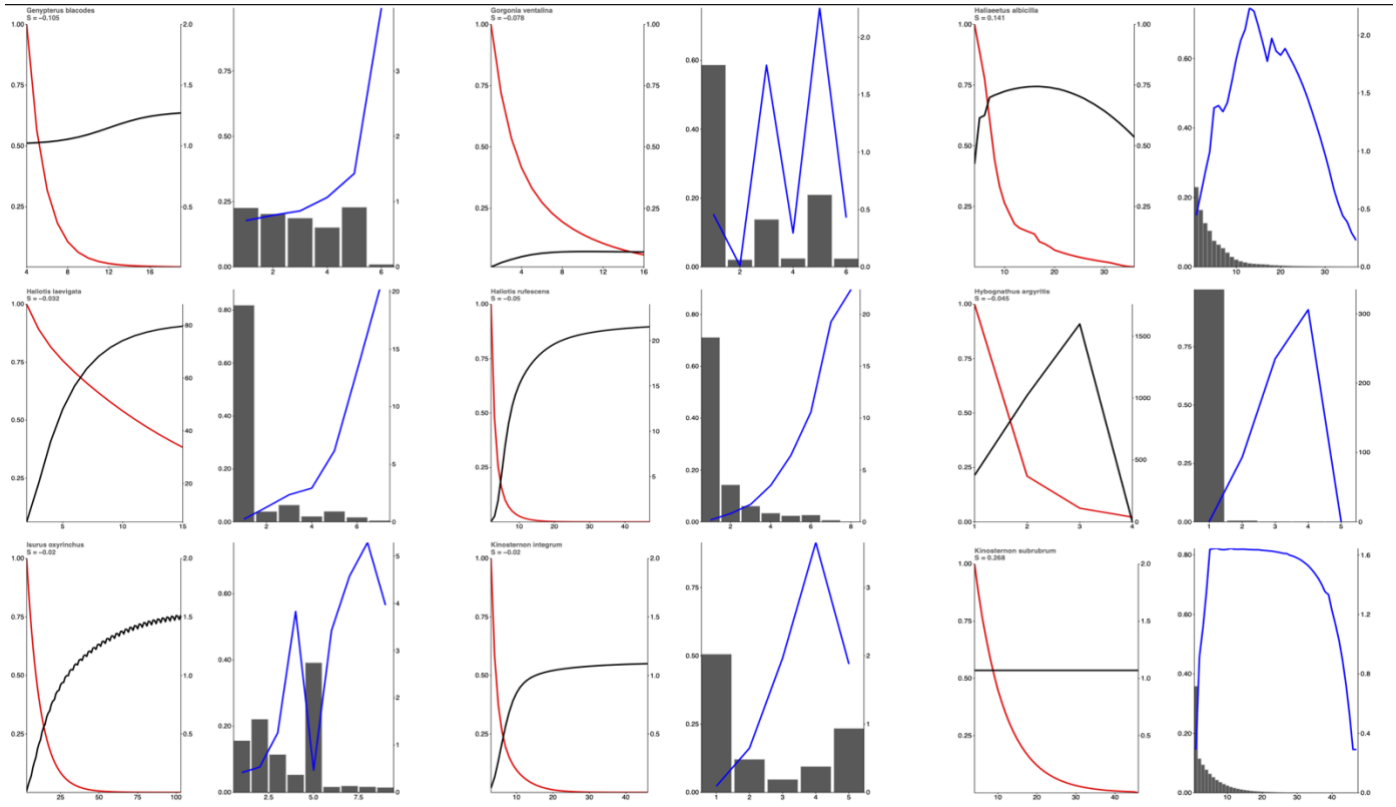
Appendix A

Fig. S3. For each species in our analysis, survivorship (red) and reproduction (black) age-trajectories are shown from the age at maturity on the left-hand side. Reproductive value (blue) and stable (st)age proportions (which sum to 1) for each life stage are also shown for each species on the right-hand side. A) Animals. B) Plants.

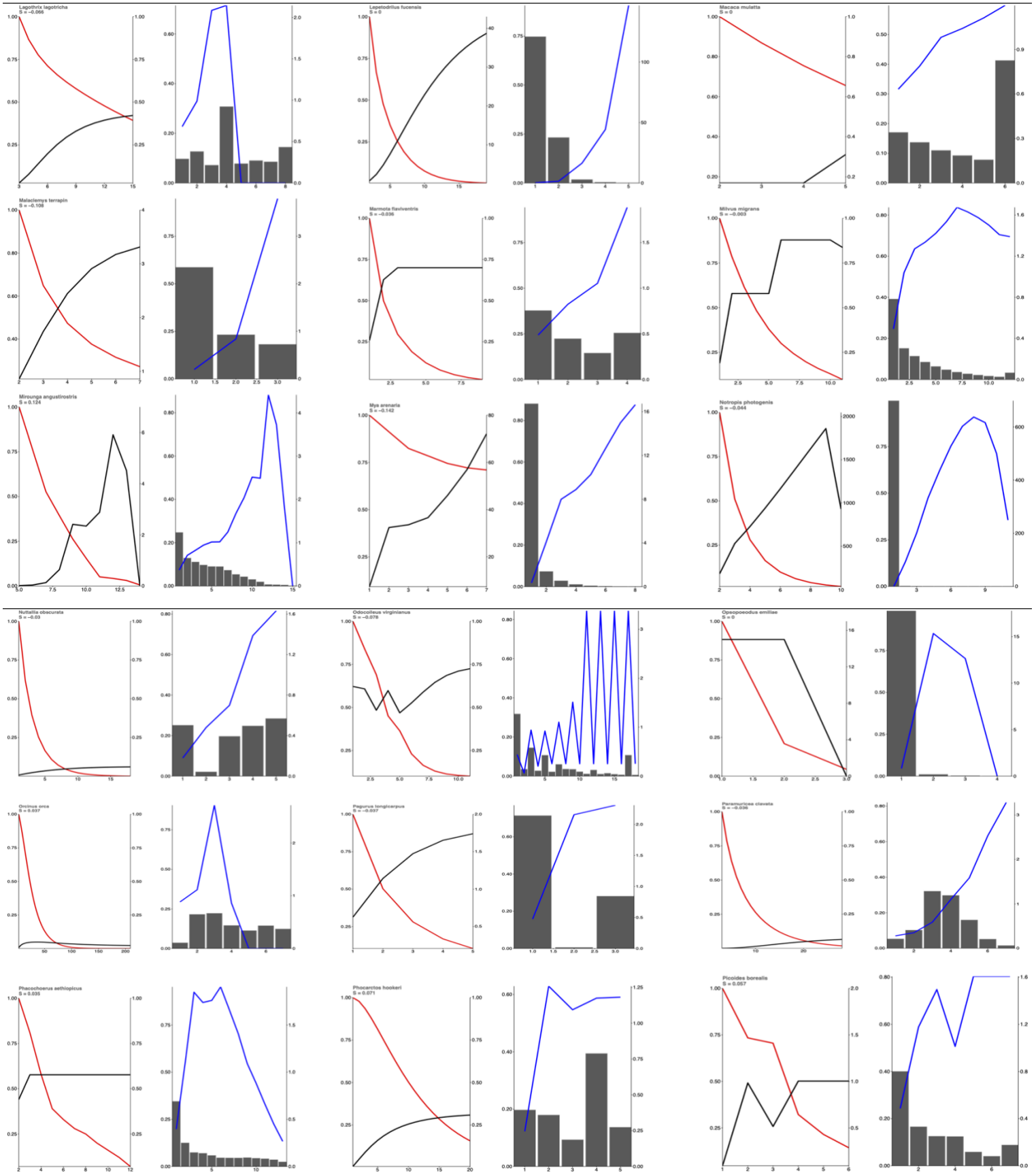
(*a*)

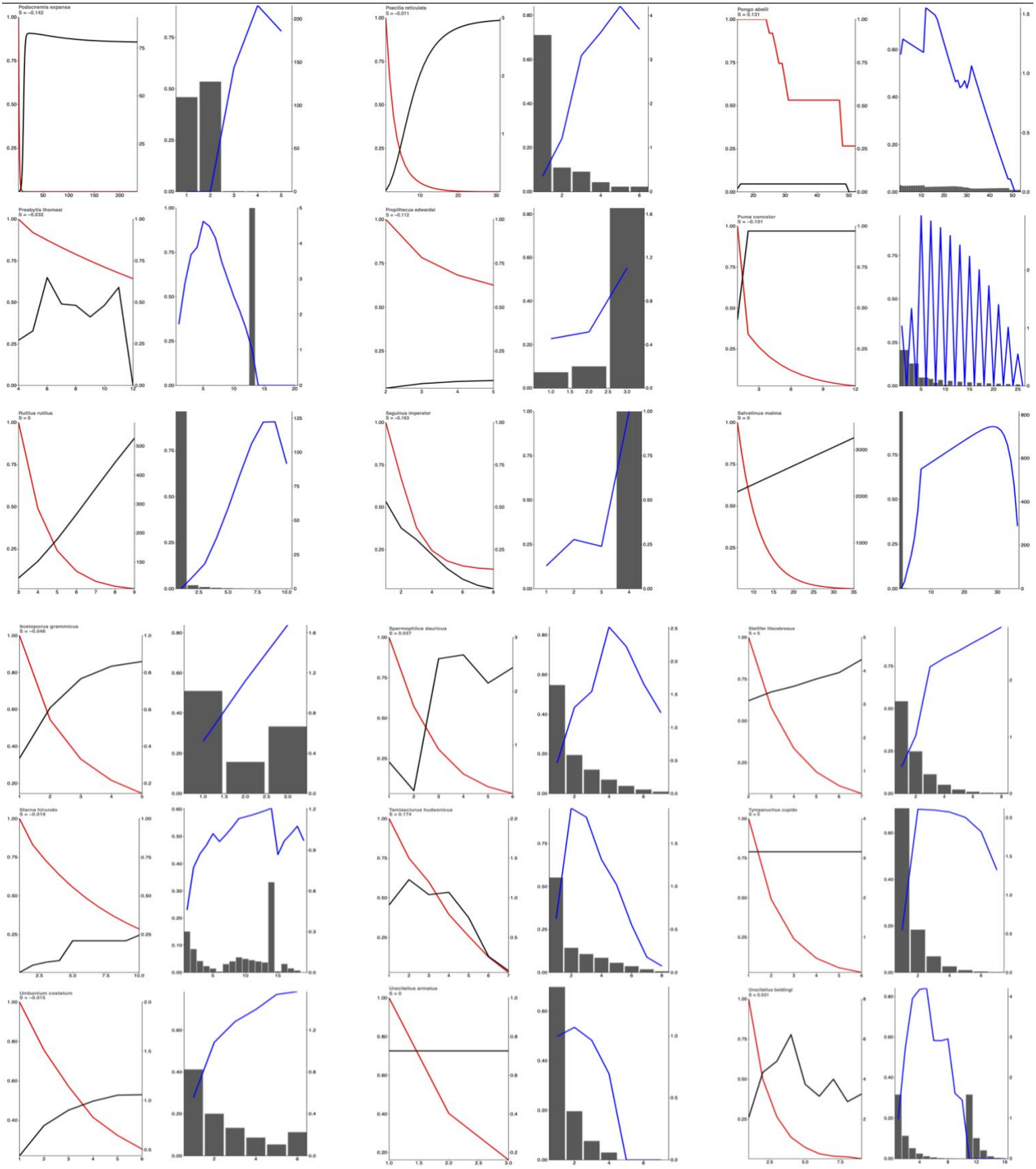


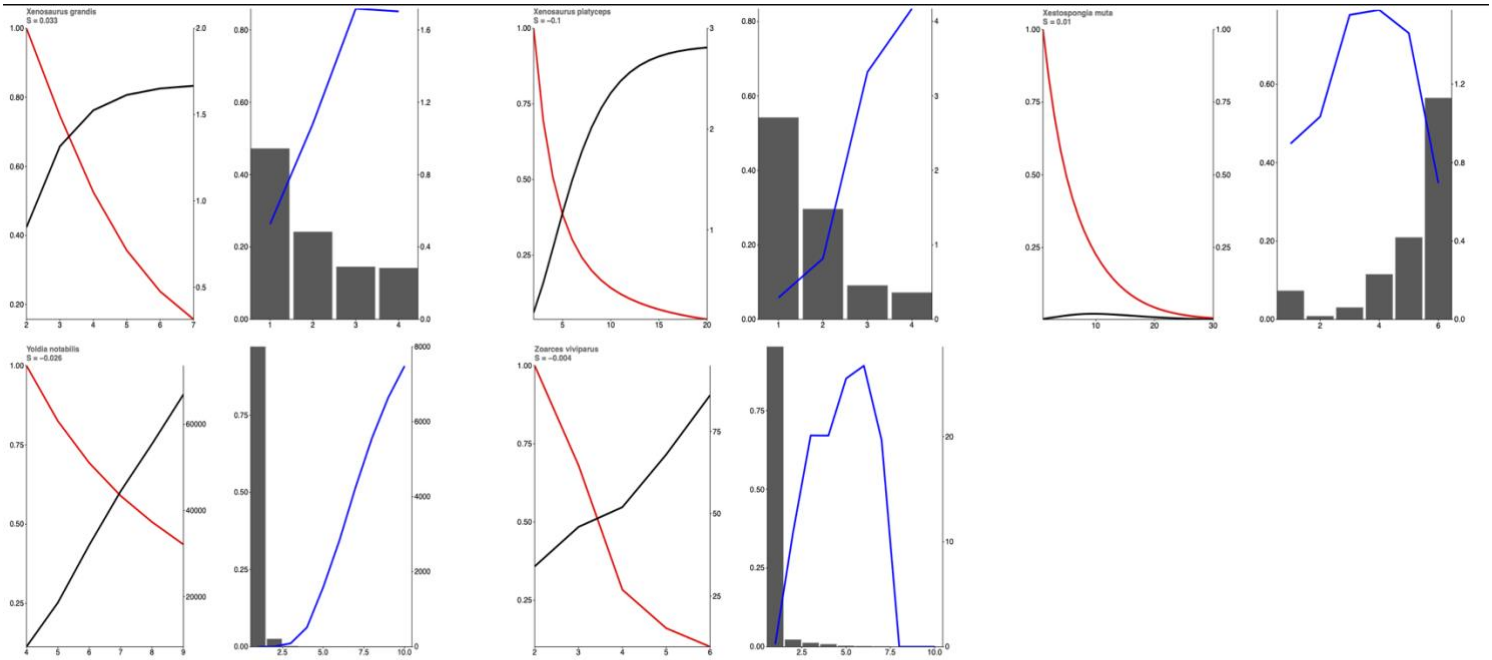

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Dasypus novemcinctus
S = 0.015
Didelphis aurita
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Elephas maximus
S = 0.247
Emydura macquarii
S = −0.143
Epidalea calamita
S = 0
Erinyzon sucetta
S = −0.071
Eumetopias jubatus
S = 0.05
Falco concolor
S = −0.033


Genyptterus blacodes
S = −0.105
Gorgonia ventalina
S = −0.078
Haliaeetus albicilla
S = 0.141
Heliotis laevigata
S = −0.032
Heliotis rufescens
S = −0.05
Hybognathus argyritis
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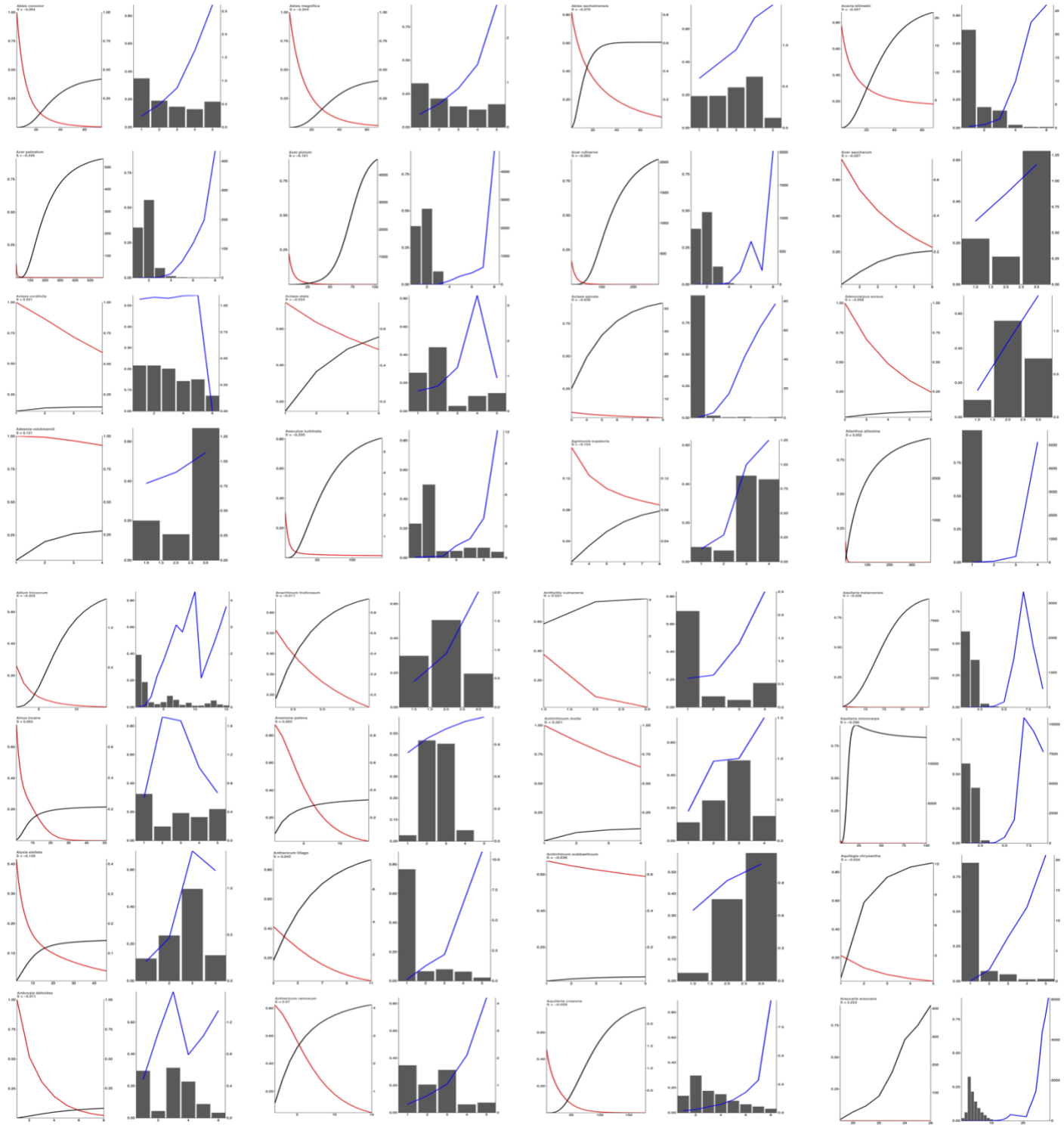
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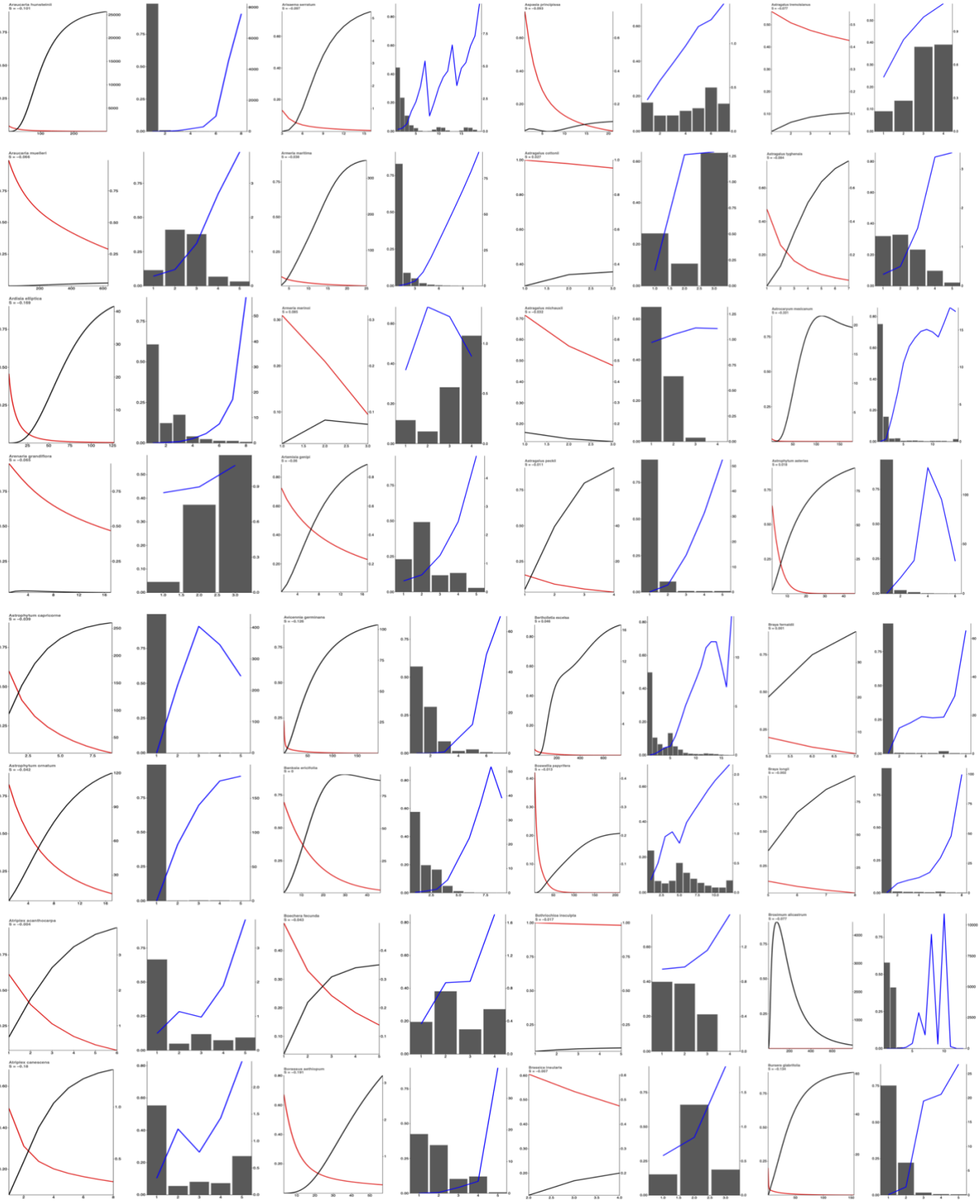

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S = 0
Macaca mulatta
S = 0
Malaclemys terrapin
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Marmota flaviventris
S = −0.036
Milvus migrans
S = −0.003
Mirounga angustirostris
S = 0.124
Ilya arenaria
S = −0.142
Notropis photogenis
S = −0.044
Nuttallia obscurata
S = −0.03
Odocoileus virginianus
S = −0.078
Opsopoeodus emiliae
S = 0
Orcinus orca
S = 0.037
Pagurus longicarpus
S = −0.037
Paramuricea clavata
S = −0.036
Phacochoerus aethiopicus
S = 0.035
Phocarctos hookeri
S = 0.071
Picoides borealis
S = 0.057

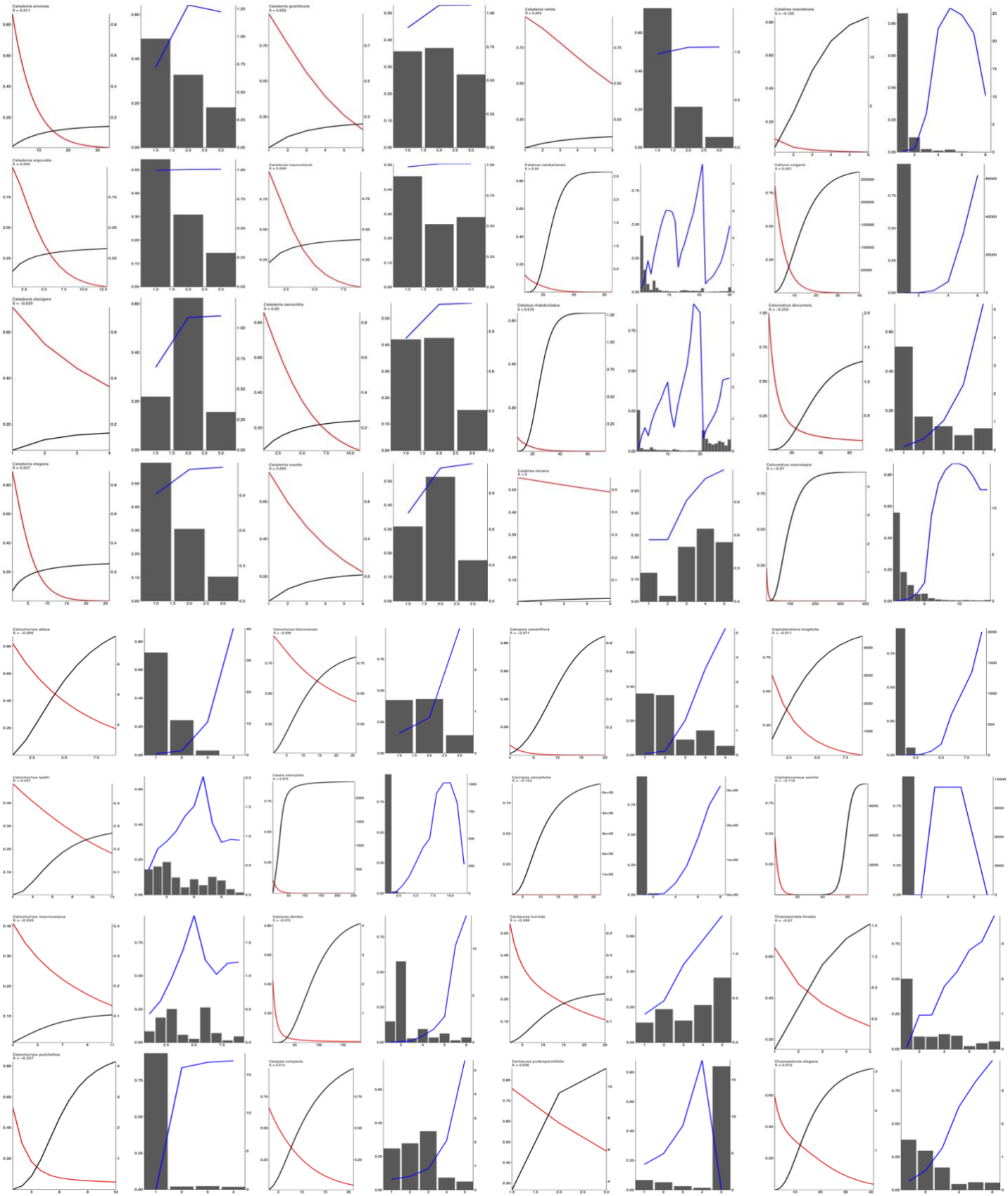

Podocnemis expansa
S = -0.142
Poecilia reticulata
S = -0.011
Pongo abelii
S = 0.131
Presbytis thomasi
S = -0.032
Propithecus edwardsi
S = -0.112
Puma concolor
S = -0.101
Rutilus rutilus
S = 0
Saguinus imperator
S = -0.103
Salvelinus malma
S = 0
Sceloporus grammicus
S = -0.046
Spermophilus dauricus
S = 0.037
Stellifer lllecebrosus
S = 0
Sterna hirundo
S = -0.019
Tamiasciurus hudsonicus
S = 0.174
Tympanuchus cupido
S = 0
Umbonium costatum
S = -0.015
Urocitellus armatus
S = 0
Urocitellus beldingi
S = 0.031

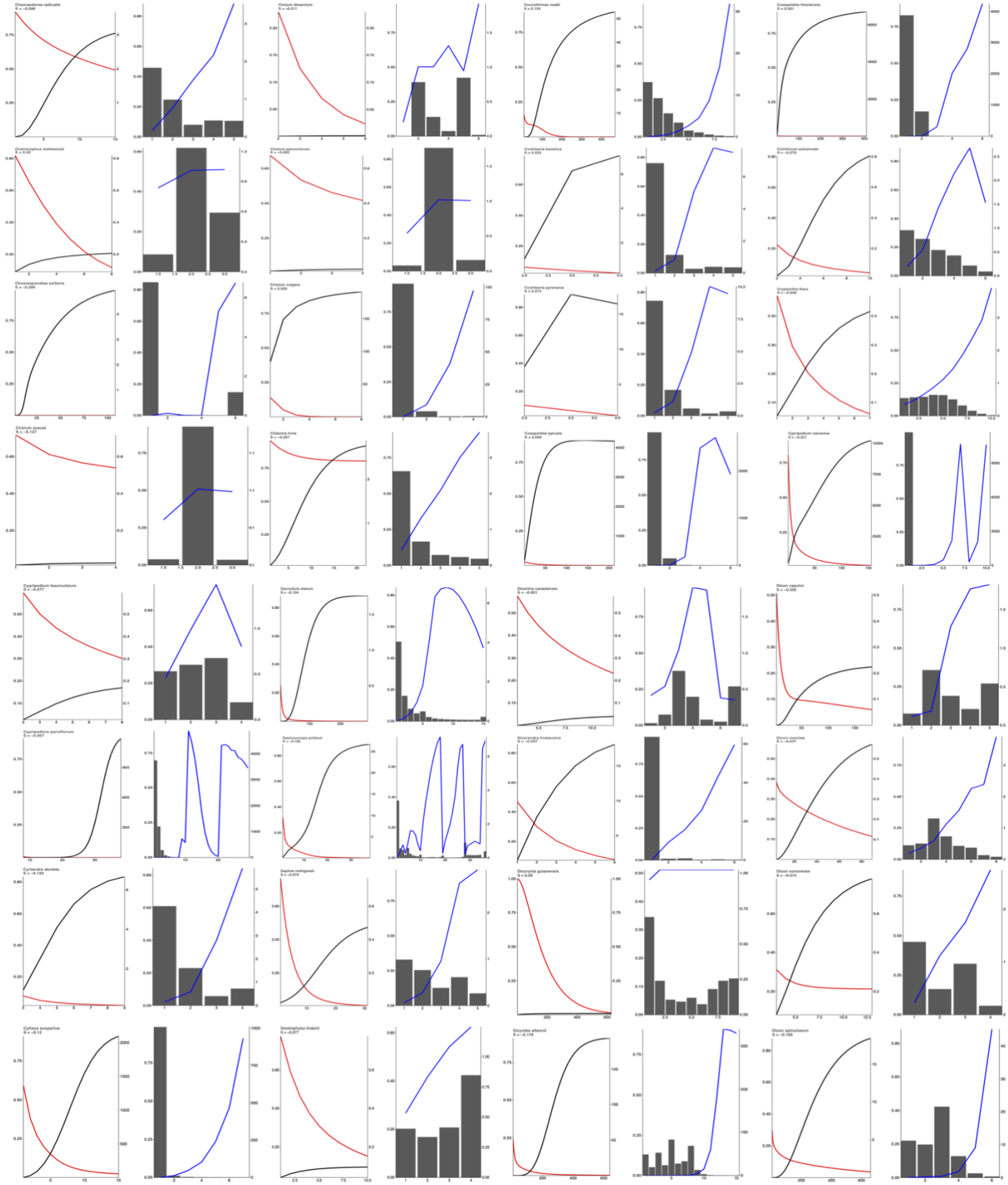

Xenosaurus grandis
S = 0.033
Xenosaurus platyceps
S = -0.1
Xestospongia muta
S = 0.01
Yoldia notabilis
S = -0.026
Zoarces viviparus
S = -0.004

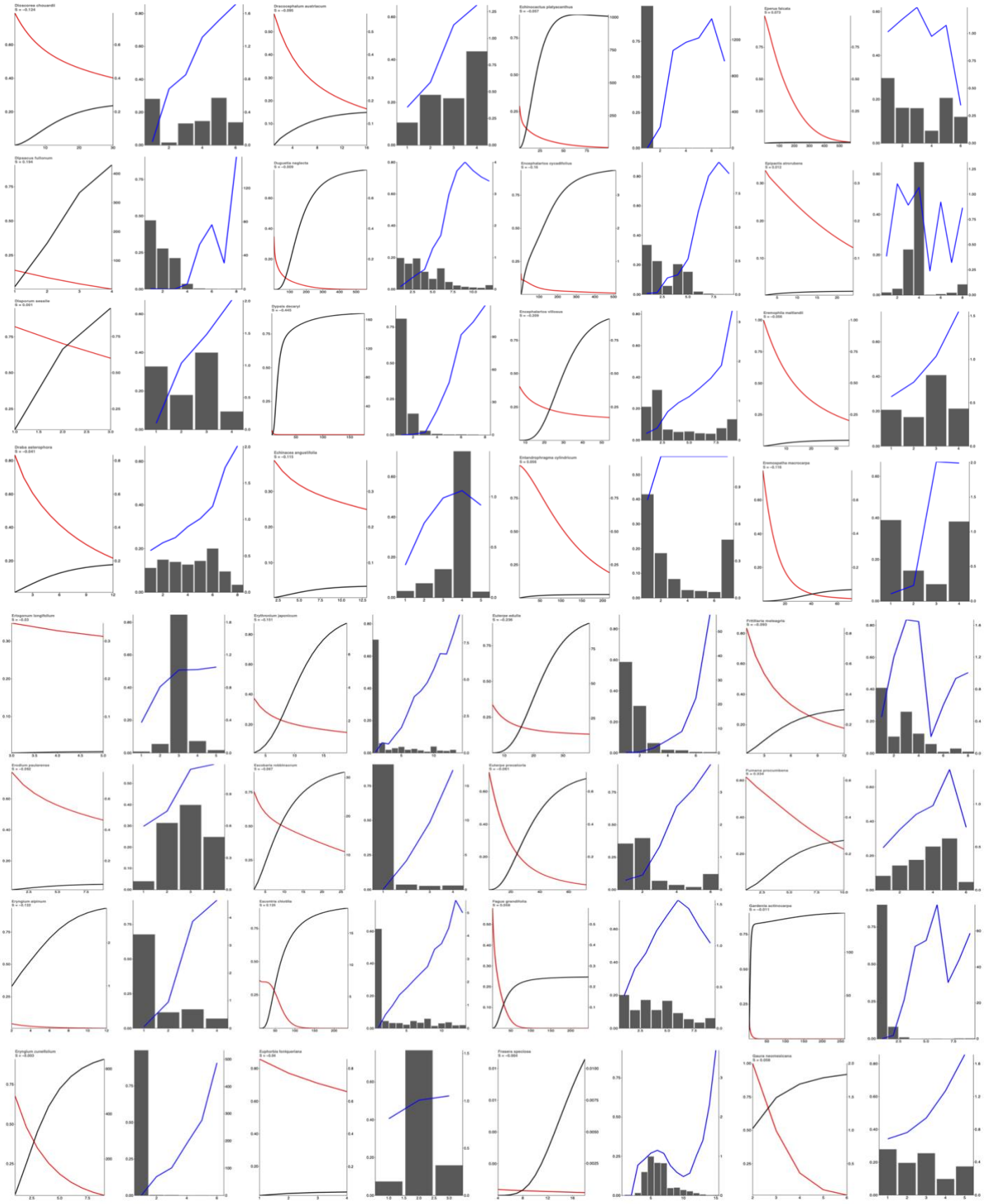
(b)

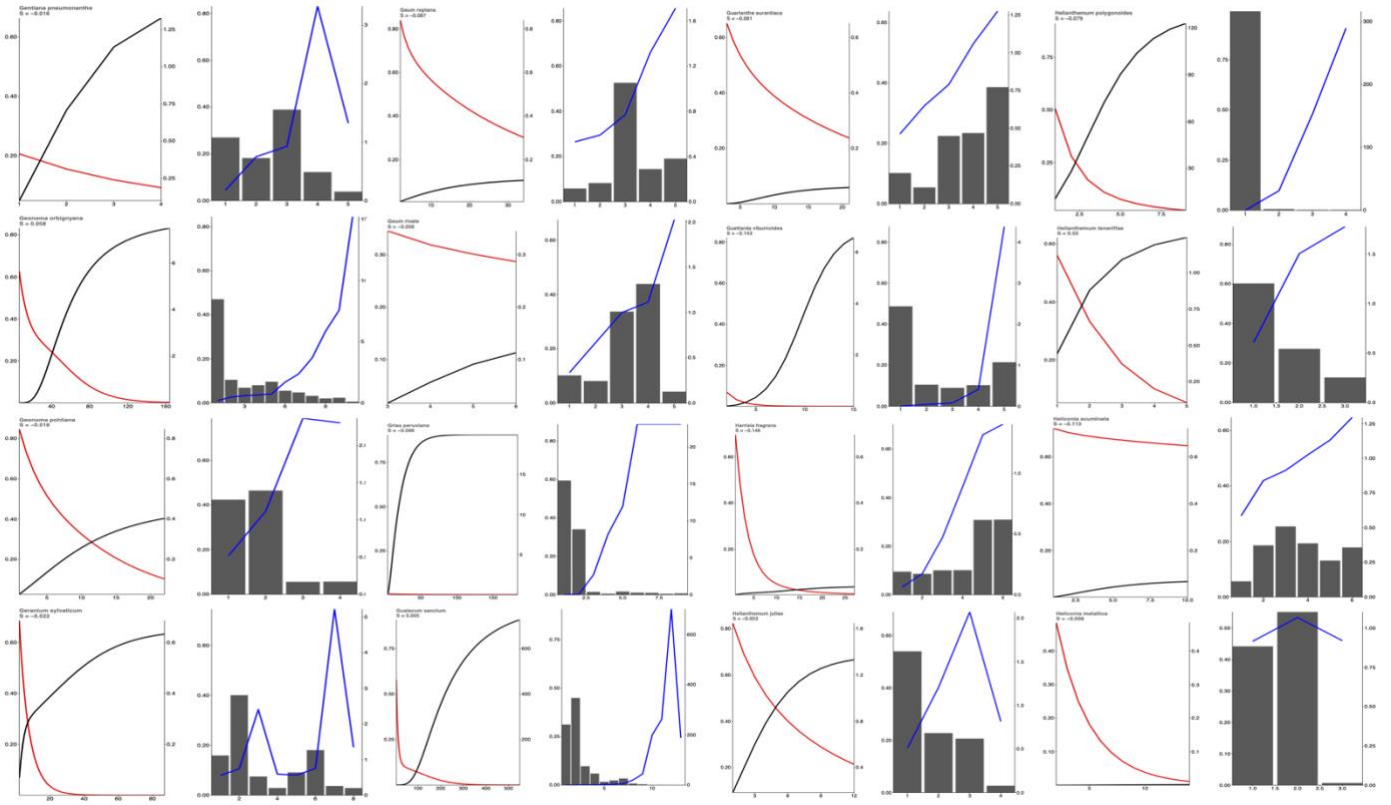


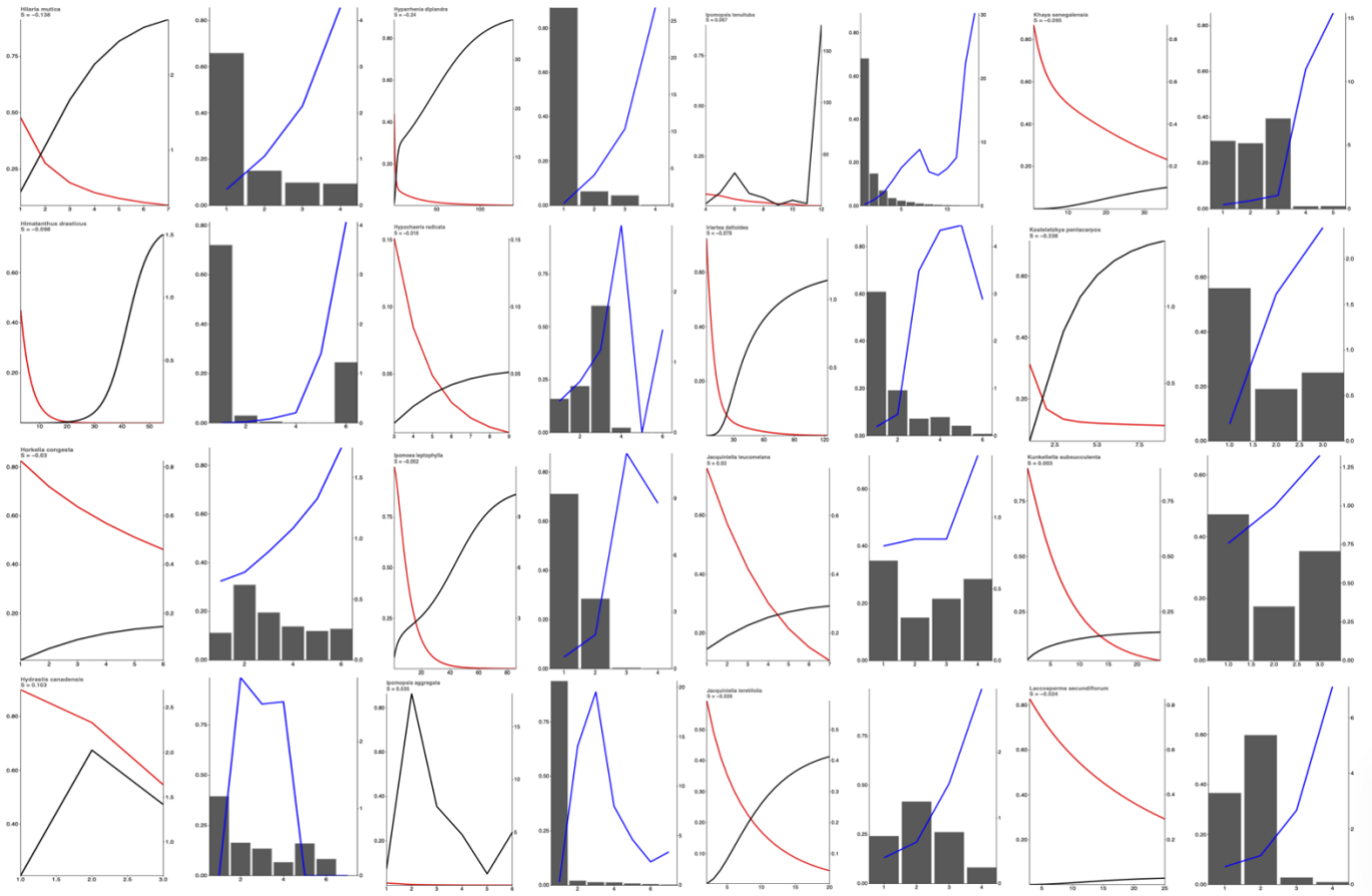


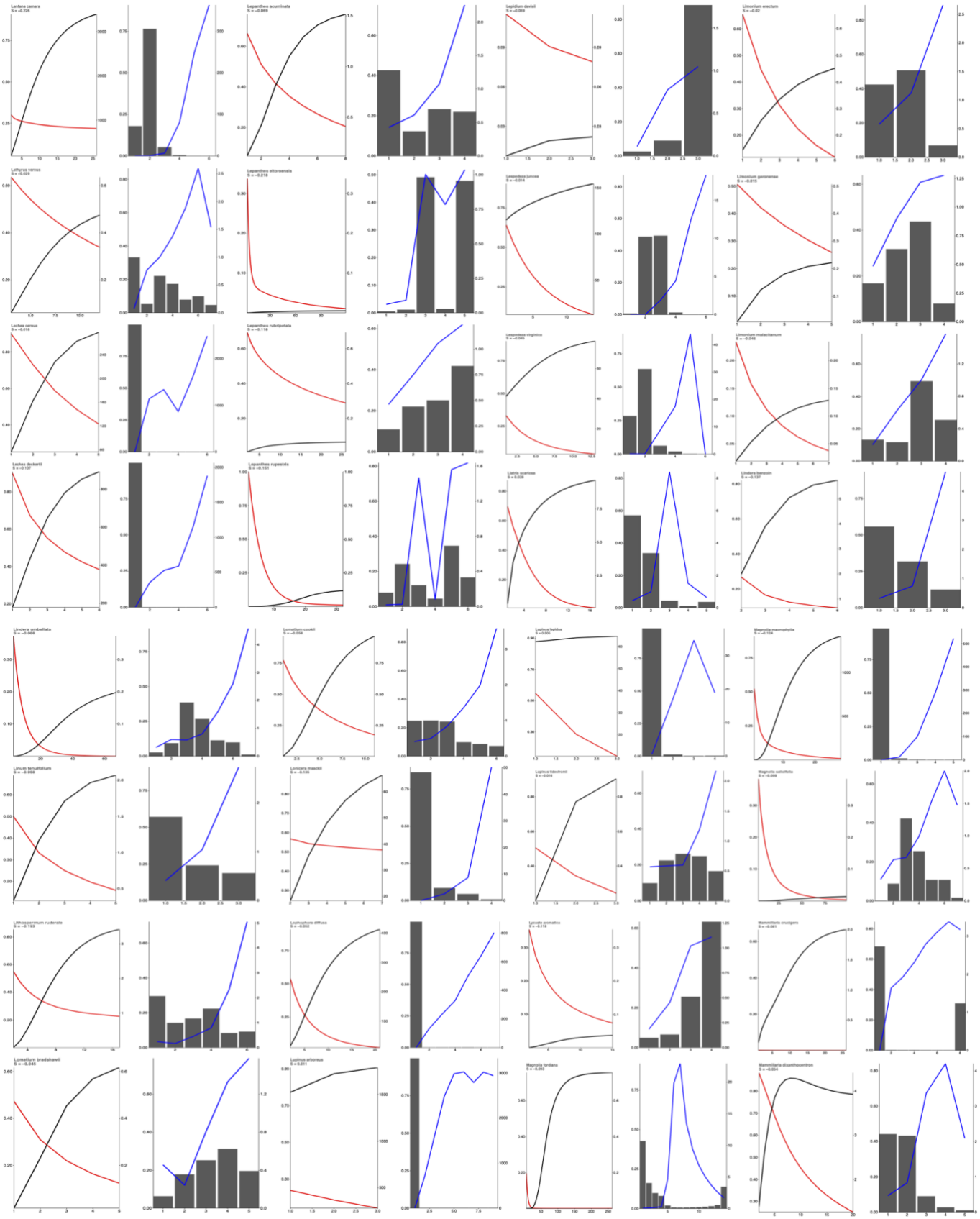


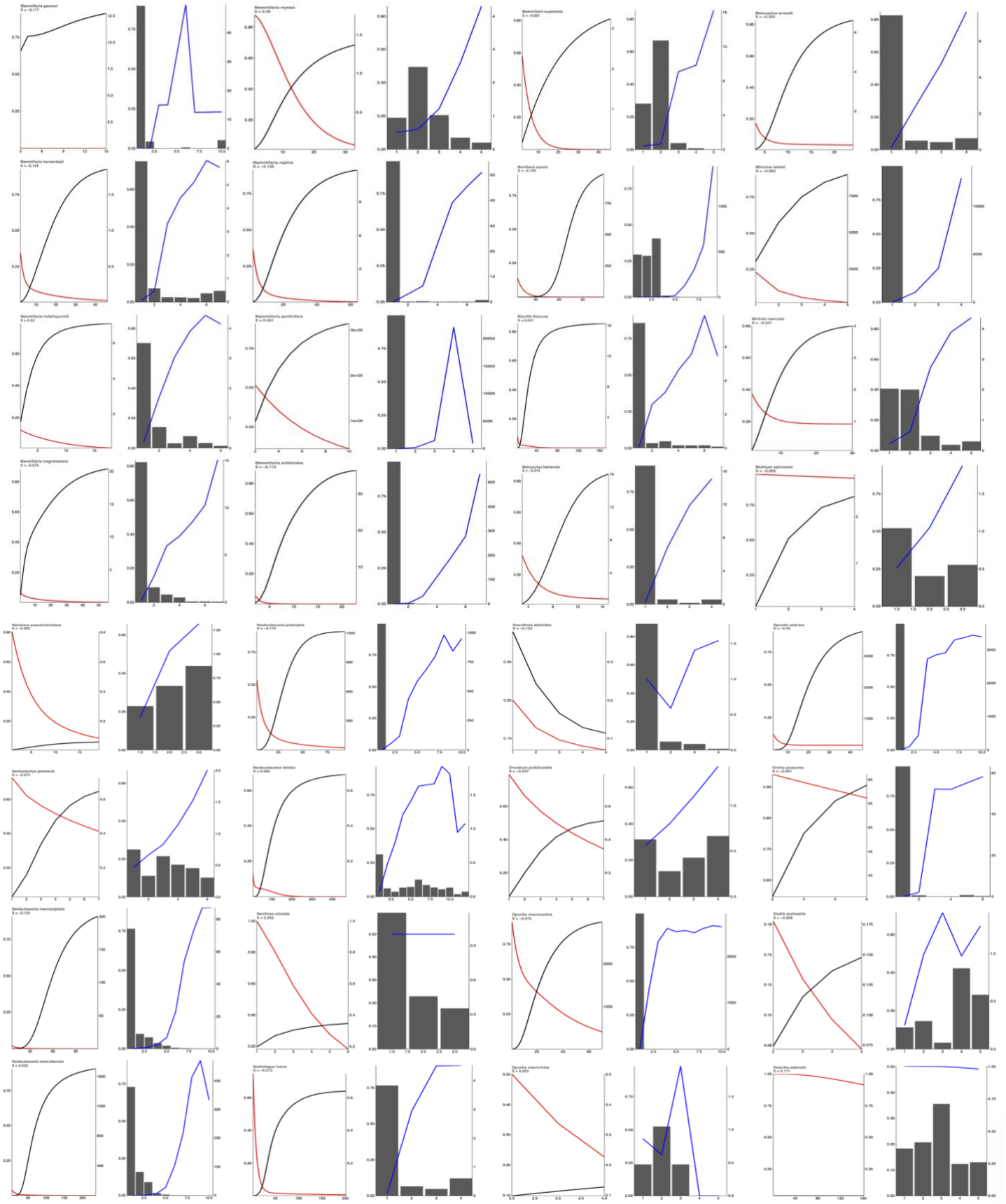


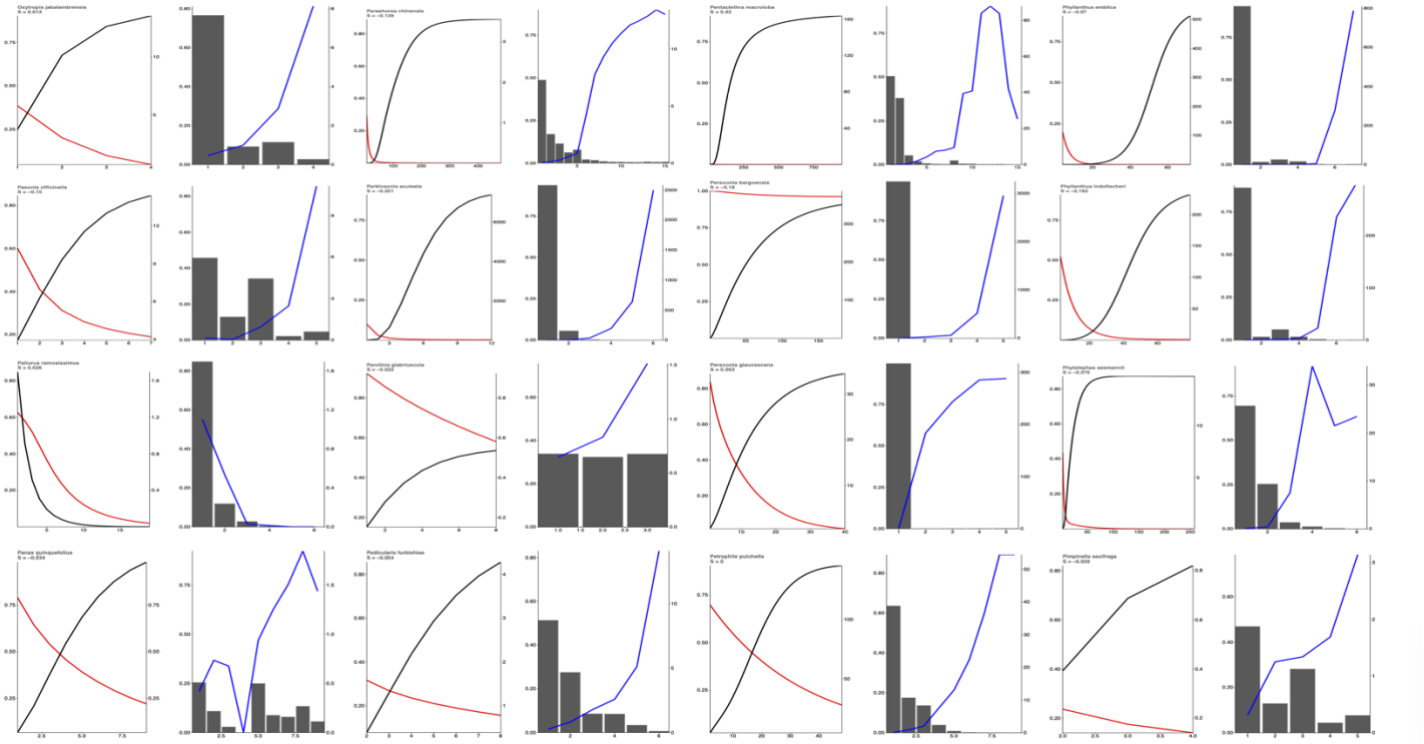


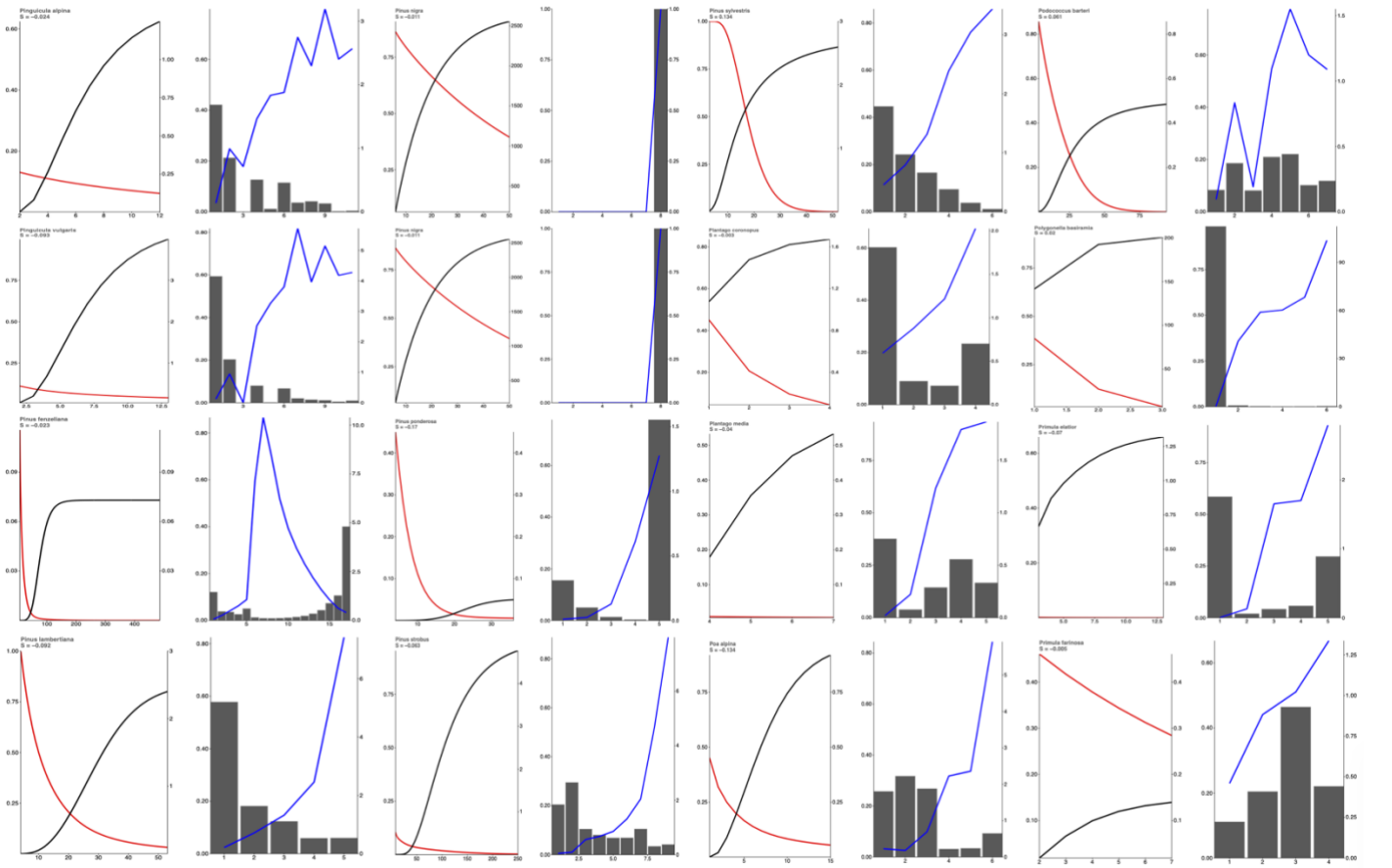


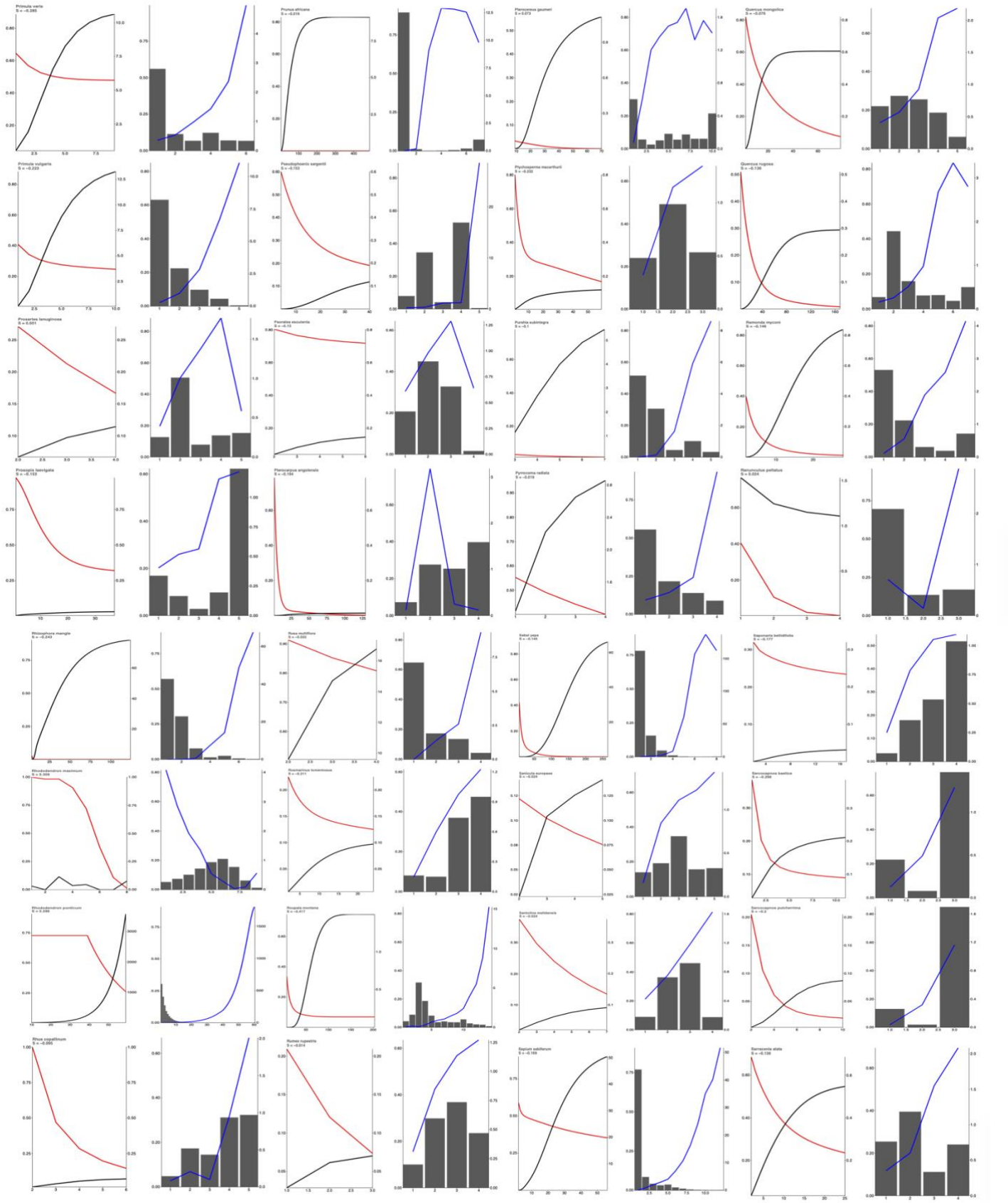


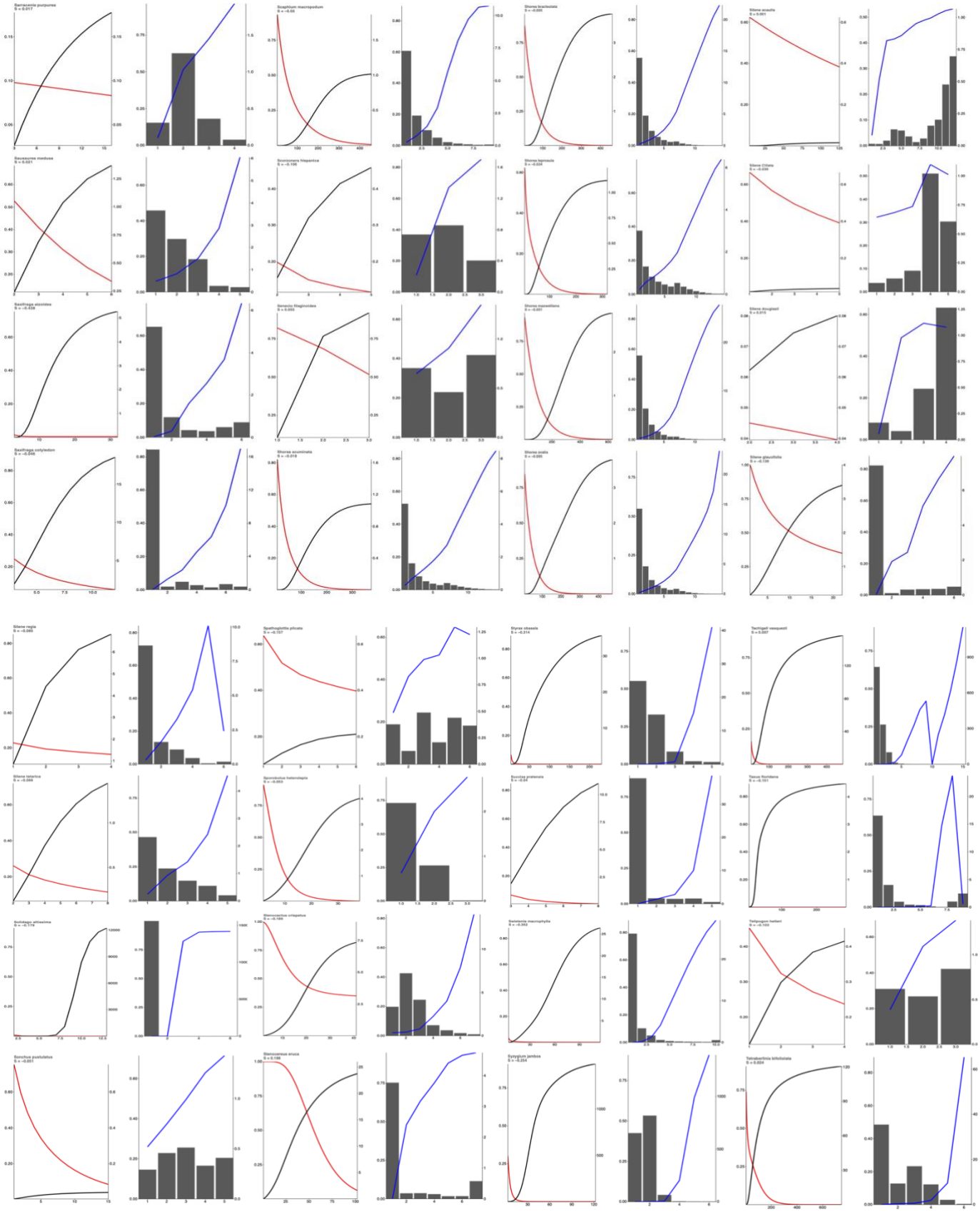


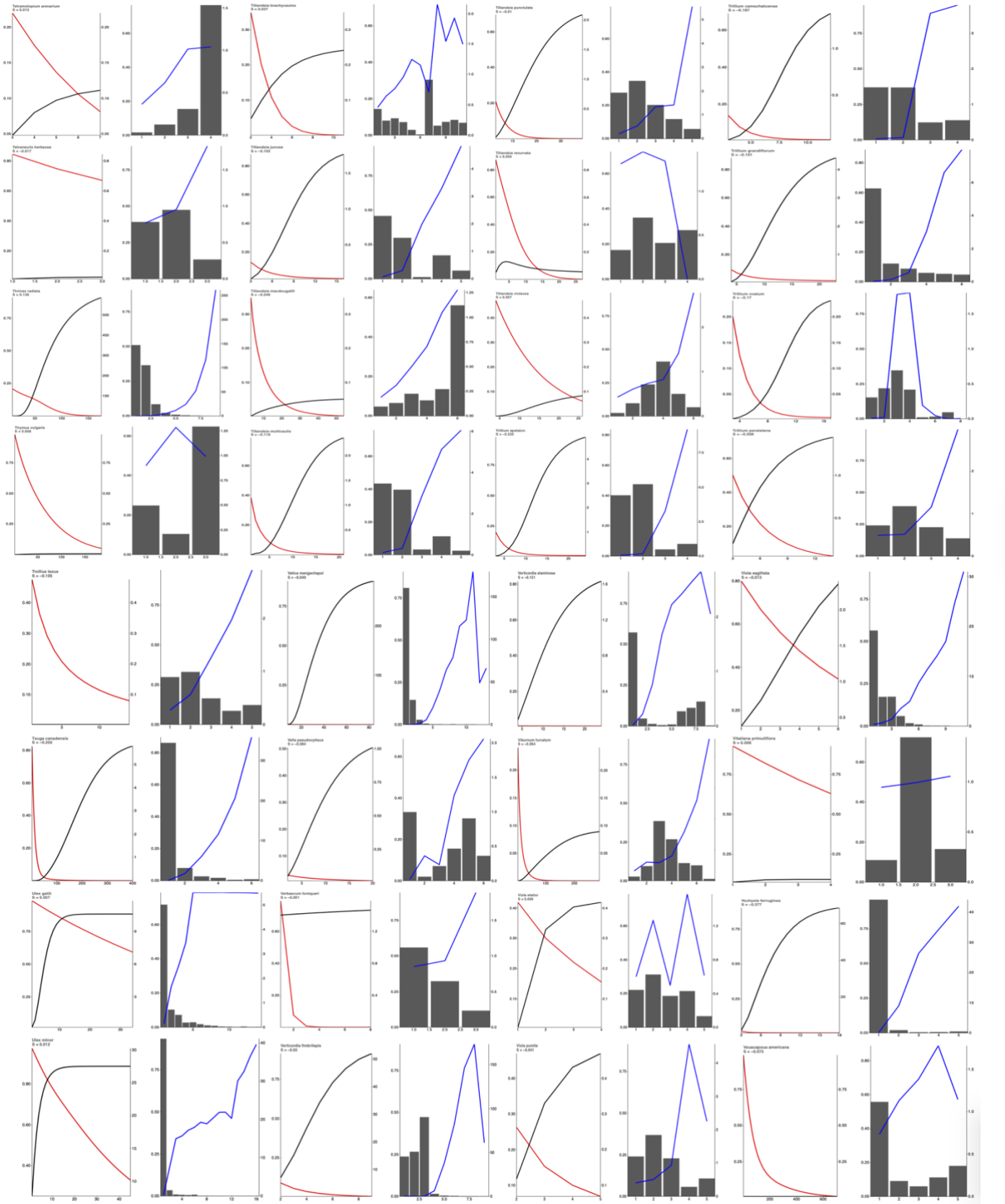












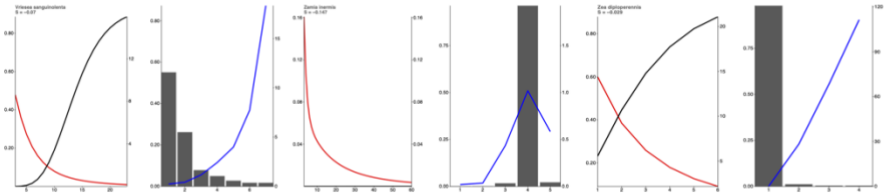


Table S1. Rate of actuarial senescence for 80 animal species. Each animal species for which we quantified a rate for actuarial senescence is listed, along with their respective taxonomic class, categorisation of senescence and value of *S*, and relevant source study. Values of *S* that fell within $-0.109 \leq S \leq 0.109$ were categorised as not different from zero and therefore equivalent to negligible senescence.

	Species	Class	Senescence category	Rate of senescence (*S*)	Source study
1	*Ammocrypta pellucida*	Actinopterygii	Negligible	0.000	Finch, M. *et al*. Recovery Potential Modelling of Eastern Sand Darter (*Ammocrypta pellucida*) in Canada. *Canadian Science Advisory Secretariat – Fish & Oceans Canada.* (2011).
2	*Astroblepus ubidiai*	Actinopterygii	Negligible	0.083	Vélez-Espino, L.A. Population viability and perturbation analyses in remnant populations of the Andean catfish *Astroblepus ubidiai. Ecology of freshwater fish*, **14(2),** 125-138 (2005).
3	*Clinocottus analis*	Actinopterygii	Negligible	0.020	Davis, J., & Levin, L. Importance of pre-recruitment life-history stages to population dynamics of the woolly sculpin *Clinocottus analis*. *Marine Ecology Progress Series*. **234**, 229–246. (2002).
4	*Cyprinodon diabolis*	Actinopterygii	Negative	-0.187	Beissinger, S. R. Digging the pupfish out of its hole: risk analyses to guide harvest of Devils Hole pupfish for captive breeding. *PeerJ*. **2**, e549 (2014).
5	*Erimyzon sucetta*	Actinopterygii	Negligible	-0.071	Young, A.M. & Koops, M.A. Recovery Potential Modelling of Lake Chubsucker (*Erimyzon sucetta*) in Canada. *Canadian Science Advisory Secretariat – Fish & Oceans Canada.* (2011).
6	*Genypterus blacodes*	Actinopterygii	Negligible	-0.105	González-Olivares, E., Aránguiz-Acuña, A., Ramos-Jiliberto, R., & Rojas-Palma, A. Demographical analysis of the pink ling *Genypterus blacodes* (Schneider 1801) in the austral demersal fishery: A matrix approach evaluating harvest and non-harvest states. *Fisheries Research*, **96(2-3)**, 216–222 (2009).
7	*Hybognathus argyritis*	Actinopterygii	Negligible	-0.045	Young, A.M. & Koops, M.A. Recovery Potential Modelling of *Hybognathus argyritis* in Canada. *Canadian Science Advisory Secretariat – Fish & Oceans Canada.* (2013).
8	*Notropis photogenis*	Actinopterygii	Negligible	-0.044	Young, A.M. & Koops, M.A. Recovery Potential Modelling of *Notropis photogenis* in Canada. *Canadian Science Advisory Secretariat – Fish & Oceans Canada.* (2012).
9	*Opsopoeodus emiliae*	Actinopterygii	Negligible	0.000	Young, A.M. & Koops, M.A. Recovery Potential Modelling of *Opsopoeodus emiliae* in Canada. *Canadian Science Advisory Secretariat – Fish & Oceans Canada.* (2012).
10	*Poecilia reticulata*	Actinopterygii	Negligible	-0.011	Bronikowski, A.M., Clark, M.E., Rodd, F.H. & Reznick, D.N. Population-dynamic consequences of predator-induced life history variation in the guppy (*Poecilia reticulata)*. *Ecology*. **83(8**), 2194-2204. (2002).
11	*Rutilus rutilus*	Actinopterygii	Negligible	0.000	Otjacques, W., De Laender, F. & Kestemont, P. Discerning the causes of a decline in a common European fish, the roach (*Rutilus rutilus*): A modelling approach. *Ecol. Model.* **322,** 92-100 (2016).

12	*Salvelinus malma*	Actinopterygii	Negligible	0.000	Ng, E.L., Frederick, J.P. & Quist, M.C. Population Dynamics Evaluation of Alternative Management Strategies for Nonnative Lake Trout in Priest Lake, Idaho. *N. Am. J. Fish. Manage.* **36,** 40-54 (2016).
13	*Stellifer illecebrosus*	Actinopterygii	Negligible	0.000	Foster, S. J., & Vincent, A. C. J. Advice in spite of great uncertainty: assessing and addressing bycatch of small fishes with limited data using *Stellifer illecebrosus* as a case study. *Aquatic Conservation: Marine and Freshwater Ecosystems*, **22(5)**, 639–651. (2012).
14	*Zoarces viviparus*	Actinopterygii	Negligible	-0.004	Bergek, S., Ma, Q., Franzén, F. & Appleberg, M. From individuals to populations: impacts of environmental pollution on natural eelpout populations. *Ecotoxicol. Environ. Saf.* **79,** 1-12 (2012).
15	*Epidalea calamita*	Amphibia	Negligible	0.000	Di Minin, E., & Griffiths, R. A. Viability analysis of a threatened amphibian population: modelling the past, present and future. *Ecography*, **34(1),** 162–169. (2010).
16	*Gorgonia ventalina*	Anthozoa	Negligible	-0.078	Sabat, A.M., & Toledo-Hernandez, C. Viability of Sea Fan Populations Impacted by Disease: Recruitment versus Incidence. *Journal of Marine Biology.* Vol 2015, Article ID 987060. (2015).
17	*Leptogorgia virgulata*	Anthozoa	Negative	-0.191	Gotelli, N.J. Demographic Models for *Leptogorgia virgulata*, A Shallow-Water Gorgonian. *Ecology.* **72(2),** 457-467 (1991).
18	*Paramuricea clavata*	Anthozoa	Negligible	-0.036	Linares, C., & Doak, D. Forecasting the combined effects of disparate disturbances on the persistence of long-lived gorgonians: a case study of *Paramuricea clavata*. *Marine Ecology Progress Series*, **402,** 59–68. (2010).
19	*Amazona vittata*	Aves	Negligible	0.000	Beissinger, S.R. *et al.* Anatomy of a bottleneck: diagnosing factors limiting population growth in the peurto rican parrot. *Ecol. Mono.* **78(2),** 185-203 (2008).
20	*Bostrychia hagedash*	Aves	Negligible	0.000	Duckworth, G. & Harebottle, D. Demography and population ecology of the Hadeda Ibis (*Bostrychia hagedash)* at its expanding range edge in South Africa. *J. Orni.* **153(2),** 421-430 (2012).
21	*Haliaeetus albicilla*	Aves	Positive	0.141	Krüger, O., Grünkorn, T. & Struwe-Juhl, B. The return of the white-tailed eagle (*Haliaeetus albicilla*) to northern Germany: Modelling the past to predict the future. *Biol. Cons.* **143(3),** 710-721 (2010).
22	*Milvus migrans*	Aves	Negligible	-0.003	Sergio, F., Tavecchia, G., Blas, J., López, L., Tanferna, A., & Hiraldo, F. Variation in age-structured vital rates of a long-lived raptor: Implications for population growth. *Basic and Applied Ecology*, **12(2),** 107–115. (2011).
23	*Picoides borealis*	Aves	Negligible	0.057	Maguire, L. A., Wilhere, G. F., & Dong, Q. Population Viability Analysis for Red-Cockaded Woodpeckers in the Georgia Piedmont. *The Journal of Wildlife Management*, **59(3)**, 533. (1995).
24	*Sterna hirundo*	Aves	Negligible	-0.019	Szostek, K.L., Schaub, M. & Becker, P.H. Immigrants are attracted by local pre-breeders and recruits in a seabird colony. *J. Anim. Ecol.* **83(5),** 1015-1024 (2014).

25	*Tympanuchus cupido*	Aves	Negligible	0.000	Fefferman, N.H. & Reed, J.M. A Vital Rate Sensitivity Analysis for Nonstable Age Distributions and Short-Term Planning. *J. Wild. Manage*. **70(3),** 649-656 (2010).
26	*Mya arenaria*	Bivalvia	Negative	-0.142	Brousseau, D.J. Population dynamics of the soft-shell clam *Mya arenaria. Marine. Biol.* **50(1),** 63-71 (1978).
27	*Nuttallia obscurata*	Bivalvia	Negligible	-0.030	Dudas, S. E., Dower, J. F., & Anholt, B. R. Invasion dynamics of the varnish clam (*Nuttallia obscurata):* A matrix demographic modelling approach. *Ecology*, **88(8),** 2084–2093. (2007).
28	*Yoldia notabilis*	Bivalvia	Negligible	-0.026	Nakaoka, M. Demography of the Marine Bivalve *Yoldia notabilis* in Fluctuating Environments: An Analysis Using a Stochastic Matrix Model. *Oikos*, **79(1)**, 59. (1997).
29	*Daphnia pulex*	Branchiopoda	Positive	0.332	Frank, P.W., Boll, C.D. & Kelly, R.W. Vital Statistics of Labortory Cultures of *Daphnia pulex* DeGeer as Related to Density. *Physiol and Biochem. Zool.* **30(4),** 287-305. (1957).
30	*Amphimedon compressa*	Demospongiae	Negligible	0.010	Mercado-Molina, A. E., Sabat, A. M., & Yoshioka, P. M. Demography of the demosponge *Amphimedon compressa*: Evaluation of the importance of sexual versus asexual recruitment to its population dynamics. *Journal of Experimental Marine Biology and Ecology*, **407(2),** 355–362. (2011).
31	*Xestospongia muta*	Demospongiae	Negligible	0.010	Pawlik, J., McMurray, S., & Henkel, T. Abiotic factors control sponge ecology in Florida mangroves. *Marine Ecology Progress Series*, **339**, 93–98. (2007).
32	*Isurus oxyrinchus*	Elasmobranchii	Negligible	-0.020	Tsai, W.-P., Sun, C.-L., Punt, A. E., & Liu, K.-M. Demographic analysis of the shortfin mako shark, *Isurus oxyrinchus*, in the Northwest Pacific using a two-sex stage-based matrix model. *ICES Journal of Marine Science*, **71(7),** 1604–1618. (2014).
33	*Haliotis laevigata*	Gastropoda	Negligible	-0.032	Fordham, D.A. *et al.* Population dynamics can be more important than physiological limits for determining range shifts under climate change. *GCB.* **19(10),** 3224-3237 (2013).
34	*Haliotis rufescens*	Gastropoda	Negligible	-0.050	Rogers-Bennett, L., & Leaf, R. T. Elasticity Analyses of Size-Based Red and White Abalone Matrix Models: Management and Conservation. *Ecological Applications*, **16(1),** 213–224. (2006).
35	*Lepetodrilus fucensis*	Gastropoda	Negligible	0.000	Kelly, N., & Metaxas, A. Understanding population dynamics of a numerically dominant species at hydrothermal vents: a matrix modeling approach. *Marine Ecology Progress Series*, **403**, 113–128. (2010).
36	*Umbonium costatum*	Gastropoda	Negligible	-0.015	Noda, T. & Nakao, S. Dynamics of an Entire Population of the Subtidal Snail *Umbonium costatum:* The Importance of Annual Recruitment Fluctuation. *J. Anim. Ecol.* **65(2),** 196. (1996).
37	*Cardisoma guanhumi*	Malacostraca	Negligible	0.017	Rodriguez-Fourquet, PhD thesis, 2004.
38	*Pagurus longicarpus*	Malacostraca	Negligible	-0.037	Damiani, C. C. (2005). Integrating direct effects and trait-mediated indirect effects using a projection matrix model. *Ecology*, **86(8),** 2068–2074 (2005).
39	*Acinonyx jubatus*	Mammalia	Negligible	0.098	Lubben, J., Tenhumberg, B., Tyre, A. & Rebarber, R. Management recommendations based on matrix projection models: The importance of considering biological limits. *Biol Cons.* **141(2),** 517-523 (2008).

40	*Alces alces*	Mammalia	Positive	0.134	Kvalnes, T. *et al.* Harvest-induced phenotypic selection in an island population of moose, *Alces alces. Evol.* **70(7),** 1486-1500 (2016).
41	*Bos frontalis*	Mammalia	Positive	0.187	Ahrestani, F.S. Iyer, S. Heitkönig, I.M.A. & Herbert, H.T.P. Life-history traits of gaur *Bos gaurus:* a first analysis. *Mamm. Rev.* **41(1),** 75-84 (2011).
42	*Callorhinus ursinus*	Mammalia	Positive	0.242	Barlow, J. & Boveng, P. Modeling age-specific mortality for marine mammal populations. *Marine. Mamm. Sci.* **7(1),** 50-65 (1991).
43	*Callospermophilus lateralis*	Mammalia	Negligible	0.000	Hostetler, J.A., Kneip, E., Van Vuren, D.H. Oli, M.K. Stochastic Population Dynamics of a Montane Ground-Dwelling Squirrel. *PloS1*. **7(3),** e34379 (2012).
44	*Didelphis aurita*	Mammalia	Negative	-0.222	Ferreira, M.S. *et al.* Life history of a neotropical marsupial: Evaluating potential contributions of survival and reproduction to population growth rate. *Mamm. Biol.* **78(6),** 406-411 (2013).
45	*Elephas maximus*	Mammalia	Positive	0.247	Goswami, V.R., Vasudev, D. & Oli, M.K. The importance of conflict-induced mortality for conservation planning in areas of human–elephant co-occurrence. *Biol. Cons.* **176,** 191-198 (2014).
46	*Eumetopias jubatus*	Mammalia	Negligible	0.050	Holmes, E.E. & York, A.E. Using Age Structure to Detect Impacts on Threatened Populations: a Case Study with Steller Sea Lions. *Cons. Biol.* **17(6),** 1794-1806 (2003).
47	*Lagothrix lagotricha*	Mammalia	Negligible	-0.066	Defler, T.R. & Stevenson, P.R. The Woolly Monkey*. Behavior, Ecology, Systematics, and Captive Research.* (Springer, 2014).
48	*Macaca mulatta*	Mammalia	Negligible	0.000	Blomquist, G.E., Sade, D.S. & Berard, J.D. Rank-Related Fitness Differences and Their Demographic Pathways in Semi-Free-Ranging Rhesus Macaques (*Macaca mulatta*). *I.J. Prim.* **32(1),** 193-208 (2011).
49	*Marmota flaviventris*	Mammalia	Negligible	-0.036	Ozgul, A., Oli, M. K., Armitage, K. B., Blumstein, D. T., & Van Vuren, D. H. Influence of Local Demography on Asymptotic and Transient Dynamics of a Yellow-Bellied Marmot Metapopulation. *Am. Nat.* **173(4),** 517–530 (2009).
50	*Mirounga angustirostris*	Mammalia	Positive	0.124	Clinton, W.L. & Le Boeuf, B.J. Sexual Selection's Effects on Male Life History and the Pattern on Male Mortality. *Ecol.* **74(6),** 1884-1892 (1993).
51	*Orcinus orca*	Mammalia	Negligible	0.037	Velez-Espino, L.A. et al. Comparative demography and viability of northeastern Pacific resident killer whale populations at risk. *Can Tech Report Fish & Aq Sci.* (2014).
52	*Phacochoerus aethiopicus*	Mammalia	Negligible	0.035	Rodgers, W.A. Warthog ecology in south east Tanzania. *Mammalia.* **48(3),** 327–350 (1984).
53	*Phocarctos hookeri*	Mammalia	Negligible	0.071	Meyer, S., Robertson, B. C., Chilvers, B. L., & Krkošek, M. Population dynamics reveal conservation priorities of the threatened New Zealand sea lion *Phocarctos hookeri*. *Marine Biology*, **162(8),** 1587–1596 (2015).
54	*Pongo abelii*	Mammalia	Positive	0.131	Wich, S.A. *et al.* Life history of wild Sumatran orangutans (*Pongo abelli).J. Hum. Evol.* **47(6),** 385-398 (2004).
55	*Presbytis thomasi*	Mammalia	Negligible	-0.032	Wich, S.A. *et al.* Demography and life history of Thomas langurs (*Presbytis thomasi). Am. J. Prim.* **69(6),** 641-651 (2006).

56	*Propithecus edwardsi*	Mammalia	Negative	-0.112	Dunham, A.E., Erhart, E.M. Overdoff, D.J. & Wright, P.C. Evaluating effects of deforestation, hunting, and El Niño events on a threatened lemur. *Biol. Cons.* **141(1),** 287-297 (2008).
57	*Saguinus imperator*	Mammalia	Negative	-0.163	Watsa, PhD Thesis, 2013.
58	*Urocitellus armatus*	Mammalia	Negligible	0.000	Oli, M.K. Slade, N.A. & Dobson, F.S. Effect of density reduction on uinta groun d squirrels: Analysis of life table response experiments. *Ecology.* **82(7),** 1921-1929 (2001).
59	*Urocitellus beldingi*	Mammalia	Negligible	0.031	Sherman, P.W. & Morton, M.L. Demography of Belding's Ground Squirrels. *Ecology.* **65(5),** 1617-1628 (1984).
60	*Spermophilus dauricus*	Mammalia	Negligible	0.037	Luo, J. & Fox, B.J. Life-Table Comparisons between Two Ground Squirrels. *J. Mamm.* **71(3),** 364-370 (1990).
61	*Tamiasciurus hudsonicus*	Mammalia	Positive	0.174	McAdam, A.G., Boutin, S., Sykes, A.K. & Humphries, M.M. Life histories of female red squirrels and their contributions to population growth and lifetime fitness. *Ecoscience.* **14(3),** 362-369 (2007).
62	*Chelodina expansa*	Reptilia	Negligible	-0.064	Spencer, R.J., & Thompson, M.B. Experimental Analysis of the Impact of Foxes on Freshwater Turtle Populations. *Conservation Biology*, **19(3),** 845–854 (2005).
63	*Chrysemys picta*	Reptilia	Negligible	0.075	Mitchell, J. C. Population Ecology and Life Histories of the Freshwater Turtles *Chrysemys picta* and *Sternotherus odoratus* in an Urban Lake. *Herpetological Monographs*, **2**, 40 (1988).
64	*Crocodylus niloticus*	Reptilia	Negligible	-0.049	Hutton, PhD thesis, 1984.
65	*Emydura macquarii*	Reptilia	Negative	-0.143	Spencer, R.-J., & Thompson, M. B. (2005). Experimental Analysis of the Spencer, R.J., & Thompson, M.B. Experimental Analysis of the Impact of Foxes on Freshwater Turtle Populations. *Conservation Biology*, **19(3),** 845–854 (2005).
66	*Kinosternon integrum*	Reptilia	Negligible	-0.020	Macip-Rios, R. et al. Demography of two populations of the Mexican mud turtle (*Kinosternon integrum)* in central Mexico. *Herp J.* **21(4),** 235-245 (2011).
67	*Kinosternon subrubrum*	Reptilia	Positive	0.268	Frazer, N.B, Whitfield Gibbons, J. & Greene, J.L. Life History and Demography of the Common Mud Turtle *Kinosternon Subrubrum* in South Carolina, USA. *Ecology.* **72(6),** 2218-2231 (1991).
68	*Malaclemys terrapin*	Reptilia	Negligible	-0.108	Crawford, B. A., Maerz, J. C., Nibbelink, N. P., Buhlmann, K. A., & Norton, T. M. Estimating the consequences of multiple threats and management strategies for semi-aquatic turtles. *Journal of Applied Ecology*, *51(2)*, 359–366 (2014).
69	*Podocnemis expansa*	Reptilia	Negative	-0.142	Mogollones, S. C., Rodríguez, D. J., Hernández, O., & Barreto, G. R. A Demographic Study of the Arrau Turtle (*Podocnemis expansa*) in the Middle Orinoco River, Venezuela. *Chelonian Conservation and Biology*, **9(1),** 79–89 (2010).
70	*Sceloporus grammicus*	Reptilia	Negligible	-0.046	Pérez-Mendoza, H. A., Zúñiga-Vega, J. J., Zurita-Gutiérrez, Y. H., Fornoni, J., Solano-Zavaleta, I., Hernández-Rosas, A. L., & Molina-Moctezuma, A. Demographic Importance of the Life-Cycle Components in *Sceloporus grammicus*. *Herpetologica*, **69(4),** 411–435 (2013).
71	*Xenosaurus grandis*	Reptilia	Negligible	0.033	Zúñiga-Vega, J. J., Valverde, T., Rojas-Gonzalez, R.I. & Lemos-Espinal, J.A. Analysis of the population dynamics of an endangered lizard

					(*Xenosaurus grandis*) through the use of projection matrices. Copeia. 2, 324-335 (2007).
72	*Xenosaurus platyceps*	Reptilia	Negligible	-0.100	Jones, C., Rojas-González, I., Lemos-Espinal, J., & Zúñiga-Vega, J. Demography of *Xenosaurus platyceps* (Squamata: Xenosauridae): a comparison between tropical and temperate populations. *Amphibia-Reptilia*, **29(2)**, 245–256 (2008).
73	*Connochaetes taurinus*	Mammalia	Positive	0.185	Attwell, C.A.M. Population ecology of the blue wildebeest *Connochaetes taurinus taurinus* in Zululand, South Africa. *Af. J. Ecol.* **20(3),** 147-168 (1982).
74	*Larus heermanni*	Aves	Positive	0.303	Velarde, E. & Ezcurra, E. Are seabirds' life history traits maladaptive under present oceanographic variability? The case of Heermann's Gull (*Larus heermanni*). *The Condor.* **120(2),** 388-401 (2018).
75	*Arctocephalus australis*	Mammalia	Negligible	-0.016	Lima, M. & Paez, E. Demogrpahy and Population Dynamics of South American Fur Seals. *Journal of Mammalogy.* **78(3),** 914-920 (1997).
76	*Dama dama*	Mammalia	Positive	0.164	McElligot, A.G., Altwegg, R. & Hayden, T.J. Age-specific survival and reproductive probabilities: evidence for senescence in male fallow deer (*Dama dama*)**.** *Proc R Soc Lond B.* 269, 1129-3 (2002).
77	*Dasypus novemcinctus*	Mammalia	Negligible	0.015	Oli, M., Loughry, W.J., Caswell, H. & Perez-Heydrich, C**.** Dyanmics of leprosy in nine-banded armadillos: Net reproductive number and effects on host population dynamics. *Ecol Modelling.* **350,** 100-108 (2017).
78	*Falco concolor*	Aves	Negligible	-0.033	McGrady, M.J. *et al.* Sooty Falcon *Falco concolor* reproduction and population dynamics on the islands in the Sea of Oman. *Ibis.* **159(4),** 828-840 (2017).
79	*Odocoileus virginianus*	Mammalia	Negligible	-0.078	Edmunds, PhD Thesis, 2013.
80	*Ammocrypta pellucida*	Mammalia	Negligible	-0.101	Lambert, C.M.S. *et al.* Cougar Population Dynamics and Viability in the Pacific Northwest. *J. Wild. Manage.* **70(1),** 246-254 (2006).

Table S2. Rate of actuarial senescence for 395 plant species. Each plant species for which we quantified a rate for actuarial senescence is listed, along with their respective taxonomic class, categorisation of senescence, value of *S*, and relevant source study. Values of *S* that fell within $-0.129 \leq S \leq 0.129$ were categorised as not different from zero and therefore equivalent to negligible senescence.

	Species	Taxonomic class	Senescence cateogry	Rate of senescence (*S*)	Source study
1	*Liatris scariosa*	Magnoliopsida	Negligible	0.028	Ellis, M. M. *et al*. Matrix population models from 20 studies of perennial plant populations. *Ecology.* **93(4),** 951–951. (2012).
2	*Tetraneuris herbacea*	Magnoliopsida	Negligible	-0.017	Campbell, L.G. & Husband, L.G. Impact of clonal growth on effective population size in *Hymenoxys herbacea* (Asteraceae). *Nature. Hered.* **94,** 526-532 (2005).
3	*Coespeletia spicata*	Magnoliopsida	Negligible	0.008	Silva, J. F., Trevisan, M. C., Estrada, C. A., & Monasterio, M. Comparative demography of two giant caulescent rosettes (*Espeletia timotensis* and *E. spicata*) from the high tropical Andes. *Global Ecology and Biogeography*. **9(5),** 403–413 (2000).
4	*Coespeletia timotensis*	Magnoliopsida	Negligible	0.001	Silva, J. F., Trevisan, M. C., Estrada, C. A., & Monasterio, M. Comparative demography of two giant caulescent rosettes (*Espeletia timotensis* and *E. spicata*) from the high tropical Andes. *Global Ecology and Biogeography*. **9(5),** 403–413 (2000).
5	*Ambrosia deltoidea*	Magnoliopsida	Negligible	-0.011	Goldberg, D.E. & Turner, R.M. Vegetation Change and Plant Demography in Permenant Plots in the Sonoran Desert. *Ecology.* **67(3),** 695-712 (1986).
6	*Echinacea angustifolia*	Magnoliopsida	Negligible	-0.115	Hurlburt, PhD thesis, 1999.
7	*Pyrrocoma radiata*	Magnoliopsida	Negligible	-0.019	Pfingsten, PhD Thesis, 2013.
8	*Solidago altissima*	Magnoliopsida	Negative	-0.179	Schmid, JPDP, 1990.
9	*Tetramolopium arenarium*	Magnoliopsida	Negligible	0.013	Falkner, M.B., Laven, R.D. & Aplet, G.H. Experiments on germination and early growth of three rare and endemic species of Hawaiian *Tetramolopium* (Asteraceae). *Biol. Cons.* **80(1),** 39-47 (1997).
10	*Artemisia genipi*	Magnoliopsida	Negligible	-0.060	Marcante, S., Winkler, E., & Erschbamer, B. Population dynamics along a primary succession gradient: do alpine species fit into demographic succession theory? *Annals of Botany*. **103(7),** 1129–1143 (2009).
11	*Santolina melidensis*	Magnoliopsida	Negligible	-0.024	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
12	*Senecio filaginoides*	Magnoliopsida	Negligible	0.055	Cipriotti, P.A. & Aguiar, M.R. Direct and indirect effects of grazing constrain shrub encroachment in semi-arid Patagonian steppes. *Appl. Veg. Sci.* **15(1),** 35-47 (2011).
13	*Hypochaeris radicata*	Magnoliopsida	Negligible	-0.016	Jongejans, E. & De Kroon, H. Space versus time variation in the population dynamics of three co-occurring perennial herbs. *J. Ecol.* **93(4),** 681-692 (2005).
14	*Sonchus pustulatus*	Magnoliopsida	Negligible	-0.051	Silva, J.L., Mejías, J.A., González, G. & Begoña, M. Demographic vulnerability in cliff-dwelling sonchus species endemic to the

					western Mediterranean. *Bas. Appl. Eco.* **16(4),** 316-324 (2015).
15	*Scorzonera hispanica*	Magnoliopsida	Negligible	-0.106	Münzbergová, Z. Effect of population size on the prospect of species survival. *Fol. Geobot.* **41(2),** 137-150 (2006).
16	*Centaurea horrida*	Magnoliopsida	Negligible	-0.096	Pisanu, S. *et al.* Demographic effects of large, introduced herbivores on a long-lived endemic plant. *Plant. Ecol.* **213(10),** 1453-1553 (2012).
17	*Centaurea podospermifolia*	Magnoliopsida	Negligible	0.006	Emery, S.M. & Gross, K.L. Effects of timing of prescribed fire on the demography of an invasive plant, spotted knapweed *Centaurea maculosa. J. Appl. Ecol.* **42(1),** 60-69 (2005).
18	*Cheirolophus metlesicsii*	Magnoliopsida	Negligible	0.030	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
19	*Saussurea medusa*	Magnoliopsida	Negligible	0.021	Law, W., Salick, J. & Knight, T.M. The effects of pollen limitation on population dynamics of snow lotus (*Saussurea medusa* and *S. laniceps*, Asteraceae): Threatened Tibetan medicinal plants of the eastern Himalayas. *Plant. Ecol.* **210(2),** 342-357 (2010).
20	*Cirsium pannonicum*	Magnoliopsida	Negligible	-0.092	Münzbergová, Z. Determinants of species rarity: population growth rates of species sharing the same habitat. *Am. J. Bot.* **92(12),** 1987-1994 (2005).
21	*Cirsium acaule*	Magnoliopsida	Negligible	-0.127	Münzbergová, Z. Determinants of species rarity: population growth rates of species sharing the same habitat. *Am. J. Bot.* **92(12),** 1987-1994 (2005).
22	*Cirsium dissectum*	Magnoliopsida	Negligible	-0.011	Jongejans, E., de Vere, N. & de Kroon, H. Demographic vulnerability of the clonal and endangered meadow thistle. *Plant. Ecol.* **198,** 225-240 (2008).
23	*Cirsium vulgare*	Magnoliopsida	Negligible	0.035	Bullock, J.M., Clear Hill, B. & Silvertown, J. Demography of *Cirsium Vulgare* in a Grazing Experiment. *J. Ecol.* **82(1),** 101-111 (1994).
24	*Carlina vulgaris*	Magnoliopsida	Negligible	0.022	Jongejans, E. *et al.* Region versus site variation in the population dynamics of three short-lived perennials. *J. Ecol.* **98(2),** 279-289 (2010).
25	*Lomatium bradshawii*	Magnoliopsida	Negligible	-0.045	Kaye, T.N. & Pyke, D.A. The effect of stochastic technique on estimates of population viability from transition matrix models. *Ecology.* **84(6)**, 1464-1476 (2003).
26	*Lomatium cookii*	Magnoliopsida	Negligible	-0.058	Kaye, T.N. & Pyke, D.A. The effect of stochastic technique on estimates of population viability from transition matrix models. *Ecology.* **84(6)**, 1464-1476 (2003).
27	*Pimpinella saxifraga*	Magnoliopsida	Negligible	-0.038	Auestad, I., Rydgren, K., Jongejans, E. & de Kroon, H. *Pimpinella saxifrage* is maintained in road verges by mosaic management. *Biol. Cons.* **143(4),** 899-907 (2010).
28	*Eryngium cuneifolium*	Magnoliopsida	Negligible	-0.003	Menges, E. S., & Quintana-Ascencio, P. F. Population viability with fire in *Eryngium cuneifolium:* deciphering a decade of demographic data. *Ecological Monographs.* 7**4(1**), 79–99 (2004).

29	*Eryngium maritimum*	Magnoliopsida	Negligible	-0.043	Curie, C.M., Stabbetorp, O.E. & Nordal, I. *Eryngium maritimum*, biology of a plant at its northernmost localities. *Nor. J. Bot.* **24(5),** 617-628 (2008).
30	*Eryngium alpinum*	Magnoliopsida	Negligible	-0.122	Andrello, M. *et al.* Effects of management regimes and extreme climatic events on plant population viability in *Eryngium alpinum*. *Biological Conservation*. **147(1),** 99–106 (2012).
31	*Sanicula europaea*	Magnoliopsida	Negligible	-0.024	Gustafsson, C. & Ehrlén, J. Effects of intraspecific and interspecific density on the demography of a perennial herb, *Sanicula europaea. Oikos.* **100(2),** 317-324 (2003).
32	*Mulinum spinosum*	Magnoliopsida	Negligible	-0.009	Cipriotti, P.A. & Aguiar, M.R. Direct and indirect effects of grazing constrain shrub encroachment in semi-arid Patagonian steppes. *Appl. Veg. Sci.* **15(1),** 35-47 (2011).
33	*Panax quinquefolius*	Magnoliopsida	Negligible	-0.034	Charron, D., & Gagnon, D. The Demography of Northern Populations of *Panax Quinquefolium* (American Ginseng). *The Journal of Ecology*. **79(2),** 431. (1991).
34	*Nardostachys jatamansi*	Magnoliopsida	Negligible	-0.074	Ghimire, S.K. *et al.* Demographic variation and population viability in a threatened Himalayan medicinal and aromatic herb *Nardostachys grandiflora:* matrix modelling of harvesting effects in two contrasting habitats. *J. Appl. Ecol.* **45,** 41-51 (2008).
35	*Dipsacus fullonum*	Magnoliopsida	Positive	0.194	Werner, P.A. & Caswell, H. Population Growth Rates and Age Versus Stage-Distribution Models for Teasel (Dipsacus Sylvestris Huds.) *Ecology.* **58(5),** 1103-1111 (1977).
36	*Succisa pratensis*	Magnoliopsida	Negligible	-0.040	Milden, PhD Thesis, 2005.
37	*Lonicera maackii*	Magnoliopsida	Negative	-0.136	Burns, J.H. *et al.* Greater sexual reproduction contributes to differences in demography of invasive plants and their noninvasive relatives. *Ecology.* **94(5),** 995-1004 (2013).
38	*Viburnum furcatum*	Magnoliopsida	Negligible	-0.064	Hara, M., Kanno, H., Hirabuki, Y., & Takehara, A. Population dynamics of four understorey shrub species in beech forest. *Journal of Vegetation Science.* **15(4),** 475–484. (2004).
39	*Eremophila maitlandii*	Magnoliopsida	Negligible	-0.056	Watson, I.W. Westoby, M. & Holm. A.McR. Demography of Two Shrub Species from an Arid Grazed Ecosystem in Western Australia 1983-93. *J. Ecol.* **85(6),** 815-832 (1997).
40	*Dicerandra frutescens*	Magnoliopsida	Negligible	-0.037	Menges, E. S., Quintana Ascencio, P. F., Weekley, C. W., & Gaoue, O. G. Population viability analysis and fire return intervals for an endemic Florida scrub mint. *Biological Conservation.* **127(1),** 115–127 (2006).
41	*Thymus vulgaris*	Magnoliopsida	Negligible	0.008	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
42	*Dracocephalum austriacum*	Magnoliopsida	Negligible	-0.095	Andrello, PhD thesis, 2010.
43	*Rosmarinus tomentosus*	Magnoliopsida	Negative	-0.211	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
44	*Pedicularis furbishiae*	Magnoliopsida	Negligible	-0.054	Menges, E.S. Population Viability Analysis for an Endangered Plant. *Cons. Biol.* **4(1),** 52-62 (1990).

45	*Mimulus lewisii*	Magnoliopsida	Negligible	-0.062	Angert, A.L. Demography of Central and Marginal Populations of Monkeyflowers (*Mimulus cardinalis* and *M. Lewisii*)**.** *Ecol.* **87(8),** 2014-2025 (2006).
46	*Avicennia germinans*	Magnoliopsida	Negligible	-0.126	Lopez-Hoffman, L., Ackerley, D. D., Aanten, N. P. R., Denoyer, J. L., & Martinez-Ramos, M. Gap-dependence in mangrove life-history strategies: a consideration of the entire life cycle and patch dynamics. *Journal of Ecology.* **95(6**), 1222–1233 (2007).
47	*Pinguicula vulgaris*	Magnoliopsida	Negligible	-0.093	Svensson, B. M., Carlsson, B. A., Karlsson, P. S., & Nordell, K. O. Comparative Long-Term Demography of Three Species of Pinguicula. *The Journal of Ecology.* **81(4),** 635 (1993).
48	*Pinguicula alpina*	Magnoliopsida	Negligible	-0.024	Svensson, B. M., Carlsson, B. A., Karlsson, P. S., & Nordell, K. O. Comparative Long-Term Demography of Three Species of Pinguicula. *The Journal of Ecology.* **81(4),** 635 (1993).
49	*Lantana camara*	Magnoliopsida	Negative	-0.226	Raghu, S., Osunkoyo, O.O., Perrett, C. & Pichancourt, J-B. Historical demography of *Lantana camara L.* reveals clues about the influence of land use and weather in the management of this widespread invasive species. *Bas. Appl. Ecol.* **15(7),** 565-572 (2014).
50	*Verbascum fontqueri*	Magnoliopsida	Negligible	-0.001	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
51	*Anarrhinum fruticosum*	Magnoliopsida	Negligible	-0.011	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
52	*Plantago media*	Magnoliopsida	Negligible	-0.040	Eriksson, Å., & Eriksson, O. Population dynamics of the perennial *Plantago media* in semi-natural grasslands. *Journal of Vegetation Science.* **11(2),** 245–252 (2000).
53	*Plantago coronopus*	Magnoliopsida	Negligible	-0.003	Villellas, J. *et al.* Plant performance in central and northern peripheral populations of the widespread *Plantago coronopus. Ecography*. **36(2)**, 136-145 (2012).
54	*Antirrhinum molle*	Magnoliopsida	Negligible	0.001	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
55	*Antirrhinum subbaeticum*	Magnoliopsida	Negligible	-0.036	Iriondo; Albert; Gimemez; Lozana; Escuerdo. 978-84-8014-746-0
56	*Ramonda myconi*	Magnoliopsida	Negative	-0.146	Xavier Picó, F., & Riba, M. *Plant Ecology.* **161(1),** 1–13 (2002).
57	*Cyrtandra dentata*	Magnoliopsida	Negligible	-0.122	Bialic-Murphy, L., Gaoue, O. G., & Kawelo, K. Microhabitat heterogeneity and a non-native avian frugivore drive the population dynamics of an island endemic shrub, *Cyrtandra dentata. Journal of Applied Ecology.* **54(5),** 1469-1477. (2017).
58	*Cryptantha flava*	Magnoliopsida	Negligible	-0.048	Salguero-Gómez, R. *et al.* Long-term individual-level population dynamics of a native desert chamaephyte. *Ecology.* **95(2),** 557-577 (2014).
59	*Lithospermum ruderale*	Magnoliopsida	Negative	-0.193	Bricker, M., & Maron, J. Post dispersal seed predation limits the abundance of a long-lived perennial forb *(Lithospermum ruderale*). *Ecology.* **93(3),** 532–543 (2012).
60	*Gardenia actinocarpa*	Magnoliopsida	Negligible	-0.011	Osunkoya, O.O. Two-sex population projection of the endemic and dioecious rainforest shrub, *Gardenia*

					actinocarpa (Rubiaceae). *Biol. Cons.* **114(1),** 39-51 (2003).
61	*Guettarda viburnoides*	Magnoliopsida	Negative	-0.143	Loayza, A. P., & Knight, T. Seed dispersal by pulp consumers, not "legitimate" seed dispersers, increases *Guettarda viburnoides* population growth. *Ecology.* **91(9),** 2684–2695 (2010).
62	*Himatanthus drasticus*	Magnoliopsida	Negligible	-0.098	Baldauf, C., Corrêa, C. E., Ferreira, R. C., & dos Santos, F. A. M. Assessing the effects of natural and anthropogenic drivers on the demography of *Himatanthus drasticus* (Apocynaceae): Implications for sustainable management. *Forest Ecology and Management.* **354**, 177–184 (2015).
63	*Alyxia stellata*	Magnoliopsida	Negative	-0.139	Wong, T. M., & Ticktin, T. Using population dynamics modelling to evaluate potential success of restoration: a case study of a Hawaiian vine in a changing climate. *Environmental Conservation*. **42(1),** 20–30 (2014).
64	*Frasera speciosa*	Magnoliopsida	Negligible	-0.004	Che-Castaldo, J.P. & Inouye, D.W. The effects of dataset length and mast seeding on the demography of *Frasera speciosa*, a long-lived monocarpic plant. *Ecosphere.* **2(11),** 1-18 (2011).
65	*Gentiana pneumonanthe*	Magnoliopsida	Negligible	-0.016	Oostermeijer, J.G.B. *et al.* Temporal and Spatial Variation in the Demography of *Gentiana Pneumonanthe*, a Rare Perennial Herb. *J. Ecol.* **84(2),** 153-166 (1996).
66	*Ipomoea leptophylla*	Magnoliopsida	Negligible	-0.052	Keeler, K. H. Survivorship and Recruitment in a Long-lived Prairie Perennial, *Ipomoea leptophylla* (Convolvulaceae). *American Midland Naturalist*. **126(1),** 44 (1991).
67	*Calluna vulgaris*	Magnoliopsida	Negligible	0.001	Scandrett, E. & Gimmingham, C.H. A model of *Calluna* population dynamics; the effects of varying seed and vegetative regeneration. *Vegetatio*. **84(2),** 143-152 (1989).
68	*Rhododendron maximum*	Magnoliopsida	Positive	0.309	McGraw, J. B. Effects of age and size on life histories and population growth of *Rhododendron maximum* shoots. *American Journal of Botany.* **76(1),** 113–123 (1989).
69	*Rhododendron ponticum*	Magnoliopsida	Positive	0.296	Salguero-Gomez, MSc thesis, 2004.
70	*Sarracenia alata*	Magnoliopsida	Negative	-0.138	Brewer, J.S. A demographic analysis of fire-stimulated establishment of *Sarracenia alata* (Sarraceniaceae). *Am. J. Bot.* **88(7),** 1250-1257 (2001).
71	*Sarracenia purpurea*	Magnoliopsida	Negligible	0.017	Gotelli, N.J. & Ellison, A.M. Forecasting extinction risk with nonstationary matrix models. *Ecol. Appl.* **16(1),** 51-61 (2006).
72	*Styrax obassis*	Magnoliopsida	Negative	-0.314	Abe, S., Nakashizuka, T., & Tanaka, H. Effects of canopy gaps on the demography of the subcanopy tree *Styrax obassia*. *Journal of Vegetation Science*, **9(6),** 787–796 (1998).
73	*Primula farinosa*	Magnoliopsida	Negligible	-0.005	Lindborg, R. & Ehrlén, J. Evaluating the Extinction Risk of a Perennial Herb: Demographic Data versus Historical Records**.** *Cons. Biol.* **16(3),** 683-690 (2002).
74	*Primula vulgaris*	Magnoliopsida	Negative	-0.223	Valdés, A., García, D., García, M. B., & Ehrlén, J. Contrasting effects of different landscape characteristics on population

					growth of a perennial forest herb. *Ecography.* **37(3),** 230–240 (2013).
75	*Primula veris*	Magnoliopsida	Negative	-0.285	Lethilia, K., Syrjanen, K., Leimu, R., Garcia, M. B., & Ehrlen, J. Habitat Change and Demography of *Primula veris*: Identification of Management Targets. *Conservation Biology.* **20(3),** 833–843 (2006).
76	*Primula elatior*	Magnoliopsida	Negligible	-0.070	Jacquemyn, H., & Brys, R. Effects of stand age on the demography of a temperate forest herb in post-agricultural forests. *Ecology*. **89(12**), 3480–3489 (2008).
77	*Vitaliana primuliflora*	Magnoliopsida	Negligible	0.006	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
78	*Ardisia elliptica*	Magnoliopsida	Negative	-0.169	Koop, A. L., & Horvitz, C. C. Projection matrix analysis of the demography of an invasive, non-native shrub (*Ardisia elliptica)*. *Ecology.* **86(10),** 2661–2672 (2005).
79	*Bertholletia excelsa*	Magnoliopsida	Negligible	0.046	Zuidema, P. A., & Boot, R. G. A. Demography of the Brazil nut tree (*Bertholletia excelsa*) in the Bolivian Amazon: impact of seed extraction on recruitment and population dynamics. *Journal of Tropical Ecology.* **18(1),** 1–31 (2002).
80	*Grias peruviana*	Magnoliopsida	Negligible	-0.086	Peters, *Ecol & Manag Non-timber Forest Resources*, 1995.
81	*Manilkara zapota*	Magnoliopsida	Negative	-0.193	Cruz-Rodríguez, J. A., López-Mata, L., & Valverde, T. A comparison of traditional elasticity and variance-standardized perturbation analyses: a case study with the tropical tree species *Manilkara zapota* (Sapotaceae). *Journal of Tropical Ecology*. **25(2),** 135–146 (2009).
82	*Ipomopsis aggregata*	Magnoliopsida	Negligible	0.035	Brody, A.K., Price, M.V. & Waser, N.M. Life-history consequences of vegetative damage in scarlet gilia, a monocarpic plant. *Oikos.* **116(6),** 975-985 (2007).
83	*Ipomopsis tenuituba*	Magnoliopsida	Negligible	0.067	Campbell, D.R. & Waser, N.M. Evolutionary Dynamics of an *Ipomopsis* Hybrid Zone: Confronting Models with Lifetime Fitness Data. *Am. Nat.* **169(3),** 298-310 (2007).
84	*Pterocereus gaumeri*	Magnoliopsida	Negligible	0.073	Méndez, M., Durán, R., Olmsted, I. & Oyama, K. Population Dynamics of *Pterocereus gaumeri*, a Rare and Endemic Columnar Cactus of Mexico. *Biotropica.* **36(4),** 492-504 (2004).
85	*Harrisia fragrans*	Magnoliopsida	Negative	-0.146	Rae, J. G., & Ebert, T. A. Demography of the Endangered Fragrant Prickly Apple Cactus, Harrisia fragrans. *International Journal of Plant Sciences.* **163(4),** 631–640. (2002).
86	*Melocactus ernestii*	Magnoliopsida	Negative	-0.335	Hughes, F.M. *et al.* Demographic processes and anthropogenic threats of lithophytic cells in eastern Brazil. *Braz. J. Bot.* **41,** 631-640 (2018).
87	*Melocactus bahiensis*	Magnoliopsida	Negative	-0.212	Hughes, F.M. *et al.* Demographic processes and anthropogenic threats of lithophytic cells in eastern Brazil. *Braz. J. Bot.* **41,** 631-640 (2018).
88	*Stenocereus eruca*	Magnoliopsida	Positive	0.188	Clark-Tapia, PhD thesis, 2004.
89	*Escontria chiotilla*	Magnoliopsida	Negligible	0.125	Ortega-Baes, PhD thesis, 2001.
90	*Cephalocereus senilis*	Magnoliopsida	Negligible	-0.116	Cedillo Castillo, MSc thesis, 2007.

91	*Neobuxbaumia mezcalaensis*	Magnoliopsida	Negligible	0.032	Esparza-Olguin, L., Valverde, T. & Mandujano, M.C. Comparative demographic analysis of three *Neobuxbaumia* species (cactaceae) with differing degree of rarity. *Population Ecology.* **47(3)** 229-245 (2005).
92	*Neobuxbaumia macrocephala*	Magnoliopsida	Negligible	-0.123	Esparza-Olguin, L., Valverde, T. & Mandujano, M.C. Comparative demographic analysis of three *Neobuxbaumia* species (cactaceae) with differing degree of rarity. *Population Ecology.* **47(3)** 229-245 (2005).
93	*Neobuxbaumia polylopha*	Magnoliopsida	Negative	-0.174	Arroyo-Cosultchi, G., Golubov, J. & Mandujano, M.C. Pulse seedling recruitment on the population dynamics of a columnar cactus: Effect of an extreme rainfall event. *Acta Oecologica.* **71**, 52-60 (2016)
94	*Neobuxbaumia tetetzo*	Magnoliopsida	Negligible	0.082	Esparza-Olguin, L., Valverde, T. & Mandujano, M.C. Comparative demographic analysis of three *Neobuxbaumia* species (cactaceae) with differing degree of rarity. *Population Ecology.* **47(3)** 229-245 (2005).
95	*Mammillaria solisioides*	Magnoliopsida	Negligible	-0.113	Rodriguez Ortega, PhD Thesis, 2008.
96	*Mammillaria gaumeri*	Magnoliopsida	Negligible	-0.117	Ferrer M, Dura´n R, Me´ndez M, Dorantes A, Dzib G. Dina´mica poblacional de genets y ramets de *Mammillaria gaumeri* cacta´cea ende´mica de Yucata´n. Bol. Soc. Bot. Me´xico **89,** 83 – 105 (2011).
97	*Mammillaria huitzilopochtli*	Magnoliopsida	Negligible	0.020	Martinez, A-F., Medina, G.I.M., Golubov, J., Montana, C. & Mandujano, M.C. Demography of an endangered epidemic rupicolous cactus. *Plant Ecology.* **210,** 53-66 (2010).
98	*Mammillaria crucigera*	Magnoliopsida	Negligible	-0.081	Contreras, C., & Valverde, T. Evaluation of the conservation status of a rare cactus (*Mammillaria crucigera*) through the analysis of its population dynamics. *Journal of Arid Environments.* **51(1),** 89–102 (2002).
99	*Mammillaria mystax*	Magnoliopsida	Negligible	0.080	Saldivar Sanches, Navarro Carbajal, Cact Suc Mex, 2012.
100	*Mammillaria dixanthocentron*	Magnoliopsida	Negligible	-0.054	Ramos Lopez, Msc Thesis, 2007.
101	*Mammillaria supertexta*	Magnoliopsida	Negligible	-0.001	Avendano Calco, MSc thesis, 2007.
102	*Mammillaria magnimamma*	Magnoliopsida	Negligible	-0.075	Valverde, T., Quijas, S., López-Villavicencio, M., & Castillo, S. Population dynamics of *Mammillaria magnimamma (Cactaceae*) in a lava-field in central Mexico. *Plant Ecology (formerly Vegetatio)*. **170(2),** 167–184 (2004).
103	*Mammillaria pectinifera*	Magnoliopsida	Negligible	0.001	Valverde, P.L. & Zavala-Hurtado, J.A. Assessing the ecological status of *Mammillaria pectinifera* Weber (cactaceae), a rare and threatened species endemic of the Tehuacan-Cuitcatlan Region in Central Mexico. *Journal of Arid Environments.* **64(2)** 193-208 (2006).
104	*Mammillaria hernandezii*	Magnoliopsida	Negative	-0.149	Rodriguez Ortega, PhD thesis, 2008.
105	*Mammillaria napina*	Magnoliopsida	Negative	-0.156	Rodriguez Ortega, PhD thesis, 2008.
106	*Escobaria robbinsorum*	Magnoliopsida	Negligible	-0.087	Schalzel, R.J., Reichenbacher, F.W. & Rutman, S. Demographic study of the rare coryphantha *robbinsorum* (cacraceae) in southeastern Arizona. *Madroño.* **42,** 332-348 (1995).
107	*Lophophora diffusa*	Magnoliopsida	Negligible	-0.052	Dias Segura, MSc Thesis, 2013.

108	*Stenocactus crispatus*	Magnoliopsida	Negative	-0.189	López Flores, D., Navarro Carbajal, M.dC. *Cactaceas.* (2009).
109	*Echinocactus platyacanthus*	Magnoliopsida	Negligible	-0.057	Jiménez-Sierra, C., Mandujano, M. C., & Eguiarte, L. E. Are populations of the candy barrel cactus *(Echinocactus platyacanthus)* in the desert of Tehuacán, Mexico at risk? Population projection matrix and life table response analysis. *Biological Conservation.* **135(2),** 278–292 (2007).
110	*Astrophytum ornatum*	Magnoliopsida	Negligible	-0.042	V Zepeda-Martínez, MC Mandujano, FJ Mandujano, JK Golubov. What can the demography of *Astrophytum ornatum* tell us of its endangered status? *Journal of arid environments.* **88**, 244-249 (2013).
111	*Astrophytum capricorne*	Magnoliopsida	Negligible	-0.039	Mandujano, M.C. *et al.* The population dynamics of an endemic collectible cactus. *Acta. Oceo.* **63,** 1-7 (2015).
112	*Astrophytum asterias*	Magnoliopsida	Negligible	0.018	Martinez-Avalos, PhD thesis, 2007.
113	*Opuntia macrorhiza*	Magnoliopsida	Negligible	0.002	Harida, C.V., Keeler, K.H. & Tenhumber, B. Variation in the local population dynamics of the short-lived *Opuntia macrorhiza* (Cactaceae). *Ecology.* **96(3),** 800-807 (2015).
114	*Opuntia macrocentra*	Magnoliopsida	Negligible	-0.075	Mandujano, M. C., Golubov, J., & Huenneke, L. F. Effect of reproductive modes and environmental heterogeneity in the population dynamics of a geographically widespread clonal desert cactus. *Population Ecology.* **49(2),** 141–153 (2007).
115	*Opuntia microdasys*	Magnoliopsida	Negative	-0.178	Carrillo Angeles, PhD thesis, 2011.
116	*Opuntia rastrera*	Magnoliopsida	Negative	-0.42	Mandujano, M. C., Montan~a, C., Franco, M., Golubov, J., & Flores-Martínez, A. Integration of demographic anual variability in a clonal desert cactus. *Ecology.* **82(2),** 344–359 (2001).
117	*Silene tatarica*	Magnoliopsida	Negligible	-0.066	Jäkäläniemi, A., Tuomi, J. & Siikamäki, P. Conservation of Species in Dynamic Landscapes: Divergent Fates of *Silene tatarica* Populations in Riparian Habitats. *Cons. Biol.* **20(3),** 844-852 (2006).
118	*Silene Ciliata*	Magnoliopsida	Negligible	-0.039	Giménez-Benavides, L. *et al.* Demographic processes of upward range contraction in a long-lived Mediterranean high mountain plant. *Ecography.* **34(1),** 85-93 (2011).
119	*Silene acaulis*	Magnoliopsida	Negligible	0.001	Morris, W. & Doak, D. Life history of the long-lived gynodioecious cushion plant *Silene acaulis* (Caryophyllaceae), inferred from size-based population projection matrices. *Am J Bot.* 85(6): 784 (1998).
120	*Silene douglasii*	Magnoliopsida	Negligible	0.015	Kephart, S.R. & Paladino, C. Demographic change and microhabitat Variability in a Grassland Endemic, *Silene Douglasii* Var. *oraria* (Caryophyllaceae). *Am. J. Bot.* **84(2),** 179-189 (1997).
121	*Silene regia*	Magnoliopsida	Negligible	-0.085	Menges, E.S. & Dolan, R.W. Demographic viability of populations of *Silene regia* in midwestern prairies: relationships with fire management, genetic variation, geographic location, population size and isolation. *J. Ecol.* **86(1),** 63-78 (2002).

122	*Silene glaucifolia*	Magnoliopsida	Negative	-0.139	García, M.B., Guzmán, D. & Goñi, D. An evaluation of the status of five threatened plant species in the Pyrenees. *Biol. Cons.* 103(2), 151-161 (2002).
123	*Saponaria bellidifolia*	Magnoliopsida	Negative	-0.177	Csergö, A-M., Molnár, E. & García, M.B. Dynamics of isolated *Saponaria bellidifolia* Sm. populations at northern range periphery**.** *Pop. Ecol.* **53(2),** 393-403 (2010).
124	*Arenaria grandiflora*	Magnoliopsida	Negligible	-0.065	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
125	*Atriplex acanthocarpa*	Magnoliopsida	Negligible	-0.004	Verhulst, J., Montaña, C., Mandujano, M. C., & Franco, M. Demographic mechanisms in the coexistence of two closely related perennials in a fluctuating environment. *Oecologia.* **156(1),** 95–105 (2008).

126	*Atriplex canescens*	Magnoliopsida	Negative	-0.180	Verhulst, J., Montaña, C., Mandujano, M. C., & Franco, M. Demographic mechanisms in the coexistence of two closely related perennials in a fluctuating environment. *Oecologia.* **156(1),** 95–105 (2008).
127	*Rumex rupestris*	Magnoliopsida	Negligible	-0.014	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
128	*Polygonella basiramia*	Magnoliopsida	Negligible	0.020	Witt, PhD Thesis, 2004.
129	*Eriogonum longifolium*	Magnoliopsida	Negligible	-0.030	Satterthwaite, W.H., Menges, E.S. & Quintana-Ascencio, P.F. Assessing scrub barkwheat population viability in relation to fire using multiple modeling techniques. *Ecol. Appl.* **12(6),** 1672-1687 (2002).
130	*Limonium malacitanum*	Magnoliopsida	Negligible	-0.046	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
131	*Limonium geronense*	Magnoliopsida	Negligible	-0.015	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
132	*Limonium erectum*	Magnoliopsida	Negligible	-0.020	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
133	*Limonium carolinianum*	Magnoliopsida	Negative	-0.156	Baltzer, J. L., Reekie, E. G., Hewlin, H. L., Taylor, P. D., & Boates, J. S. Impact of flower harvesting on the salt marsh plant *Limonium carolinianum*. *Canadian Journal of Botany.* **80(8),** 841–851 (2002).
134	*Armeria merinoi*	Magnoliopsida	Negligible	0.085	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
135	*Armeria maritima*	Magnoliopsida	Negligible	-0.038	Lefebvre, C., & Chandler-Mortimer, A. Demographic Characteristics of the Perennial Herb *Armeria maritima* on Zilc Lead Mine Wastes. *The Journal of Applied Ecology.* **21(1),** 255 (1984).
136	*Kunkeliella subsucculenta*	Magnoliopsida	Negligible	0.003	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
137	*Lathyrus vernus*	Magnoliopsida	Negligible	-0.029	**Erhlen, J.** Demography of the Perennial Herb *Lathyrus Vernus. II*. Herbivory and Population Dynamics. *J. Ecol.* **83(2),** 297-308 (1995).
138	*Astragalus tremolsianus*	Magnoliopsida	Negligible	-0.077	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
139	*Astragalus michauxii*	Magnoliopsida	Negligible	-0.032	Wall, W.A. et al. Demographic effects of fire on two endemic plant species in the longleaf pine-wiregrass ecosystem. *Plant. Ecol.* **213(7),** 1093-1104 (2012).
140	*Astragalus cottonii*	Magnoliopsida	Negligible	0.027	Kaye, MsC thesis, 1990.
141	*Astragalus tyghensis*	Magnoliopsida	Negligible	-0.084	Kaye, T.N. & Pyke, D.A. The effect of stochastic technique on estimates of population viability from transition matrix models. *Ecology.* **84(6)**, 1464-1476 (2003).
142	*Astragalus peckii*	Magnoliopsida	Negligible	-0.011	Martin, E. F., & Meinke, R. J. Variation in the demographics of a rare central Oregon endemic, *Astragalus peckii* Piper (Fabaceae), with fluctuating levels of herbivory. *Population Ecology*. **54(3),** 381–390 (2012).
143	*Oxytropis jabalambrensis*	Magnoliopsida	Negligible	0.013	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
144	*Anthyllis vulneraria*	Magnoliopsida	Negligible	0.021	Bastrensa, B., Lebreton, J-D. & Thompson, J.D. Predicting Demographic Change in Response to Herbivory: A Model of the

					Effects of Grazing and Annual Variation on the Population Dynamics of *Anthyllis Vulneraria. J. Ecol.* **83(4),** 603-611 (1995).
145	*Psoralea esculenta*	Magnoliopsida	Negative	-0.130	Castle, PhD thesis, 2006.
146	*Lespedeza juncea*	Magnoliopsida	Negligible	-0.014	Schutzenhofer, M.R. & Knight, T.M. Population-level effects of augmented herbivory on *Lespedeza cuneate:* Implications for biological control. *Ecol. Appl.* **17(4),** 965-971 (2007).
147	*Lespedeza virginica*	Magnoliopsida	Negligible	-0.045	Schutzenhofer, M.R., Valone, T.J. & Knight, T.M. Herbivory and population dynamics of invasive and native *Lespedeza*. *Oceo.* **161(1),** 57-66 (2009).
148	*Phaseolus lunatus*	Magnoliopsida	Negligible	-0.054	Degreef, J., Baudoin, J-P. & Rocha, O.J. Case studies on breeding systems and its consequences for germplasm conservation. *Gen. Res. Crop. Evo.* **44(5),** 429-438 (1997).
149	*Lupinus tidestromii*	Magnoliopsida	Negligible	-0.016	Dangremond, E.M., Pardini, E.A. & Knight, T.M. Apparent competition with an invasive plant hastens the extinction of an endangered lupine. *Ecol.* **91(8),** 2261-2271 (2010).
150	*Lupinus lepidus*	Magnoliopsida	Negligible	0.005	Bishop, PhD Thesis, 1996.
151	*Lupinus arboreus*	Magnoliopsida	Negligible	0.011	Kauffman, M.J & Maron, J.L. Consumers Limit the Abundance and Dynamics of a Perennial Shrub with a Seed Bank. *Am. Nat.* **168(4),** 454-470 (2006).
152	*Ulex gallii*	Magnoliopsida	Negligible	0.007	Stokes, K.E., Bullcok, J.M. & Watkinson, A.R. Population dynamics across a parapatric range boundary: *Ulex gallii* and *Ulex minor. J. Ecol.* **92(1),** 142-155 (2004).
153	*Ulex minor*	Magnoliopsida	Negligible	0.012	Stokes, K.E., Bullcok, J.M. & Watkinson, A.R. Population dynamics across a parapatric range boundary: *Ulex gallii* and *Ulex minor. J. Ecol.* **92(1),** 142-155 (2004).
154	*Adenocarpus aureus*	Magnoliopsida	Negligible	-0.008	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
155	*Cytisus scoparius*	Magnoliopsida	Negligible	-0.120	Neubert, M. G., & Parker, I. M. Projecting Rates of Spread for Invasive Species. *Risk Analysis.* **24(4),** 817–831 (2004).
156	*Adesmia volckmannii*	Magnoliopsida	Positive	0.131	Cipriotti, P.A. & Aguiar, M.R. Direct and indirect effects of grazing constrain shrub encroachment in semi-arid Patagonian steppes. *Appl. Veg. Sci.* **15(1),** 35-47 (2011).
157	*Pterocarpus angolensis*	Magnoliopsida	Negative	-0.194	Desmet, P.G., Shackleton, C.M. & Robinson, E.R. The population dynamics and life-history attributes of a *Pterocarpus angolensis* population in the Northern Province, South Africa. South African *Journal of Botany*. **62(3**) 160-166 (1996).
158	*Acacia bilimekii*	Magnoliopsida	Negative	-0.247	Jimenez-Lobato, V. & Valverde, T. Population dynamics of the shrub *Acacia bilimekii* in a semi-desert region in central Mexico. *J. Arid. Enviro.* **65(1),** 29-45 (2006).
159	*Prosopis laevigata*	Magnoliopsida	Negative	-0.153	Bernal, PhD thesis, 2004.
160	*Pentaclethra macroloba*	Magnoliopsida	Negligible	0.020	Hartshorn, PhD thesis, 1972.
161	*Parkinsonia aculeata*	Magnoliopsida	Negative	-0.251	Raghu, S., Wilson, J. R., & Dhileepan, K. Refining the process of agent selection through understanding plant demography and plant response to herbivory. *Australian*

					Journal of Entomology. **45(4),** 308–316 (2006).
162	*Tachigali vasquezii*	Magnoliopsida	Negligible	0.007	Poorter, L. *et al.* A monocarpic tree species in a polycarpic world: how can *Tachigali vasquezii*maintain itself so successfully in a tropical rain forest community? *J. Ecol.* **93(2),** 268-278 (2005).
163	*Chamaecrista lineata*	Magnoliopsida	Negligible	-0.07	Liu, H., Menges, E.S. & Quintana-Ascencio, P.F. Population viability analyses of *Chamaecrista keyensis*: effects of fire season and frequency. *Ecol. Appl.* **15(1),** 210-221 (2005).
164	*Cassia nemophila*	Magnoliopsida	Negligible	0.078	Silander, J. A. Demographic variation in the Australian desert cassia under grazing pressure. *Oecologia.* **60(2),** 227–233 (1983).
165	*Vouacapoua americana*	Magnoliopsida	Negligible	-0.073	Chagneau, P., Mortier, F. & Picard, N. Designing permanent sample plots by using a spatially hierarchical matrix population model. *J.R.Stat. Soc. C.* **58(3),** 345-367 (2009).
166	*Dicorynia guianensis*	Magnoliopsida	Negligible	0.050	Picard, N., Ouédraogo, D., & Bar-Hen, A. Choosing classes for size projection matrix models. *Ecological Modelling.* **221(19),** 2270–2279 (2010).
167	*Tetraberlinia bifoliolata*	Magnoliopsida	Negligible	0.024	Norghauer, J. M., & Newbery, D. M. Seed fate and seedling dynamics after masting in two African rain forest trees. *Ecological Monographs***, 81(3),** 443–469 (2011).
168	*Dicymbe altsonii*	Magnoliopsida	Negative	-0.178	Zagt;Boot PhD thesis, 1997.
169	*Eperua falcata*	Magnoliopsida	Negligible	0.073	Chagneau, P., Mortier, F., & Picard, N. (2009). Designing permanent sample plots by using a spatially hierarchical matrix population model. *Journal of the Royal Statistical Society: Series C (Applied Statistics).* **58(3),** 345–367 (2009).
170	*Horkelia congesta*	Magnoliopsida	Negligible	-0.030	Kaye; Benfield, Report, 2004.
171	*Agrimonia eupatoria*	Magnoliopsida	Negative	-0.154	Kiviniemi, K. Population dynamics of *Agrimonia eupatoria* and *Geum rivale*, two perennial grassland species. *Plant. Ecol.* **159,** 153-159 (2002).
172	*Rosa multiflora*	Magnoliopsida	Negligible	-0.032	Burns, J.H. *et al.* Greater sexual reproduction contributes to differences in demography of invasive plants and their noninvasive relatives. *Ecology.* **94(5),** 995-1004 (2013).
173	*Geum reptans*	Magnoliopsida	Negligible	-0.087	Weppler, T., Stoll, P., & Stocklin, J. The relative importance of sexual and clonal reproduction for population growth in the long-lived alpine plant *Geum reptans*. *Journal of Ecology*. **94(4),** 869–879 (2006).
174	*Geum rivale*	Magnoliopsida	Negligible	-0.056	Kiviniemi, K. Population dynamics of *Agrimonia eupatoria* and *Geum rivale*, two perennial grassland species. *Plant. Ecol.* **159,** 153-159 (2002).
175	*Purshia subintegra*	Magnoliopsida	Negligible	-0.100	Maschinski, J., Baggs, J. E., Quintana-Ascencio, P. F., & Menges, E. S. Using Population Viability Analysis to Predict the Effects of Climate Change on the Extinction Risk of an Endangered Limestone Endemic Shrub, Arizona Cliffrose. *Conservation Biology.* **20(1),** 218–228. (2006).
176	*Prunus africana*	Magnoliopsida	Negligible	-0.016	Stewart, PhD thesis, 2001.

177	*Brosimum alicastrum*	Magnoliopsida	Negligible	-0.077	Peters, PhD thesis, 1989.
178	*Cecropia obtusifolia*	Magnoliopsida	Negative	-0.152	Alvarez-Buylla, E. R. Density Dependence and Patch Dynamics in Tropical Rain Forests: Matrix Models and Applications to a Tree Species. *Am.Nat.* **143(1)**, 155–191 (1994).
179	*Paliurus ramosissimus*	Magnoliopsida	Negligible	0.026	Ishihama, F., Fujii, S., Yamamoto, K., & Takada, T. Estimation of dieback process caused by herbivory in an endangered root-sprouting shrub species, *Paliurus ramosissimus (Lour.) Poir.*, using a shoot-dynamics matrix model. Population *Ecology.* **56(2),** 275–288 (2013).
180	*Alnus incana*	Magnoliopsida	Negligible	0.063	Heunekke, L.F. & Marks, P.L. Stem Dynamics of the Shrub *Alnus Incana* SSP. Rugosa: Transition Matrix Models. *Ecology.* **68(5),** 1234-1242 (1987).
181	*Quercus mongolica*	Magnoliopsida	Negligible	-0.076	Hiura, T. & Fujiwara, K. Density-dependence and co-existence of conifer and broad-leaved trees in a Japanese northern mixed forest. *J. Veg. Sci.* **10(6),** 843-850 (1999).
182	*Quercus rugosa*	Magnoliopsida	Negative	-0.136	Bonil; Valverde (unpublished).
183	*Castanea dentata*	Magnoliopsida	Negative	-0.272	Davelos, A. L., & Jarosz, A. M. Demography of American chestnut populations: effects of a pathogen and a hyperparasite. *Journal of Ecology.* **92(4),** 675–685 (2004).
184	*Fagus grandifolia*	Magnoliopsida	Negligible	0.058	Batista, W. B., Platt, W. J., & Macchiavelli, R. E. Demography of a Shade-Tolerant Tree (*Fagus grandifolia*) in a Hurricane-Disturbed Forest. *Ecology.* **79(1),** 38 (1998).
185	*Nothofagus fusca*	Magnoliopsida	Negative	-0.313	Enright, N. & Ogden, J. Applications of transition matrix models in forest dynamics: *Araucaria* in Papua New Guinea and *Nothofagus* in New Zealand. *Aus. J. Ecol.* **4(1),** 3-23 (1979).
186	*Viola pumila*	Magnoliopsida	Negligible	-0.041	Eckstein, R., Danihelka, J., & Otte, A. Variation in life-cycle between three rare and endangered floodplain violets in two regions: implications for population viability and conservation. *Biologia.* **64(1).** (2009).
187	*Viola elatior*	Magnoliopsida	Negligible	0.006	Eckstein, R.L. & Otte, A. Evidence for consistent trait-habitat relations in two closely related violets of contiguous habitat types from a fertilisation experiment. *Flora.* **199(3),** 234-246 (2004).
188	*Viola sagittata*	Magnoliopsida	Negligible	-0.013	Solbrig, O.T., Sarandon, R. & Bossert, W. A Density-Dependent Growth Model of a Perennial Herb, *Viola fimbriatula*. *Am. Nat.* **131(3),** 385-400 (1988).
189	*Phyllanthus indofischeri*	Magnoliopsida	Negative	-0.162	Ticktin, T., Ganesan, R., Paramesha, M., & Setty, S. Disentangling the effects of multiple anthropogenic drivers on the decline of two tropical dry forest trees. *Journal of Applied Ecology.* **49(4),** 774–784 (2012).
190	*Phyllanthus emblica*	Magnoliopsida	Negligible	-0.07	Ellis, M. M. *et al*. Matrix population models from 20 studies of perennial plant populations. *Ecology.* **93(4**), 951–951 (2012).
191	*Rhizophora mangle*	Magnoliopsida	Negative	-0.243	Lopez Hoffman, L., Ackerly, D. D., Anten, N. P. R., Denoyer, J. L., & Martinez-Ramos, M. (2007). Gap-dependence in mangrove life-history strategies: a consideration of the entire life cycle and patch dynamics. *Journal of Ecology.* **95(6),** 1222–1233 (2007).

192	*Euphorbia fontqueriana*	Magnoliopsida	Negligible	-0.04	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
193	*Sapium sebiferum*	Magnoliopsida	Negative	-0.169	Renne, PhD thesis, 2001.
194	*Linum tenuifolium*	Magnoliopsida	Negligible	-0.068	Münzbergová, Z. Comparative demography of two co-occurring *Linum* species with different distribution patterns. *Plant Biology*. **15(6),** 963–970 (2013).
195	*Oxalis acetosella*	Magnoliopsida	Negligible	-0.008	Berg, H. Population dynamics in *Oxalis acetosella*: the significance of sexual reproduction in a clonal, cleistogamous forest herb. *Ecography*. **25(2),** 233–243 (2002).
196	*Guaiacum sanctum*	Magnoliopsida	Negligible	0.005	CITES, Plants Committee, 2008.
197	*Brassica insularis*	Magnoliopsida	Negligible	-0.007	Noël, N. et al. Interaction of climate, demography and genetics: a ten-year study of *Brassica insularis*, a narrow endemic Mediterranean species. *Cons. Gen.* **11(2),** 509-526 (2010).
198	*Vella pseudocytisus*	Magnoliopsida	Negligible	-0.084	Lozano, F.D., Moreno Saiz, J.C. & Schwartz, M.W. Demographic modeling and monitoring cycle in a long-lived endangered shrub. *J. Nat. Cons.* **19(6),** 330-338 (2011).
199	*Cochlearia bavarica*	Magnoliopsida	Negligible	0.022	Abs, C. Differences in the life histories of two *Cochlearia* species. *Folia Geobotanica*, **34(1),** 33–45 (1999).
200	*Cochlearia pyrenaica*	Magnoliopsida	Negligible	0.074	Abs, C. Differences in the life histories of two *Cochlearia* species. *Folia Geobotanica*, **34(1),** 33–45. (1999).
201	*Draba asterophora*	Magnoliopsida	Negligible	-0.041	Putnam, PhD Thesis, 2013.
202	*Braya fernaldii*	Magnoliopsida	Negligible	0.001	Squires, PhD Thesis, 2010.
203	*Braya longii*	Magnoliopsida	Negligible	-0.002	Squires, PhD Thesis, 2010.
204	*Boechera fecunda*	Magnoliopsida	Negligible	-0.043	Lesica, P., & Shelly, J. S. Effects of reproductive mode on demography and life history in *Arabis fecunda* (Brassicaceae). *American Journal of Botany*. **82(6),** 752–762 (1995).
205	*Lepidium davisii*	Magnoliopsida	Negligible	-0.069	Bernatus, Report, 1995.
206	*Parolinia glabriuscula*	Magnoliopsida	Negligible	-0.022	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
207	*Kosteletzkya pentacarpos*	Magnoliopsida	Negative	-0.338	Pino, J., Pico, F. X., & De Roa, E. Population dynamics of the rare plant *Kosteletzkya pentacarpos* (Malvaceae): a nine-year study. *Botanical Journal of the Linnean Society*. **153(4),** 455–462 (2007).
208	*Scaphium macropodum*	Magnoliopsida	Negligible	-0.030	Yamada, T., Yamada, Y., Okuda, T., & Fletcher, C. Soil-related variations in the population dynamics of six dipterocarp tree species with strong habitat preferences. *Oecologia*. **172(3),** 713–724 (2012).
209	*Shorea maxwelliana*	Magnoliopsida	Negligible	-0.001	Yamada, T., Yamada, Y., Okuda, T., & Fletcher, C. Soil-related variations in the population dynamics of six dipterocarp tree species with strong habitat preferences. *Oecologia*. **172(3),** 713–724 (2012).
210	*Shorea acuminata*	Magnoliopsida	Negligible	-0.018	Yamada, T., Yamada, Y., Okuda, T., & Fletcher, C. Soil-related variations in the population dynamics of six dipterocarp tree species with strong habitat preferences. *Oecologia*. **172(3),** 713–724 (2012).

211	*Shorea leprosula*	Magnoliopsida	Negligible	-0.024	Yamada, T., Yamada, Y., Okuda, T., & Fletcher, C. Soil-related variations in the population dynamics of six dipterocarp tree species with strong habitat preferences. *Oecologia*. **172(3),** 713–724 (2012).
212	*Shorea ovalis*	Magnoliopsida	Negligible	-0.005	Yamada, T., Yamada, Y., Okuda, T., & Fletcher, C. Soil-related variations in the population dynamics of six dipterocarp tree species with strong habitat preferences. *Oecologia*. **172(3),** 713–724. (2012).
213	*Shorea bracteolata*	Magnoliopsida	Negligible	-0.005	Yamada, T., Yamada, Y., Okuda, T., & Fletcher, C. Soil-related variations in the population dynamics of six dipterocarp tree species with strong habitat preferences. *Oecologia*. **172(3),** 713–724 (2012).
214	*Parashorea chinensis*	Magnoliopsida	Negative	-0.139	Chien, P. D., Zuidema, P. A., & Nghia, N. H. Conservation prospects for threatened Vietnamese tree species: results from a demographic study. *Population Ecology*. **50(2),** 227–237 (2008).
215	*Vatica mangachapoi*	Magnoliopsida	Negligible	-0.049	Hu; Wang. *Acta Ecol Sin*. (1988).
216	*Fumana procumbens*	Magnoliopsida	Negligible	0.034	Bengtsson, K. *Fumana Procumbens* on Oland--Population Dynamics of a Disjunct Species at the Northern Limit of its Range. *The Journal of Ecology*. **81(4),** 745 (1993).
217	*Lechea cernua*	Magnoliopsida	Negligible	-0.018	Witt, PhD Thesis, 2004.
218	*Lechea deckertii*	Magnoliopsida	Negligible	-0.107	Witt, PhD Thesis, 2004.
219	*Helianthemum juliae*	Magnoliopsida	Negligible	-0.053	Marrero-Gómez, M. V., Oostermeijer, J. G. B., Carqué-Álamo, E., & Bañares-Baudet, Á. Population viability of the narrow endemic *Helianthemum juliae* (CISTACEAE) in relation to climate variability. *Biological Conservation*. **136(4),** 552–562 (2007).
220	*Helianthemum teneriffae*	Magnoliopsida	Negligible	0.020	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
221	*Helianthemum polygonoides*	Magnoliopsida	Negligible	-0.079	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
222	*Daphne rodriguezii*	Magnoliopsida	Negligible	-0.079	Rodríguez-Pérez, J., & Traveset, A. Demographic consequences for a threatened plant after the loss of its only disperser. Habitat suitability buffers limited seed dispersal. *Oikos.* **121(6),** 835–847 (2011).

223	*Aquilaria microcarpa*	Magnoliopsida	Negative	-0.396	Soehartono, T., & C. Newton, A. Conservation and sustainable use of tropical trees in the genus *Aquilaria* II. The impact of gaharu harvesting in Indonesia. *Biological Conservation.* **97(1),** 29–41 (2001).
224	*Aquilaria malaccensis*	Magnoliopsida	Negligible	-0.026	Soehartono, T., & C. Newton, A. Conservation and sustainable use of tropical trees in the genus *Aquilaria* II. The impact of gaharu harvesting in Indonesia. *Biological Conservation.* **97(1),** 29–41 (2001).
225	*Aquilaria crassna*	Magnoliopsida	Negligible	-0.009	Zhang, L., Brockelman, W. Y., & Allen, M. A. Matrix analysis to evaluate sustainability: The tropical tree *Aquilaria crassna*, a heavily poached source of agarwood. *Biological Conservation.* **141(6),** 1676–1686 (2008).
226	*Khaya senegalensis*	Magnoliopsida	Negligible	-0.095	Gaoue, O. G., & Ticktin, T. Effects of Harvest of Nontimber Forest Products and Ecological Differences between Sites on the Demography of African Mahogany. *Conservation Biology.* **24(2),** 605–614 (2010).
227	*Swietenia macrophylla*	Magnoliopsida	Negative	-0.352	Verwer, C., Peña-Claros, M., van der Staak, D., Ohlson-Kiehn, K., & Sterck, F. J. (2008). Silviculture enhances the recovery of overexploited mahogany *Swietenia macrophylla*. *Journal of Applied Ecology*, **45(6),** 1770–1779 (2008).
228	*Entandrophragma cylindricum*	Magnoliopsida	Negligible	0.056	Picard, N., Yalibanda, Y., Namkosserana, S. & Baya, F. Estimating the stock recovery rate using matrix models. *Forest. Ecol. Manage.* **255(10),** 3597-3605 (2008).
229	*Ailanthus altissima*	Magnoliopsida	Negligible	0.002	Burns, J. H. *et al.* Greater sexual reproduction contributes to differences in demography of invasive plants and their non-invasive relatives. *Ecology.* **94(5),** 995–1004 (2013).
230	*Acer palmatum*	Magnoliopsida	Negative	-0.339	Tanaka, H. *et al.* Comparative demography of three coexisting Acer species in gaps and under closed canopy. *Journal of Vegetation Science.* **19(1),** 127–138 (2008).
231	*Acer rufinerve*	Magnoliopsida	Negligible	-0.082	Tanaka, H. *et al.* Comparative demography of three coexisting Acer species in gaps and under closed canopy. *Journal of Vegetation Science.* **19(1),** 127–138 (2008).
232	*Acer pictum*	Magnoliopsida	Negative	-0.161	Tanaka, H. *et al.* Comparative demography of three coexisting Acer species in gaps and under closed canopy. *Journal of Vegetation Science.* **19(1),** 127–138 (2008).
233	*Acer saccharum*	Magnoliopsida	Negligible	-0.027	Lin, Y. & Augspurger, C.K. Impact of spatial heterogeneity of neighborhoods on long-term population dynamics of sugar maple (Acer saccharum). *Plant. Biol.* **255,** 3589-3596 (2008).
234	*Aesculus turbinata*	Magnoliopsida	Negative	-0.356	Kaneko, Y., Takada, T. & Kawana, S. Population biology of *Aesculus turbinate:* A demographic analysis using transition matrices on a natural population along a riparian environmental gradient. *Plant Species Biology.* **14(1),** 47-68 (1999).
235	*Boswellia papyrifera*	Magnoliopsida	Negligible	-0.013	Groenendijk, P., Eshete, A., Sterck, F. J., Zuidema, P. A., & Bongers, F. Limitations to sustainable frankincense production: blocked regeneration, high adult mortality and declining populations. *Journal of Applied Ecology.* **49(1),** 164–173 (2011).

236	*Bursera glabrifolia*	Liliopsida	Negative	-0.134	Hernández-Apolinar, M., Valverde, T., & Purata, S. Demography of *Bursera glabrifolia*, a tropical tree used for folk woodcrafting in Southern Mexico: An evaluation of its management plan. *Forest Ecology and Management.* **223(1-3),** 139–151 (2006).
237	*Rhus copallinum*	Magnoliopsida	Negligible	-0.095	Thaxton, PhD thesis, 2003.
238	*Choerospondias axillaris*	Magnoliopsida	Negative	-0.384	Brodie, J. F., Helmy, O. E., Brockelman, W. Y., & Maron, J. L. Functional differences within a guild of tropical mammalian frugivores. *Ecology.* **90(3),** 688–698 (2009).
239	*Clidemia hirta*	Magnoliopsida	Negative	-0.297	DeWalt, S.J. Population Dynamics and Potential for Biological Control of an Exotic Invasive Shrub in Hawaiian Rainforests. *Biol. Inv.* **8,** 1145-1158 (2006).
240	*Verticordia fimbrilepis*	Magnoliopsida	Negligible	-0.030	Yates, C.J. & Ladd, P.G. Using population viability analysis to predict the effect of fire on the extinction risk of an endangered shrub *Verticordia fimbrilepis* subsp. *fimbrilepis* in a fragmented landscape. *Plant. Ecol.* **211,** 305-319 (2010).
241	*Verticordia staminosa*	Magnoliopsida	Negative	-0.151	Yates, C.J. Ladd, P.G., Coates, D.J. & McArthur, S. Hierarchies of cause: understanding rarity in an endemic shrub *Verticordia staminosa* (Myrtaceae) with a highly restricted distribution. *Aus. J. Bot.* **55(3),** 194-205 (2006).
242	*Syzygium jambos*	Magnoliopsida	Negative	-0.254	Brown, K. A., Spector, S., & Wu, W. Multi-scale analysis of species introductions: combining landscape and demographic models to improve management decisions about non-native species. *Journal of Applied Ecology*, **45(6)**, 1639–1648 (2008).

243	*Vochysia ferruginea*	Magnoliopsida	Negative	-0.377	Boucher, D. H., & Mallona, M. A. Recovery of the rain forest tree *Vochysia ferruginea* over 5 years following Hurricane Joan in Nicaragua: a preliminary population projection matrix. Forest *Ecology and Management*, **91(2-3),** 195–204 (1997).
244	*Oenothera deltoides*	Magnoliopsida	Negligible	-0.123	Thomson, D.M. Matrix Models as a Tool for Understanding Invasive Plant and Native Plant Interactions. *Cons. Biol.* **19(3),** 917-928 (2005).
245	*Gaura neomexicana*	Magnoliopsida	Negligible	0.058	Floyd, S.K. & Ranker, T.A. Analysis of a Transition Matrix Model for *Gaura neomexicana* Ssp. coloradensis (Onagraceae) Reveals Spatial and Temporal Demographic Variability. *I. J. Plant. Sci.* **159(5),** 853-863 (1998).

246	*Geranium sylvaticum*	Magnoliopsida	Negligible	-0.022	Ramula, S., Toivonen, E., & Mutikainen, P. Demographic Consequences of Pollen Limitation and Inbreeding Depression in a Gynodioecious Herb. *International Journal of Plant Sciences.* **168(4),** 443–453 (2007).
247	*Erodium paularense*	Magnoliopsida	Negligible	-0.092	Iriondo; Albert; Gimemez; Lozana; Escuerdo 978-84-8014-746-0
248	*Saxifraga aizoides*	Magnoliopsida	Negative	-0.428	Marcante, S., Winkler, E., & Erschbamer, B. Population dynamics along a primary succession gradient: do alpine species fit into demographic succession theory? *Annals of Botany.* **103(7),** 1129–1143 (2009).
249	*Saxifraga cotyledon*	Magnoliopsida	Negligible	-0.046	Dinnétz, P., & Nilsson, T. Population viability analysis of *Saxifraga cotyledon*, a perennial plant with semelparous rosettes. *Plant Ecology.* **159(1)**, 61–71 (2002).
250	*Paeonia officinalis*	Magnoliopsida	Negative	-0.15	Andrieu, E. *et al.* Forest-cutting rapidly improves the demographic status of *Paeonia officinalis*, a species threatened by forest closure. *Pop. Ecol.* **55(1),** 147-158 (2012).
251	*Banksia ericifolia*	Magnoliopsida	Negligible	0.000	Bradstock, R. A., & O'Connell, M. A. (1988). Demography of woody plants in relation to fire: *Banksia ericifolia* L.f. and *Petrophile pulchella* (Schrad) R.Br. *Austral Ecology*. **13(4),** 505–518 (1988).
252	*Roupala montana*	Magnoliopsida	Negative	-0.417	Hoffmann, W.A. Fire and Population Dynamics of Woody Plants in a Neotropical Savanna: Matrix Model Projections. *Ecology.* **80(4),** 1360-1369 (1999).
253	*Petrophile pulchella*	Magnoliopsida	Negligible	0.000	Bradstock, R. A., & O'Connell, M. A. (1988). Demography of woody plants in relation to fire: *Banksia ericifolia* L.f. and *Petrophile pulchella* (Schrad) R.Br. *Austral Ecology*. **13(4),** 505–518 (1988).
254	*Persoonia glaucescens*	Magnoliopsida	Negligible	0.003	McKenna, PhD thesis, 2007.
255	*Persoonia bargoensis*	Magnoliopsida	Negative	-0.18	McKenna, PhD thesis, 2007.
256	*Ranunculus peltatus*	Magnoliopsida	Negligible	0.024	Idestam-Almquist, PhD Thesis, 1998.
257	*Anemone patens*	Magnoliopsida	Negligible	0.063	Williams, J.L. & Crone, E.E. The impact of invasive grasses on the population growth of *Aneone patens*, a long-lived native forb. *Ecology*. **87 (12)** 3200-3208 (2006).

258	*Trollius laxus*	Magnoliopsida	Negligible	-0.105	Scanga, S. E. Population dynamics in canopy gaps: nonlinear response to variable light regimes by an understory plant. *Plant Ecology.* **215(8),** 927–935 (2014).
259	*Actaea spicata*	Magnoliopsida	Negligible	-0.048	Fröborg, H., & Eriksson, O. Predispersal seed predation and population dynamics in the perennial understorey herb *Actaea spicata*. *Canadian Journal of Botany*, **81(11),** 1058–1069 (2003).
260	*Actaea cordifolia*	Magnoliopsida	Negligible	0.031	Cook, PhD Thesis, 1993.
261	*Actaea elata*	Magnoliopsida	Negligible	-0.024	Kaye, T.N. & Pyke, D.A. The effect of stochastic technique on estimates of population viability from transition matrix models. *Ecology.* 84(6) 1464-1476 (2003).
262	*Aquilegia chrysantha*	Magnoliopsida	Negligible	-0.024	Stubben, PhD Thesis, 2007.
263	*Hydrastis canadensis*	Magnoliopsida	Negligible	0.103	Sinclair, PhD Thesis, 2002.
264	*Sarcocapnos pulcherrima*	Magnoliopsida	Negative	-0.200	Salinas, M. J., Suárez, V., & Blanca, G. Demographic structure of three species of *Sarcocapnos (Fumariaceae*) as a basis for their conservation. *Canadian Journal of Botany.* **80(4),** 360–369 (2002).
265	*Sarcocapnos baetica*	Magnoliopsida	Negative	-0.298	Salinas, M. J., Suárez, V., & Blanca, G. Demographic structure of three species of *Sarcocapnos (Fumariaceae*) as a basis for their conservation. *Canadian Journal of Botany.* **80(4),** 360–369 (2002).
266	*Dicentra canadensis*	Magnoliopsida	Negligible	-0.051	Lin, C.-H., Miriti, M. N., & Goodell, K. Demographic consequences of greater clonal than sexual reproduction in *Dicentra canadensis.* Ecology and *Evolution.* **6(12),** 3871–3883 (2016).
267	*Tolumnia varigata*	Liliopsida	Negligible	-0.044	Calvo, R.N. Evolutionary Demograhy of Orchids: Intensity and Frequency of Pollination and the Cost of Fruiting. *Ecology.* **74(4),** 1033-1042 (1993).

268	*Aspasia principissa*	Liliopsida	Negligible	-0.093	Zotz, G., & Schmidt, G. Population decline in the epiphytic orchid *Aspasia principissa*. *Biological Conservation*, **129(1),** 82–90 (2006).
269	*Oncidium poikilostalix*	Liliopsida	Negligible	-0.037	Raventós, J. *et al*. Comparison of transient and asymptotic perturbation analyses of three epiphytic orchid species growing in coffee plantations in Mexico: effect on conservation decisions. *Plant. Ecol. Div.* **11(2),** 133-145 (2018).
270	*Telipogon helleri*	Liliopsida	Negligible	-0.103	Raventós, J. *et al*. Comparison of transient and asymptotic perturbation analyses of three epiphytic orchid species growing in coffee plantations in Mexico: effect on conservation decisions. *Plant. Ecol. Div.* 11(2), 133-145 (2018).
271	*Lycaste aromatica*	Liliopsida	Negligible	-0.118	Winkler, M., Hülber, K., & Hietz, P. Population dynamics of epiphytic orchids in a metapopulation context. *Annals of Botany*, **104(5),** 995–1004 (2009).
272	*Dendrophylax lindenii*	Liliopsida	Negligible	-0.077	Raventos J., Gonzalez, E., Mujica, E., & Doak, D. F. Population Viability Analysis of the Epiphytic Ghost Orchid (*Dendrophylax lindenii*) in Cuba. *Biotropica.* **47(2),** 179-189 (2015).
273	*Lepanthes acuminata*	Liliopsida	Negligible	-0.069	Raventós, J. *et al*. Comparison of transient and asymptotic perturbation analyses of three epiphytic orchid species growing in coffee plantations in Mexico: effect on conservation decisions. *Plant. Ecol. Div.* 11(2), 133-145 (2018).
274	*Lepanthes rupestris*	Liliopsida	Negative	-0.151	Tremblay, R. L., & Ackerman J. D. Gene flow and effective population size in *Lepanthes (Orchidaceae*): a case for genetic drift. *Biological Journal of the Linnean Society.* **72(1),** 47–62 (2001).
275	*Lepanthes rubripetala*	Liliopsida	Negligible	-0.118	Schödelbauerová I., Tremblay, R. L., & Kindlmann, P. Prediction vs. reality: Can a PVA model predict population persistence 13 years later? *Biodiversity and Conservation.* **19(3)**, 637-650 (2009).
276	*Lepanthes eltoroensis*	Liliopsida	Negative	-0.218	Tremblay, R.L. & Ackerman, J.D. Gene flow and effective population size in *Lepanthes (Orchidaceae*): a case for genetic drift. *Biological Journal of the Linnean Society*, **72(1),** 47–62 (2001).
277	*Guarianthe aurantiaca*	Liliopsida	Negligible	-0.081	Mondragón, N. Population viability analysis for *Guarianthe aurantiaca*, an ornamental epiphytic orchid harvested in Southeast Mexico. *Plant. Spp. Biol.* **24(1),** 35-41 (2009).
278	*Jacquiniella leucomelana*	Liliopsida	Negligible	0.02	Winkler, M., Hülber, K., & Hietz, P. Population dynamics of epiphytic orchids in a metapopulation context. *Annals of Botany*, **104(5),** 995–1004 (2009).
279	*Jacquiniella teretifolia*	Liliopsida	Negligible	-0.026	Winkler, M., Hülber, K., & Hietz, P. Population dynamics of epiphytic orchids in a metapopulation context. *Annals of Botany*, **104(5),** 995–1004 (2009).
280	*Spathoglottis plicata*	Liliopsida	Negative	-0.157	Falcón, W., Ackerman, J.D. & Tremblay, R.L. Quantifying how acquired interactions with native and invasive insects influence

					population growth rates of a non-indigenous plant. *Bio. Inv.* **19(3),** 895-911 (2017).
281	*Epipactis atrorubens*	Liliopsida	Negligible	0.012	Hens, H. *et al.* Low population viability in small endangered orchid populations: Genetic variation, seedling recruitment and stochasticity. *Biol. Cons.* **210(A),** 174-183 (2017).
282	*Cephalanthera longifolia*	Liliopsida	Negligible	-0.011	Shefferson, R.P. *et al.* Linking vegetative dormancy to fitness in two long-lived herbaceous perennials. *Ecosphere.* **3(2),** 1-19 (2012).
283	*Orchis purpurea*	Liliopsida	Negligible	-0.001	Jacquemyn, H., Brys, R. & Jongejans, E. Seed limitation restricts population growth in shaded populations of a perennial woodland orchid. *Ecology.* **91(1),** 119-129 (2010).
284	*Neotinea ustulata*	Liliopsida	Negligible	0.055	Shefferson, R.P. & Tali, K. Dormancy is associated with decreased adult survival in the burnt orchid, *Neotinea ustulata*. *J. Ecol.* **95(1),** 217-225 (2006).
285	*Caladenia rosella*	Liliopsida	Negligible	0.002	Tremblay, R. L. *et al.* Population dynamics of *Caladenia*: Bayesian estimates of transition and extinction probabilities. *Australian Journal of Botany.* **57(4),** 351 (2009).
286	*Caladenia oenochila*	Liliopsida	Negligible	0.030	Tremblay, R. L. *et al.* Population dynamics of *Caladenia*: Bayesian estimates of transition and extinction probabilities. *Australian Journal of Botany.* **57(4),** 351 (2009).
287	*Caladenia macroclavia*	Liliopsida	Negligible	0.049	Tremblay, R. L. *et al.* Population dynamics of *Caladenia*: Bayesian estimates of transition and extinction probabilities. *Australian Journal of Botany.* **57(4),** 351 (2009).
288	*Caladenia graniticola*	Liliopsida	Negligible	0.032	Tremblay, R. L. *et al.* Population dynamics of *Caladenia*: Bayesian estimates of transition and extinction probabilities. *Australian Journal of Botany.* **57(4),** 351 (2009).
289	*Caladenia elegans*	Liliopsida	Negligible	0.037	Tremblay, R. L. *et al.* Population dynamics of *Caladenia*: Bayesian estimates of transition and extinction probabilities. *Australian Journal of Botany.* **57(4),** 351 (2009).
290	*Caladenia argocalla*	Liliopsida	Negligible	0.055	Tremblay, R. L. *et al.* Population dynamics of *Caladenia*: Bayesian estimates of transition and extinction probabilities. *Australian Journal of Botany.* **57(4),** 351 (2009).
291	*Caladenia amonea*	Liliopsida	Negligible	0.011	Tremblay, R. L. *et al.* Population dynamics of *Caladenia*: Bayesian estimates of transition and extinction probabilities. *Australian Journal of Botany.* **57(4),** 351 (2009).
292	*Caladenia valida*	Liliopsida	Negligible	0.054	Tremblay, R. L. *et al.* Population dynamics of *Caladenia*: Bayesian estimates of transition and extinction probabilities. *Australian Journal of Botany.* **57(4),** 351 (2009).
293	*Caladenia clavigera*	Liliopsida	Negligible	-0.029	Tremblay, R. L. *et al.* Population dynamics of *Caladenia*: Bayesian estimates of transition and extinction probabilities.

					Australian Journal of Botany. **57(4),** 351 (2009).
294	*Cypripedium calceolus*	Liliopsida	Negative	-0.221	Nicole, F., Brzosko, E., & Till-Bottraud, I. Population viability analysis of *Cypripedium calceolus* in a protected area: longevity, stability and persistence. *Journal of Ecology*. **93(4),** 716–726 (2005).
295	*Cypripedium parviflorum*	Liliopsida	Negligible	-0.067	Shefferson, R.P., Warren, R.J. & Pulliam, H.R. Life-history costs make perfect sprouting maladaptive in two herbaceous perennials. *Journal of Ecology*. **102(5),** 1318-1328 (2014).

296	*Cypripedium fasciculatum*	Liliopsida	Negligible	-0.077	Crone, E.E. *et al*. Ability of Matrix Models to Explain the Past and Predict the Future of Plant Populations. *Conservation Biology*. **27(5),** 968-978 (2013).
297	*Anthericum liliago*	Liliopsida	Negligible	0.043	Cerná, L., & Münzbergová, Z. (2013). Comparative Population Dynamics of Two Closely Related Species Differing in Ploidy Level. *PLoS ONE*. **8(10),** e75563 (2013).
298	*Anthericum ramosum*	Liliopsida	Negligible	0.070	Cerná, L., & Münzbergová, Z. Comparative Population Dynamics of Two Closely Related Species Differing in Ploidy Level. *PLoS ONE*. **8(10),** e75563 (2013).
299	*Allium tricoccum*	Liliopsida	Negligible	-0.022	Nault, A., & Gagnon, D. Ramet Demography of *Allium Tricoccum*, A Spring Ephemeral, Perennial Forest Herb. *The Journal of Ecology*, **81(1),** 101 (1993).
300	*Narcissus pseudonarcissus*	Liliopsida	Negligible	-0.092	Barkham, J.P. Population Dynamics of the Wild Daffodil (*Narcissus Pseudonarcissus*): I. Clonal Growth, Seed Reproduction, Mortality and the Effects of Density. *J. Ecol.* **68(2),** 607-633 (1980).

301	*Hyparrhenia diplandra*	Liliopsida	Negative	-0.240	Garnier, L.K.M. & Dajoz, I. Evolutionary significance of awn length variation in a clonal grass of fire-prone savannas. *Ecology.* **82(6)**, 1720-1733 (2001).
302	*Bothriochloa insculpta*	Liliopsida	Negligible	-0.017	O'Connor, T.G. The Influence of Rainfall and Grazing on the Demography of Some African Savanna Grasses: A Matrix Modelling Approach. *J. Appl. Ecol.* **30(1),** 119-132 (1993).
303	*Zea diploperennis*	Liliopsida	Negligible	-0.029	Sanchez-Velasquez, L. R., Ezcurra, E., Martinez-Ramos, M., Alvarez-Buylla, E., & Lorente, R. Population dynamics of *Zea diploperennis*, an endangered perennial herb: effect of slash and burn practice. *Journal of Ecology.* **90(4),** 684–692 (2002).
304	*Hilaria mutica*	Liliopsida	Negative	-0.138	Vega, E., & Montaña, C. Spatio-temporal variation in the demography of a bunch grass in a patchy semiarid environment. *Plant Ecology Formerly `Vegetatio'*. **175(1),** 107–120 (2004).
305	*Sporobolus heterolepis*	Liliopsida	Negligible	-0.053	Dalgleish, H. J., Kula, A. R., Hartnett, D. C., & Sandercock, B. K. Responses of two bunchgrasses to nitrogen addition in tallgrass prairie: the role of bud bank demography. *American Journal of Botany*. **95(6),** 672–680 (2008).
306	*Danthonia sericea*	Liliopsida	Negative	-0.293	Moloney, K. A. Fine-Scale Spatial and Temporal Variation in the Demography of a Perennial Bunchgrass. *Ecology.* 69(5), 1588–1598 (1988).
307	*Molinia caerulea*	Liliopsida	Negative	-0.341	Jacquemyn, H., Brys, R. & Neubert, M.G. Fire increases invasive spread of *Molinia caerulea* mainly through changes in demographic parameters. *Ecol. Appl.* **15(6),** 2097-2108 (2005).
308	*Poa alpina*	Liliopsida	Negative	-0.134	Marcante, S., Winkler, E., & Erschbamer, B. Population dynamics along a primary succession gradient: do alpine species fit into demographic succession theory? *Annals of Botany.* **103(7),** 1129–1143 (2009).
309	*Tillandsia violacea*	Liliopsida	Negligible	0.007	Mondragón, D. & Ticktin, T. Demographic Effects of Harvesting Epiphytic Bromeliads and an Alternative Approach to Collection. *Cons. Biol.* **25(4),** 797-807 (2011).
310	*Tillandsia brachycaulos*	Liliopsida	Negligible	0.037	Mondragón, D., Durán, R., Ramírez, I. & Valverde, T. Temporal variation in the demography of the clonal epiphyte *Tillandsia brachycaulos* (Bromeliaceae) in the Yucatán Peninsula, Mexico. *J. Trop. Ecol.* **20(2),** 189-200 (2002).
311	*Tillandsia recurvata*	Liliopsida	Negligible	0.056	Valverde, T. & Rocio, B. Is there demographic asynchrony among local populations of *Tillandsia recurvata?:* Evidence of tis metapopulation functioning. *Bol. Soc. Bot. Mex*. **n.86** (2010).
312	*Tillandsia multicaulis*	Liliopsida	Negligible	-0.118	Winkler, M., Hülber, K., & Hietz, P. Population dynamics of epiphytic bromeliads: Life strategies and the role of host branches. *Basic and Applied Ecology*, **8(2),** 183–196 (2007).
313	*Tillandsia juncea*	Liliopsida	Negative	-0.155	Winkler, M., Hülber, K., & Hietz, P. Population dynamics of epiphytic bromeliads: Life strategies and the role of

					host branches. *Basic and Applied Ecology*, **8(2),** 183–196 (2007).
314	*Tillandsia macdougallii*	Liliopsida	Negligible	-0.046	Mondragon Chopparo D., & Ticktin, T. Demographic Effects of Harvesting Epiphytic Bromeliads and an Alternative Approach to Collection. *Conservation Biology*, **25(4),** 797–807 (2011).
315	*Tillandsia punctulata*	Liliopsida	Negligible	-0.010	Toledo-Aceves, T., Hernández-Apolinar, M., & Valverde, T. Potential impact of harvesting on the population dynamics of two epiphytic bromeliads. *Acta Oecologica*, **59,** 52–61 (2014).
316	*Vriesea sanguinolenta*	Liliopsida	Negligible	-0.070	Zotz, G. Differences in vital demographic rates in three populations of the epiphytic bromeliad, *Werauhia sanguinolenta*. *Acta Oecologica*, **28(3),** 306–312 (2005).
317	*Catopsis sessiliflora*	Liliopsida	Negligible	-0.071	Winkler, M., Hülber, K., & Hietz, P. Population dynamics of epiphytic bromeliads: Life strategies and the role of host branches. *Basic and Applied Ecology*, **8(2),** 183–196 (2007).
318	*Catopsis compacta*	Liliopsida	Negligible	0.014	Del Castillo, R. F., Trujillo-Argueta, S., Rivera-García, R., Gómez-Ocampo, Z., & Mondragón-Chaparro, D. Possible combined effects of climate change, deforestation, and harvesting on the epiphyte *Catopsis compacta*: a multidisciplinary approach. *Ecology and Evolution*, **3(11),** 3935–3946 (2013).
319	*Calathea micans*	Liliopsida	Negligible	0.000	Le Corff, J. & Horvitz, C.C. Population growth versus population spread of an ant-dispersed neotropical herb with a mixed reproductive strategy. *Ecol. Model.* **188(1),** 41-51 (2005).
320	*Calathea ovandensis*	Liliopsida	Negative	-0.165	Horvitz, C. C., & Schemske, D. W. Spatiotemporal Variation in Demographic Transitions of a Tropical Understory Herb: Projection Matrix Analysis. *Ecological Monographs*. **65(2),** 155–192 (1995).
321	*Heliconia metallica*	Liliopsida	Negligible	-0.008	Schleuning, M., Haumán, V. & Matthies, D. Flooding and canopy dynamics shape the demography of a clonal Amazon understorey herb. *J. Ecol.* **96(5),** 1045-1055 (2008).
322	*Heliconia acuminata*	Liliopsida	Negligible	-0.113	Bruna, E.M. Are plant populations in fragmented habitats recruitment limited? Tests with an Amazonian herb. *Ecol.* **84(4),** 932-947 (2003).
323	*Ptychosperma macarthurii*	Liliopsida	Negative	-0.232	Liddle, D.T. *et al.* Threat and response: A decade of decline in a regionally endangered rainforest palm affected by fire and introduced animals. *Biol. Cons.* **132,** 362-375 (2006).
324	*Rhopalostylis sapida*	Liliopsida	Negative	-0.132	Enright, N. J., & Watson, A. D. Population dynamics of the nikau palm, *Rhopalostylis sapida* (Wendl. et Drude), in a temperate forest remnant near Auckland, New Zealand. *New Zealand Journal of Botany.* **30(1),** 29–43 (1992).
325	*Dypsis decaryi*	Liliopsida	Negative	-0.445	Ratsirarson, J., Silander, J. A., & Richard, A. F. Conservation and Management of a Threatened Madagascar Palm Species, *Neodypsis decaryi*, Jumelle. *Conservation Biology.* **10(1),** 40–52. (1996).
326	*Euterpe edulis*	Liliopsida	Negative	-0.236	Silva Matos, D.M., Freckleton, R.P. & Watkinson, A.R. The role of density

					dependence in the population dynamics of a tropical palm. *Ecology.* **80(8),** 2635-2650 (1999).
327	*Euterpe precatoria*	Liliopsida	Negligible	-0.061	Otárola, M. F., & Avalos, G. Demographic variation across successional stages and their effects on the population dynamics of the neotropical palm *Euterpe precatoria*. *American Journal of Botany.* **101(6),** 1023–1028 (2014).
328	*Geonoma orbignyana*	Liliopsida	Negligible	0.058	Rodríguez-Buriticá, S., Orjuela, M. A., & Galeano, G. Demography and life history of *Geonoma orbignyana*: An understory palm used as foliage in Colombia. *Forest Ecology and Management.* **211(3),** 329–340 (2005).
329	*Geonoma pohliana*	Liliopsida	Negligible	-0.018	Souza, A.F. & Martins, F.R. Demography of the clonal palm *Geonoma brevispatha* in a Neotropical swamp forest. *Aust. Ecol.* **31(7),** 869-881 (2006).
330	*Podococcus barteri*	Liliopsida	Negligible	0.061	Bullock, S. H. Demography of an Undergrowth Palm in Littoral Cameroon. *Biotropica.* **12(4),** 247 (1980).
331	*Astrocaryum mexicanum*	Liliopsida	Negative	-0.331	Pinero, D., Martinez-Ramos, M., & Sarukhan, J. A Population Model of *Astrocaryum Mexicanum* and a Sensitivity Analysis of its Finite Rate of Increase. The *Journal of Ecology.* **72(3),** 977 (1984).
332	*Chamaedorea radicalis*	Liliopsida	Negligible	-0.086	Endress, B.A., Gorchov, D.L. & Noble, R.B. Non-timber forest product extraction: effects on harvest and browsing on an understory palm. *Ecol. Appl.* **14(4),** 1139-1153 (2004).
333	*Chamaedorea elegans*	Liliopsida	Negligible	0.016	Valverde, T., Hernandez-Apolinar, M., & Mendoza-Amarom, S. Effect of Leaf Harvesting on the Demography of the Tropical Palm *Chamaedorea elegansin* South-Eastern Mexico. *Journal of Sustainable Forestry.* **23(1),** 85–105 (2006).
334	*Iriartea deltoidea*	Magnoliopsida	Negligible	-0.078	Pinard, M. Impacts of Stem Harvesting on Populations of *Iriartea deltoidea* (Palmae) in an Extractive Reserve in Acre, Brazil. *Biotropica*. **25(1),** 2 (1993).
335	*Phytelephas seemannii*	Liliopsida	Negligible	-0.076	Bernal, R. Demography of the vegetable ivory palm *Phytelephas seemannii* in Colombia, and the impact of seed harvesting. *Journal of Applied Ecology.* **35(1),** 64–74 (1998).
336	*Pseudophoenix sargentii*	Liliopsida	Negative	-0.153	Maschinski, J. & Duqeusnel, J. Successful reintroduction of the endangered long-lives Sargent's Cherry Palm, *Pseudophoenix sargentii*, in the Florida Keys. *Biol. Cons.* **134(1),** 122-129 (2007).
337	*Coccothrinax readii*	Liliopsida	Negligible	0.104	Olmsted, I., & Alvarez-Buylla, E. R. Sustainable Harvesting of Tropical Trees: Demography and Matrix Models of Two Palm Species in Mexico. *Ecological Applications.* **5(2),** 484–500 (1995).
338	*Thrinax radiata*	Liliopsida	Positive	0.139	Olmsted, I., & Alvarez-Buylla, E. R. Sustainable Harvesting of Tropical Trees: Demography and Matrix Models of Two Palm Species in Mexico. *Ecological Applications.* **5(2),** 484–500 (1995).
339	*Sabal yapa*	Liliopsida	Negative	-0.145	Pulido, M. T., Valverde, T., & Caballero, J. Variation in the population dynamics of the palm *Sabal yapa* in a landscape shaped by shifting cultivation in the Yucatan Peninsula,

					Mexico. *Journal of Tropical Ecology.* **23(2),** 139–149 (2007).
340	*Borassus aethiopum*	Liliopsida	Negative	-0.191	Barot, S., Gignoux, J., Vuattoux, R., & Legendre, S. Demography of a savanna palm tree in Ivory Coast (Lamto): population persistence and life-history. *Journal of Tropical Ecology.* **16(5),** 637–655 (2000).
341	*Calamus rhabdocladus*	Liliopsida	Negligible	0.016	Binh, PhD thesis, 2009.
342	*Calamus nambariensis*	Liliopsida	Negligible	0.050	Binh, PhD thesis, 2009.

343	*Daemonorops poilanei*	Liliopsida	Negative	-0.183	Binh, PhD thesis, 2009.
344	*Eremospatha macrocarpa*	Liliopsida	Negligible	-0.116	Kouassi, K. I., Barot, S., Gignoux, J., & Zoro Bi, I. A. Demography and life history of two rattan species, *Eremospatha macrocarpa* and *Laccosperma secundiflorum*, in Côte d'Ivoire. *Journal of Tropical Ecology.* **24(05),** 493–503 (2008).
345	*Laccosperma secundiflorum*	Liliopsida	Negligible	-0.024	Kouassi, K. I., Barot, S., Gignoux, J., & Zoro Bi, I. A. Demography and life history of two rattan species, *Eremospatha macrocarpa* and *Laccosperma secundiflorum*, in Côte d'Ivoire. *Journal of Tropical Ecology.* **24(05),** 493–503 (2008).
346	*Mauritia flexuosa*	Liliopsida	Negligible	0.041	Holm, J. A., Miller, C. J., & Cropper, W. P. Population Dynamics of the Dioecious Amazonian Palm *Mauritia flexuosa*: Simulation Analysis of Sustainable Harvesting. *Biotropica.* **40(5),** 550–558 (2008).
347	*Fritillaria meleagris*	Liliopsida	Negligible	-0.093	Zhang, L. & Hytteborn, H. Effect of ground water regime on development and distribution of *Fritillaria meleagris*. *Ecog.* **8(4),** 237-244 (1985).
348	*Erythronium japonicum*	Liliopsida	Negative	-0.151	Kawano, S. *et al.* Demographic differentiation and life-history evolution in temperate woodland plants (1987).
349	*Calochortus macrocarpus*	Liliopsida	Negligible	-0.033	Allen, PhD thesis, 2004.
350	*Calochortus lyallii*	Liliopsida	Negligible	0.021	Allen, PhD thesis, 2004.
351	*Calochortus pulchellus*	Liliopsida	Negative	-0.227	Fiedler, P. L. Life History and Population Dynamics of Rare and Common Mariposa Lilies (*Calochortus Pursh*: Liliaceae). The *Journal of Ecology*. **75(4),** 977 (1987).
352	*Calochortus tiburonensis*	Liliopsida	Negligible	-0.036	Fiedler, P. L. Life History and Population Dynamics of Rare and Common Mariposa Lilies (*Calochortus Pursh*: Liliaceae). The *Journal of Ecology*. **75(4),** 977 (1987).
353	*Calochortus albus*	Liliopsida	Negligible	-0.005	Fiedler, P. L. Life History and Population Dynamics of Rare and Common Mariposa Lilies (*Calochortus Pursh*: Liliaceae). The *Journal of Ecology*. **75(4),** 977 (1987).
354	*Prosartes lanuginosa*	Liliopsida	Negligible	0.001	Jackson, M.M., Pearson, S.M. & Turner, M.G. Performance and population dynamics of a native understory herb differ between young and old forest stands in the Southern Appalachians. *Forest. Ecol. Manage.* **304,** 444-454 (2013).
355	*Colchicum autumnale*	Liliopsida	Negligible	-0.076	Winter, S., Jung, L. S., Eckstein, R. L., Otte, A., Donath, T. W., & Kriechbaum, M. Control of the toxic plant *Colchicum autumnalein* semi-natural grasslands: effects of cutting treatments on demography and diversity. *Journal of Applied Ecology.* **51(2),** 524–533 (2014).
356	*Disporum sessile*	Liliopsida	Negligible	0.001	Kawano, S. *et al.* Demographic differentiation and life-history evolution in temperate woodland plants (1987)
357	*Trillium persistens*	Liliopsida	Negligible	-0.008	Plank, MSc thesis, 2010.
358	*Trillium grandiflorum*	Liliopsida	Negative	-0.191	Knight, T. M. Effects of herbivory and its timing across populations of *Trillium grandiflorum (Liliaceae)*. *American Journal of Botany.* **90(8),** 1207–1214 (2003).

359	*Trillium ovatum*	Liliopsida	Negative	-0.170	Ream, PhD thesis, 2011.
360	*Trillium apetalon*	Liliopsida	Negative	-0.225	Ohara, M., Takada, T., & Kawano, S. Demography and reproductive strategies of a polycarpic perennial, *Trillium apetalon (Trilliaceae). Plant Species Biology* **16(3),** 209–217 (2001).
361	*Trillium camschatcense*	Liliopsida	Negative	-0.187	Ohara, M., Tomimatsu, H., Takada, T., & Kawano, S. Importance of life history studies for conservation of fragmented populations: A case study of the understory herb, *Trillium camschatcense*. *Plant Species Biology*. **21(1),** 1–12 (2006).
362	*Dioscorea chouardii*	Liliopsida	Negligible	-0.124	García, M.B., Guzmán, D. & Goñi, D. An evaluation of the status of five threatened plant species in the Pyrenees. *Biol. Cons.* **103(2),** 151-161 (2002).
363	*Arisaema serratum*	Liliopsida	Negligible	-0.097	Kinoshita, E. Sex Change and Population Dynamics in *Arisaema serratum*. *Plant Species Biology*. **2(1-2),** 15–28 (1987).
364	*Duguetia neglecta*	Magnoliopsida	Negligible	-0.009	Zagt;Boot PhD thesis, 1997.
365	*Oxandra asbeckii*	Magnoliopsida	Positive	0.171	Chagneau, P., Mortier, F. & Picard, N. Designing permanent sample plots by using a spatially hierarchical matrix population model. *J.R.Stat. Soc. C.* **58(3),** 345-367 (2009).
366	*Magnolia macrophylla*	Magnoliopsida	Negligible	-0.124	Sánchez-Velásquez, L.R. & Pineda-López, M.dR. Comparative demographic analysis in contrasting environments of *Magnolia dealbata*: an endangered species from Mexico. *Pop. Ecol.* **52(1),** 203-210 (2009).
367	*Magnolia salicifolia*	Magnoliopsida	Negligible	-0.099	Hara, M., Kanno, H., Hirabuki, Y., & Takehara, A. Population dynamics of four understorey shrub species in beech forest. *Journal of Vegetation Science.* **15(4),** 475–484 (2004).
368	*Magnolia fordiana*	Magnoliopsida	Negligible	-0.093	Chien, P. D., Zuidema, P. A., & Nghia, N. H. Conservation prospects for threatened Vietnamese tree species: results from a demographic study. *Population Ecology.* **50(2),** 227–237 (2008).
369	*Lindera umbellata*	Magnoliopsida	Negligible	-0.068	Hara, M., Kanno, H., Hirabuki, Y., & Takehara, A. Population dynamics of four understorey shrub species in beech forest. *Journal of Vegetation Science.* **15(4),** 475–484. (2004).
370	*Lindera benzoin*	Magnoliopsida	Negative	-0.137	Vejdani, PhD Theis, 2006.
371	*Dacrydium elatum*	Pinopsida	Negligible	-0.104	Chien, P. D., Zuidema, P. A., & Nghia, N. H. Conservation prospects for threatened Vietnamese tree species: results from a demographic study. *Population Ecology.* **50(2),** 227–237 (2008).
372	*Araucaria muelleri*	Pinopsida	Negligible	-0.066	Enright, N. J., Miller, B. P., Perry, G. L. W., Goldblum, D., & Jaffré, T. Stress-tolerator leaf traits determine population dynamics in the endangered New Caledonian conifer *Araucaria muelleri*. *Austral Ecology.* **39(1),** 60–71. (2013).
373	*Araucaria araucana*	Pinopsida	Negligible	0.023	Bekessy, S. *et al.* Monkey Puzzle Tree (*Araucaria araucana*) in Southern Chile: effects of timber and seed harvest, volcanic activity, and fire in species conservation and management: Case studies (OUP, 2004).

374	*Araucaria hunsteinii*	Pinopsida	Negligible	-0.101	Enright, N. J. Does *Araucaria hunsteinii* compete with its neighbours? Austral *Ecology.* **7(1),** 97–99 (1982).
375	*Calocedrus macrolepis*	Pinopsida	Negligible	-0.070	Chien, P. D., Zuidema, P. A., & Nghia, N. H. Conservation prospects for threatened Vietnamese tree species: results from a demographic study. *Population Ecology.* **50(2),** 227–237 (2008).
376	*Calocedrus decurrens*	Pinopsida	Negative	-0.253	Van Mantgem, P. J., & Stephenson, N. L. The accuracy of matrix population model projections for coniferous trees in the Sierra Nevada, California. *Journal of Ecology.* **93(4),** 737–747 (2005).
377	*Taxus floridana*	Pinopsida	Negative	-0.151	Kwit, C., Horvitz, C. C., & Platt, W. J. Conserving Slow-Growing, Long-Lived Tree Species: Input from the Demography of a Rare Understory Conifer, *Taxus floridana*. *Conservation Biology*, **18(2),** 432–443 (2004).
378	*Pinus ponderosa*	Pinopsida	Negative	-0.170	Buckley, Y. M. *et al.* Slowing down a pine invasion despite uncertainty in demography and dispersal. *Journal of Applied Ecology*. **42(6)**, 1020–1030 (2005).
379	*Pinus sylvestris*	Pinopsida	Positive	0.134	Usher, M. B. A Matrix Approach to the Management of Renewable Resources, with Special Reference to Selection Forests. *The Journal of Applied Ecology*. **3(2),** 355 (1966).
380	*Pinus nigra*	Pinopsida	Negligible	-0.011	Buckley, Y. M. *et al.* Slowing down a pine invasion despite uncertainty in demography and dispersal. *Journal of Applied Ecology*. **42(6)**, 1020–1030 (2005).
381	*Pinus fenzeliana*	Pinopsida	Negligible	-0.023	Chien, P. D., Zuidema, P. A., & Nghia, N. H. Conservation prospects for threatened Vietnamese tree species: results from a demographic study. *Population Ecology*. **50(2),** 227–237 (2008).
382	*Pinus lambertiana*	Pinopsida	Negligible	-0.092	Van Mantgem, P. J., & Stephenson, N. L. The accuracy of matrix population model projections for coniferous trees in the Sierra Nevada, California. Journal of *Ecology.* **93(4**), 737–747 (2005).
383	*Pinus strobus*	Pinopsida	Negligible	-0.063	Münzbergová, Z., Hadincová, V., Wild, J., & Kindlmannová, J. (2013). Variability in the Contribution of Different Life Stages to Population Growth as a Key Factor in the Invasion Success of *Pinus strobus*. *PLoS ONE*. **8(2**), e56953 (2013).
384	*Pinus maximartinezii*	Pinopsida	Negligible	0.010	López-Mata, L. The impact of seed extraction on the population dynamics of *Pinus maximartinezii*. *Acta Oecologica*, **49**, 39–44 (2013).
385	*Abies sachalinensis*	Pinopsida	Negligible	-0.076	Hiura, T., & Fujiwara, K. Density-dependence and co-existence of conifer and broad-leaved trees in a Japanese northern mixed forest. *Journal of Vegetation Science.* **10(6),** 843–850 (1999).
386	*Abies concolor*	Pinopsida	Negligible	-0.054	Van Mantgem, P.J. & Stephenson, N.L. The accuracy of matrix population models for coniferous trees in the Sierra Nevada, California. *Journal of Ecology.* **93(4)**, 737-747 (2005)
387	*Abies magnifica*	Pinopsida	Negligible	-0.044	Van Mantgem, P.J. & Stephenson, N.L. The accuracy of matrix population models for coniferous trees in the Sierra Nevada,

					California. *Journal of Ecology*. **93(4)**, 737-747 (2005)
388	*Tsuga canadensis*	Pinopsida	Negative	-0.202	Lamar, W. R., & McGraw, J. B. Evaluating the use of remotely sensed data in matrix population modeling for eastern hemlock (*Tsuga canadensis L.*). *Forest Ecology and Management*, **212(1-3**), 50–64 (2005).
389	*Encephalartos villosus*	Cycadopsida	Negative	-0.209	Raimondo, D. C., & Donaldson, J. S. Responses of cycads with different life histories to the impact of plant collecting: simulation models to determine important life history stages and population recovery times. *Biological Conservation.* **111(3),** 345–358 (2003).
390	*Encephalartos cycadifolius*	Cycadopsida	Negative	-0.160	Raimondo, D. C., & Donaldson, J. S. Responses of cycads with different life histories to the impact of plant collecting: simulation models to determine important life history stages and population recovery times. *Biological Conservation.* **111(3),** 345–358 (2003).
391	*Dioon caputoi*	Cycadopsida	Negative	-0.302	Cabrera-Toledo, PhD thesis, 2009.
392	*Dioon merolae*	Cycadopsida	Negligible	-0.047	Lázaro-Zermeño, J. M., González-Espinosa, M., Mendoza, A., Martínez-Ramos, M., & Quintana-Ascencio, P. F. Individual growth, reproduction and population dynamics of *Dioon merolae* (Zamiaceae) under different leaf harvest histories in Central Chiapas, Mexico. *Forest Ecology and Management.* **261(3),** 427–439 (2011).
393	*Dioon sonorense*	Cycadopsida	Negative	-0.314	Álverez-Yépiz, J.C., Dovčiak, M. & Búrquez, A. Persistence of a rare ancient cycad: Effects of environment and demography. *Biol. Cons.* **144(1),** 122-130 (2011).
394	*Dioon spinulosum*	Cycadopsida	Negative	-0.169	Casteneda MSc thesis, 2008.
395	*Zamia inermis*	Cycadopsida	Negative	-0.147	Octavio-Aguilar, P. *et al.* Extinction risk of *Zamia inermis*: a demographic study in its single natural population. *Biodiversity and Conservation.* **26(4),** 787-800 (2017).

Appendix B

PROCEEDINGS B

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Invited reply

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Reply to: Senescence, trait parameterization and (st)age-specific forces of selection

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We respond to the Comment of Bahry [1] regarding our review of why and where selection gradients might not decline with age [2]). Bahry [1] posits that Roper *et al.* [2] 'repeat a subtle mischaracterization of the relationship between Hamilton's indicators of selection and Caswell's generalized fitness sensitivity for (st)age-structured population projection matrices'.

However, we note that equations 3.1 and 3.2 of Roper *et al.* [2] are not the discrete time equations 9.1 and 9.2 of Bahry [1] and are instead equivalent to continuous time reformulations of Hamilton's seminal work [3] by Caswell ([4]—equations 1 and 2). Caswell [4] displays the equivalence of their Equations (1) and (2) and Hamilton's forces of selection in §2.2 of his work. We acknowledge that this confusion has arisen due to the reference of Caswell (1978) rather than Caswell (2010) in our original piece. We agree with Bahry [1]'s detailed comparison between Hamilton (1966) and Caswell (1978) and thank the author for creating the opportunity to provide further clarity. We would also like to clarify that Roper *et al.* [2] did not intend to assign equivalence between equations 6.1 and 6.2, and 9.1 and 9.2, of Bahry [1], even though, as the author notes, the differences between the continuous time and discrete time versions are trivial anyway.

To avoid any further confusion for the reader and the discipline, we have included table 1, which displays comparisons between the work of Hamilton (1966), Caswell (1978) and Caswell (2010). As displayed in table 1, and in the response by Bahry [1], Caswell (1978) generalized Hamilton's (1966) sensitivity analysis approach to stage-structured populations with discrete time, in which the rate of population growth λ, rather than the intrinsic rate of natural increase r, is the measure of fitness. This approach differs in assuming the genetic effects on survival are additive, rather than multiplicative (Hamilton (1966) assumed genetic effects subtract from mortality risk [1]), and that fecundity is 'effective' (i.e. it includes survival to the first age class) rather than indicating the total number of offspring produced. These assumptions lead to slight differences in the forces of selection acting on genes that affect mortality risk (or survival). In particular, it is residual reproductive value at age x in discrete time versus reproductive value in continuous time that balances the force of selection. For reproduction, if reproductive value at the first age class is defined as 1, then there are no differences between the discrete and continuous time forces of selection.

To summarize, the logic of Roper *et al.* [2] was to highlight the fundamental importance of the stable (st)age and reproductive value distributions when considering variation in the evolution of senescence. Equations 3.1 and 3.2 [2] were used as conceptual springboards, from which to (i) compare stable (st)age and reproductive value distributions against rates of senescence for species using matrix population models [5,6] and (ii) propose why and where across the tree of life one might expect a decline in selection pressure with age not to be as pronounced, contrary to predictions from Hamilton (1966) on the need for senescence to be universal. Bahry [1] is correct in noting that, depending on the assumptions one makes about the action of genes, selection gradients may differ in form [7]. However, this is a question of mathematical parameterization and does not detract from the main biologically driven argument of

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Table 1. Comparisons of the force of selection on mortality and fertility in Hamilton (1966), Caswell (1978) and Caswell (2010). The intrinsic natural rate of increase, r, is derived as the single real root of the Euler–Lotka equation in continuous time modelled populations, whereas the population growth rate λ ($= e^r$) is the dominant eigenvalue of a population projection matrix that considers a population in discrete time. Caswell (1978) considered the sensitivity analysis approach in discrete time, which leads to slight differences in the forces of selection. On the other hand, Caswell's (2010) modifications showed that $l(x)e^{-rx} = c(x)/b$, where $c(x)$is the stable age distribution of age class x and $b\left(\left[\int_0^\infty e^{-rx}l(x)\,dx\right]^{-1}\right)$ is the birth rate. Equivalence between Hamitlon (1966) and Caswell (2010) can be demonstrated when these relationships are inputted into Hamilton's forces of selection (rows 4 and 6 of the first column). Other definitions from the table: T = generation time, $l(x)$ = survival probability from birth to age x, p_x = survival between age x-1 and x, F_x = effective fecundity at age x, $m(x)$ = fecundity at age x and $v(x)$ = reproductive value at age x.

	Hamilton 1966	Caswell 1978	Caswell 2010
measure of fitness	r	λ	r
assumed form of genetic effect on survival probability	multiplicative	additive	multiplicative
assumed form of reproduction	age-specific fecundity (m_x)	effective fecundity (F_x) – includes survival to age 1	(st)age-specific fecundity (m_x)
how is time modelled?	interval/continuous	discrete	interval/continuous
force of selection on reproduction	$\frac{\partial r}{\partial m(x)} = \frac{e^{-rx}l(x)}{T}$	$\frac{\partial \lambda}{\partial F_x} \propto c_x v_1$	$\frac{\partial r}{\partial m(x)} = \frac{c(x)}{bT}$
mathematical relationship to Hamilton 1966	N/A	$\frac{\partial r}{\partial m(x)} = \frac{p_0}{\lambda}\left(\frac{\partial \lambda}{\partial F_x}\right) = \frac{e^{-rx}l(x)}{T}$	$l(x)e^{-rx} = c(x)/b$
force of selection on mortality/survival	$\frac{\partial r}{\partial \mu(x)} = -\frac{\int_x^\infty e^{-rx}l(y)m(y)dy}{T}$	$\frac{\partial \lambda}{\partial p_x} \propto c_x v_{x+1}$	$\frac{\partial r}{\partial \mu(x)} = \frac{-c(x)v(x)}{bT}$
mathematical relationship to Hamilton 1996	N/A	$-\frac{\partial r}{\partial \mu(x)} = \frac{\partial r}{\partial \ln p_x} = \frac{p_x}{\lambda}\left(\frac{\partial \lambda}{\partial p_x}\right) = \frac{e^{-rx}l(x)RRV_x}{T}$	$\int_x^\infty e^{-rx}l(y)m(y)dy = l(x)e^{-rx}v(x)$

Roper *et al*. [2]: that the strength of selection on a (st)age class with a given life cycle will be proportional to the genetic contribution to future populations of that (st)age class, which can be proxied by considering that (st)age class' reproductive value and stable (st)age distribution. Our conclusion is generally true regardless of whether one considers continuous or discrete time, and ignorant of the exact action of genes.

Data accessibility. This article has no additional data.

Authors' contributions. M.R.: conceptualization, writing—original draft and writing—review and editing; R.S.-G.: writing—review and editing.

Competing interests. We declare we have no competing interests.

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Appendix C

Oxford Bibliographies
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Senescence

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Introduction

Researchers are now beginning to unravel the variation in patterns of whole-organism senescence across the tree of life, yet the existence of senescence in nature itself is an evolutionary conundrum. This document introduces the literature on explanations for whole-organism senescence, starting with the early classical theories developed in the 20th century. This piece reviews extensions to these classical theories before touching on the wealth of data now available that challenges their predictions. The manuscript concludes by considering current research on more modern theories of whole-organism senescence, as the field attempts to bridge the gap between current theoretical predictions and empirical observations.

Cellular, Physiological, and Demographic Senescence

Senescence is the decline of vitality with age, from maturity until the organism dies. Underlying losses of vitality are driven by physiological breakdowns (i.e., dysfunction of functional and regulatory properties of tissues and the endocrine system) that may ultimately emerge from cellular breakdowns (Comfort 1964). If these mechanisms upscale to the whole individual, senescence can then be quantified at the demographic level as the decline in reproductive outputs and/or increase of mortality risks with age. It is noteworthy highlighting that demographic senescence is not necessarily the result of cellular senescence, as cellular declines with age may or may not upscale to demographic effects on an individual organism (Shefferson, et al. 2017). The extent to which lower-level, age-mediated breakdowns translate into demographic effects—thus influencing an individual's mortality and reproductive schedule—is not firmly resolved and remains an active area of research (Finch 1990; see also Modern Theories of Senescence—Growth Forms). This section focuses on the theories and current evidence of the drivers of the evolution of senescence at the whole-organism level, as well as the (a)biotic drivers that allow organisms to postpone its onset or even escape from it.

Comfort, A. 1964. *Ageing: The biology of senescence*. Rev. ed. London: Routledge & Kegan Paul.

Comfort's textbook surveys the knowledge about ageing from cellular mechanisms to comparative population biodemography. This is a classical text in the broad-scale study of senescence and the study of ageing as mediated by different mechanisms and translating across different scales of biological organization. Comfort does not cover a number of theoretical and empirical developments that are addressed by the more recent textbooks Finch 1990 and Shefferson, et al. 2017.

Finch, C. E. 1990. *Longevity, senescence, and the genome*. Chicago: Univ. of Chicago Press.

This textbook is a seminal review that covers ageing from the ground up—starting with cells and walking through the levels at which age-correlated dysfunctions manifest. Finch ably navigates the partitions between different levels of biological organization, drawing on expertise in the domain of the cellular level through the individual level, as well as among species. The first several chapters of Finch's book offer a concise and clear review of the semantic distinctions surrounding senescence.

Shefferson, R. P., O. R. Jones, and R. Salguero-Gómez, eds. 2017. *The evolution of senescence in the tree of life*. Cambridge, UK: Cambridge Univ. Press.

This textbook provides an overview of senescence research focusing on biological diversity in ageing. The authors provide a clear distinction amongst different types and levels of ageing. In the introductory chapter of the book, Shefferson and colleagues discuss the terminology of senescence and subfields that tackle physiological, mathematical, and evolutionary dimensions of the "senescence problem." This resource provides a general overview of the current theories on the evolution of senescence and the status quo of the understanding of senescence across different taxonomic groups.

Classical Theories of Senescence

Three evolutionary models of senescence emerged in the second half of the 20th century. While myriad variations on each of these core theories of senescence have been developed, these three models give the core explanation for why senescence occurs in nature. They are: (1) the theory of mutation accumulation in Medawar 1952, (2) the theory of antagonistic pleiotropy in Williams 1957, and (3) the theory of disposable soma in Kirkwood 1977. Medawar argues that deleterious mutations in the germ line of a species will be less effectively removed by natural selection when such mutations are expressed in older age classes. This is because in the wild few individuals survive long enough to be adversely affected by late-acting, deleterious genes. With progressively older ages at which genes act, natural selection is less effective at removing deleterious genes from the genome. Thus, deleterious mutations expressed late in life tend to accumulate at a higher frequency than they could earlier in life, when the strength of natural selection is stronger. The theory posed in Williams 1957 builds on these ideas, along with Weissman 1881, exploring the implications of separation in the germ line (which forms the reproductive cell line) from the somatic line (which forms the cell line that dies with each individual life) on natural selection. In antagonistic pleiotropy, the genes with detrimental late-life effects can still be under positive selection if they have early-life fitness benefits. This work leads to the conclusion that insofar as reproduction (germ) and bodily maintenance (soma) are antagonistic, selection will promote early-life performance and shorter lifespan over late-life performance and a longer-lifespan. This balance can shift, depending on the extent to which maintaining the soma equates to additional germ line reproduction. The 'disposable soma' theory in Kirkwood 1977 centers on a trade-off regarding the optimal allocation between survival, maintenance, and reproduction. As it is the germ line that ensures propagation of genetic material between generations, the author argues that natural selection will favor a level of somatic investments that optimizes the success of the germline, rather than a level that would facilitate indefinite somatic survival. The three classical theories of senescence provide independent but not mutually exclusive explanations for why and when senescence should occur. All three theories rely on the concept of a selection shadow—first coined by Medawar—which explains that the fitness of an individual will be devalued over time. Hamilton 1996 (cited under Data Biases) formalizes this idea, displaying mathematically that the strength of selection on age-specific mortality and reproduction in an age-structured population does indeed decline with age. Hamilton used the Malthusian parameter (r), also known as the intrinsic rate of increase, as a measure of fitness, and quantified the partial derivatives of r with respect to a mutational change in age-specific mortality and reproduction. More detailed coverage of the role of selection shadow is advanced in G. C. Williams's 1966 book *Adaptation and Natural Selection*, and a recent review by Gaillard and Lemaître 2017 revisits key predictions of this theory. See also the separate *Oxford Bibliographies* in Evolutionary Biology article Evolutionary Biology of Aging by Grant A. Rutledge and Michael R. Rose.

Gaillard, J. M., and J. F. Lemaître. 2017. The Williams' legacy: A critical reappraisal of his nine predictions about the evolution of senescence. *Evolution* 71.12:2768–2785.

The authors reevaluate of the models discussed in Williams' paper on pleiotropy and weigh them against the evidence of decades of lab and field studies. This paper affirms Williams' predictions concerning the force of mortality, fertility, and sex-specific differences in reproduction; the authors critique William's view on the timing of the onset of senescence, the consequences of reproductive cessation, and the consistency with which ageing patterns occur in view of contemporary evidence.

Kirkwood, T. B. L. 1977. Evolution of ageing. *Nature* 270:301–304.

This paper outlines Kirkwood's theory of disposable soma. Kirkwood argues that there are mechanisms that allocate common physical

energetic, or temporal resources between germ line (reproduction) and soma (bodily maintenance). In principle, integrity of the germ line and reproduction should be favored over the soma unless somatic investment translates to germ line benefits that exceed the discounting effect of the selection shadow.

Medawar, P. B. 1952. *An unsolved problem of biology*. London: H.K. Lewis.

This book showcases Peter Medawar's theory about the role of mutation accumulation in the germ line as an explanation of the tendency for biological senescence. The book is the first clear expression of a nonadaptive theory of senescence and it coined the term "selection shadow." This seminal piece also kicked off the first wave of research into the mechanisms of senescence (e.g., Comfort 1964, cited in Cellular, Physiological, and Demographic Senescence). Its primary ideas retain currency.

Rutledge, G. A., and M. R. Rose. 2018. "Evolutionary Biology of Aging." Oxford: Oxford Bibliographies in Evolutionary Biology.

This Oxford Bibliography piece explores in greater detail the history of the early theories of senescence. The authors delve deeper into the mathematical evolutionary theories of aging, exploring the works of Hamilton, Charlesworth, and Rose. They also review studies of the quantitative genetics of aging, citing studies from key model organisms such as *Drosophila melanogaster* and *Caenorhabditis elegans*.

Weissman, A. 1881. *Ueber die Dauer des Lebens ein Vortrag*. Berlin: Humboldt.

In this piece, the author proposes that senescence is an evolved genetically programmed adaptation that has species benefits. Specifically, programmed death was proposed to be an evolved characteristic that frees resources for young, more adapted members of a population. Since then, this idea has been largely replaced by other classical theories of senescence under which this process is not an adaptation but results from accumulated mutations or trade-offs in vital functions.

Williams, G. C. 1957. Pleiotropy, natural selection, and the evolution of senescence. *Evolution* 11.4:398–411.

This paper represents the original exposition of Williams's theory of antagonistic pleiotropy and is often characterised as an expansion upon Medawar's work. This work presupposes Medawar's axiom of a selection shadow and incorporates genetic interactions. Williams deduced that connected genes (i.e., pleiotropies) that have actions with mixed fitness consequences (i.e., antagonistic) offering benefits early in life at a cost late in life will be selected for because of the selection shadow.

Extensions to the Classical Theories—Extrinsic Mortality

Extrinsic mortality supports all three classical theories of senescence. The logic underlying classical theories is that extrinsic mortality limits the attainment of old age and thus it diminishes the strength of selection at older ages. Extrinsic mortality has long been a contested area in the senescence literature. Some of the confusion around the role of extrinsic mortality in shaping the evolution of senescence arose from ambiguity in defining "extrinsic mortality". Definitions of extrinsic mortality range from "any environmental factor directly responsible for mortality" (e.g., as measured by contrasting *in situ* and *ex situ* survivorship curves) to the slope of the Gompertz curve as introduced in Ricklefs 2008, which separates a baseline mortality coefficient from a coefficient that scales the rate of mortality that changes as a function of age. Beyond definitions, there is a conceptual challenge in attributing death to extrinsic causes (independent of intrinsic mortality) due to the multiple contributing forces on mortality. The age-structure of death is often an interaction between intrinsic susceptibilities and extrinsic causes. One definition of senescence gathering consensus, as used in Koons, et al. 2014, defines extrinsic mortality operationally, based on whether it is added or subtracted from a system, regardless of vital rates. This additive mortality model introduces mortality to a structural population model, where mortality can be age-structured or non-age-structured (the structural population models to which mortality is applied may be age-structured or non-age-structured as well). Vital rates can also have age-specific density-dependent properties under this model. The age-specific density properties mean that a

rates can also have age-specific density-dependent properties under this model. The age-specific density properties mean that a uniformly distributed mortality event may result in age-specific changes in vital rates. Thus whether the age-structure of a population is influenced by mortality is conditional on both the age bias of mortality and the age bias of vital rates that respond to mortality. The extrinsic mortality hypothesis at the core of debate concerns changes in this additive component of extrinsic mortality and does not require further partitioning of intrinsic mortality from extrinsic mortality. The consequences of whether extrinsic mortality is age-structured or not is the core of one of the debates in this subfield. Williams 1957, an antagonistic pleiotropy theory paper (cited under Classical Theories of Senescence), sets out at least nine verbal hypotheses, one of which has been interpreted to indicate that species that experience greater rates of age-independent extrinsic mortality should display more rapid senescence. This prediction initiated debate about the interaction of senescence and extrinsic mortality that continues as of the writing of the present article. Recent literature has found agreement on a few points, however. Namely, age structure measures the evolutionary change; regardless of mediation by density, there must be a difference between age structure in a “low mortality” and “high mortality” environment to have any selection effect on senescence. Absent that difference, mortality is irrelevant, as noted in Abrams 1993 and Caswell 2007, and further discussed in Day and Abrams 2020, Moorad, et al. 2019, and Wensink, et al. 2017.

Abrams, P. A. 1993. Does increased mortality favor the evolution of more rapid senescence? *Evolution* 47.3:877–887.

This is an early, influential paper analytically tackling the question of why and how extrinsic mortality should lead to changes in selection for senescence. Abrams points out that increased extrinsic mortality will also reduce the population growth rate, and the two effects can cancel one another out with respect to benefits of reproducing at different age classes. The author notes that, for Williams’ prediction to hold, other factors such as a corresponding increase in reproduction rate must also occur.

Caswell, H. 2007. Extrinsic mortality and the evolution of senescence. *Trends in Ecology & Evolution* 22.4:173–174.

This short and elegant piece examines how external mortality may not necessarily result in a change in the tendency to senesce. The author demonstrates that additional (external) mortality is not in itself sufficient for senescence to be further accelerated, unless the pattern of external mortality is age dependent, in which case the effect will be either positive or negative.

Day, T., and P. A. Abrams. 2020. Density dependence, senescence, and Williams’ hypothesis. *Trends in Ecology & Evolution* 35.4:300–302.

This article defends Williams’ hypothesis that higher, age-independent adult mortality should correlate with more rapid senescence, arguing that extrinsic mortality may lead to increased selection for senescence. The authors also respond to the critique in Moorad, et al. 2019 and discuss why the predictions of Williams’ hypothesis may hold once density-dependency is accounted for. They make the case that density-dependency changes the otherwise nullifying relationship between extrinsic mortality and population growth rates, thereby increasing the strength of selection for senescence when extrinsic mortality exists.

Koons, D., M. Gamelon, J. M. Gaillard, et al. 2014. Methods for studying cause-specific senescence in the wild. *Methods in Ecology and Evolution* 5.9:924–933.

The authors introduce methods to test the hypothesis that the force of mortality imposed by environmental factors should influence the strength of natural selection against actuarial senescence and its evolution.

Moorad, J., D. Promislow, and J. Silvertown. 2019. Evolutionary ecology of senescence and a reassessment of Williams’ ‘extrinsic mortality’ hypothesis. *Trends in Ecology & Evolution* 34.6:519–530.

The authors discuss the case made for and against Williams’ extrinsic mortality hypothesis, stepping into both logical claims, such as those in Wensink, et al. 2017 as well as other empirical data. They discuss the assumptions that are necessary for the extrinsic mortality hypothesis to hold. This suggests a narrow circumscription of the conditions when one should expect clear cause and effect between extrinsic mortality and selection for senescence.

Ricklefs, R. 2008. The evolution of senescence from a comparative perspective. *Functional Ecology* 22.3:379–392.

The author provides a detailed account of the state of the art (through the year of publication) in the examination of the patterns and mechanisms of ageing in vertebrate populations. He uses a Weibull function to separate baseline mortality at maturity from the rate of senescence.

Wensink, M. J., H. Caswell, and A. Baudisch. 2017. The rarity of survival to old age does not drive the evolution of senescence. *Evolutionary Biology* 44.1:5–10.

This paper makes a direct claim against the logic of Williams' hypothesis concerning extrinsic mortality and how it drives the evolution of senescence. Wensink and colleagues reason that extrinsic mortality does not simply equate to a reduction in survivorship. Rather, in tandem with survivorship declines, competition declines will have an opposing effect of equal magnitude. Thus unless competition is asymmetric with respect to survivorship Williams' hypothesis runs up against conceptual inconsistencies.

Extensions to the Classical Theories—Germ-Soma vs. Parent-Offspring Divide

A specific prediction of the classical theories of senescence is that its consequences are limited to species with a clear distinction between germ line and soma. Williams 1957 (cited under Classical Theories of Senescence) states that the theory of antagonistic pleiotropy required an assumption of germ-soma separation, and the theory in Kirkwood 1977 (also cited under Classical Theories of Senescence) was built around the idea that the soma can be 'disposable' to ensure the survival of the germ line. In the early 2000s, however, studies displayed that senescence can be observed in unicellular organisms such as bacteria, as shown in Ackermann, et al. 2003, where no such germ-soma division is found. This discrepancy between theory and data raised questions about this major prediction of the classical theories, yet a solution may have already been found in the 1990s. A thought experiment in Partridge and Barton 1993 pre-emptively suggests such a solution: The authors imagined a multicellular organism whose somatic cells de-differentiated randomly until the organism consisted only of germ cells. The logic of the theories of senescence, these authors detail, is founded upon the fact that organisms experience a non-zero probability of death in the wild (i.e., there is always a risk of extrinsic mortality). Given this, there is no reason to believe the hypothetical organism should fare any differently. The organism will still have a non-zero probability of death or becoming unable to reproduce. The intensity of selection on the organism's survival and reproduction will therefore still decline with age, and senescence will still evolve. The critical requirement for senescence, the authors argue, is instead a distinction between the parent individual and the offspring it produces. If there is no such distinction, and subsequently no age-structure within a population, then the intensity of selection should remain constant across individuals, and senescence should not evolve. Supporting their argument, evidence of senescence in unicellular organisms to date has only come from species with asymmetrical division. Other works such as Vaupel, et al. 2004 (cited under Modern Theories of Senescence—Growth Forms) and Caswell and Salguero-Gómez 2013 have since shown that other categorizations, rather than age *per se*, can also result in differential selection among classes of individuals and govern ageing trajectories (see Modern Theories of Senescence—Growth Forms). Therefore current theory and data seem to point toward a parent-offspring divide being a necessary but not sufficient condition for the evolution of senescence. A germ-soma divide, on the other hand, contrary to original predictions, appears neither necessary nor sufficient.

Ackermann, M., S. C. Stearns, and U. Jenal. 2003. Senescence in a bacterium with asymmetric division. *Science* 300.5627:1920.

This paper found evidence for senescence in bacteria, providing critical evidence against the necessity of a germ-soma distinction for the evolution of senescence. The authors argue instead that the asymmetrical division of the bacteria (*Caulobacter crescentus*) created a clear parent-offspring divide and age structure, supporting the ideas of Partridge and Barton that this asymmetry is the fundamental requirement to the evolution of senescence.

Caswell, H., and R. Salguero-Gómez. 2013. Age, stage and senescence in plants. *Journal of Ecology* 101.3:585–595.

The authors provide evidence that the predicted age-based declines in the selection gradients for mortality and reproduction, which would explain the evolution of senescence, disappear in many plant species once other fitness-predictive traits, such as size of developmental stage, are considered.

Partridge, L., and N. H. Barton. 1993. Optimality, mutation and the evolution of ageing. *Nature* 362.6418:305–311.

This review paper categorized the evolutionary theories of senescence at the time into two distinct classes: optimality and maladaptive. The authors review the logic behind the two sets of theories, and empirical evidence for both. Their thought experiment challenging the logic behind a germ-soma distinction as a requirement for the evolution of senescence pre-dated empirical evidence of senescence in unicellular organisms in the early 2000s.

Quantifying Whole-Organism Senescence

Since all individuals age (here defined as the mere accumulation of time from birth, without any connotation on changes in fitness), reproduce, and ultimately die, whole-organism senescence can in principle be quantified across all species. The central complication in measuring whole-organism senescence is determining whether declines in vital rates are measured relative to absolute time or relative to the lifespan of a given species. Beyond the standardizing of lifespan, there are a number of more elaborate analytical techniques commonly employed to incorporate senescence with other features of structured population models—these methods generally fall into the field of mathematical demography. Weisman 1891 is one of the earliest works to explore demographic senescence. Keyfitz 1977 introduced a formal set of equations for evaluating normalized rates of demographic change. Keyfitz's work enabled rates of senescence across diverse life histories to be compared against one another. In particular, a metric oftentimes referred to as "Keyfitz entropy" (though it is important to note that it is in fact not a mathematical entropy) has been widely accepted as a standard of practice for measuring changes in survivorship over a standardized mean life expectancy. In fact, these methods—and the possibility for negligible and even negative senescence—are not new to population ecologists: the well-known survivorship curves Type I (i.e., higher rate of mortality at advanced ages), II (constant mortality rate with age), and III (higher mortality rates at earlier ages) are based on the seminal work Demetrius 1978 on entropies. Analogous methods, in a more specific application to ageing, along with more formal verbal semantics, are outlined in Baudisch 2011 for actuarial (mortality) senescence, and in Baudisch and Stott 2019 for reproductive senescence. Lifespan-normalized senescence metrics are ultimately measures of deviance from a null distribution for which age-specific survivorship and reproduction do not change over the life course of an individual. A range of metrics is used by biodemographers to estimate lifespan of an organism (termed "pace of life") which includes (1) mean lifespan, (2) maximum lifespan, (3) reproductive window, and (4) age at maturity. The appropriate measure(s) to characterize pace of life depend on the question being studied, and often more than one will be used in a study. Declines in survivorship or reproduction over lifespan-normalized time ("shape of life" or "shape of reproduction") are measured under Keyfitz's seminal metric and its later derivations.

Baudisch, A. 2011. The pace and shape of ageing. *Methods in Ecology and Evolution* 2.4:375–382.

This paper outlines the pace- and shape-of-life approach to modeling senescence in clear and accessible language. Although not novel, Baudisch packages Keyfitz's ideas into a mathematically convenient and verbally clear system of tackling standardized and nonstandardized measures of demographic change over time.

Baudisch, A., and I. Stott. 2019. A pace and shape perspective on fertility. *Methods in Ecology and Evolution* 10.11:1941–1951.

This paper is the complement of Baudisch 2011, extending the framework of the pace and shape of life to encompass reproductive senescence. Reproductive senescence involves a more complex set of curves, which requires careful attention as described in this text. This paper also outlines analytical shortcuts to measuring standardized rates of senescence for reproduction that can be used for survivorship.

Demetrius, L. 1978. Adaptive value, entropy and survivorship curves. *Nature* 275:213–214.

The author introduces three different kinds of survivorship curve (I, II and III) where survivorship declines mostly at advanced ages, is independent of age, or occurs predominantly at early ages, respectively. The author argues that each survivorship type is fixed within species, but sensitive to environmental conditions. This work introduces a measure of entropy to quantify these survivorship curves.

Keyfitz, N. 1977. *Applied mathematical demography*. New York: Wiley.

This classical demographic textbook lays out basic mathematical procedures that remain relevant to contemporary mathematical demography. The text outlines procedures for calculating Keyfitz's entropy. An updated version of the text is the 2005 version co-authored with Harold Caswell in 2005 (New York: Springer Science).

Weisman, A. 1891. *Essays upon heredity and kindred biological problems*. Oxford, UK: Clarendon Press.

In this work, the author establishes in this work his doctrine regarding the unbroken continuity of the germ plasm and the noninheritability of acquired characteristics.

Measuring Senescence in the Wild

In a 1951 lecture, Medawar commented that one should seldom expect to measure senescence in the wild because most animals (see Data Biases) would die of extrinsic causes before reaching ages at which senescence would become apparent. On the one hand, this comment raises the question (addressed in a previous section) about the evolutionary relevance of extrinsic mortality (how wild/natural/*in situ* populations respond to increased mortality); on the other hand, this question raises independent proximate questions about whether extrinsic mortality prevents underlying senescence from being realized. There is now clear evidence that senescence does occur in wild populations. A combination of comparative analyses, experimental manipulations in laboratory and mesocosm settings, and longitudinal studies where cohorts of populations are followed throughout their entire lifespan has elucidated not only the existence of senescence in nature, but also a wide diversity of senescence patterns. Some of the key works in this respect are Nussey, et al. 2008, Roach and Carey 2014, and Monaghan, et al. 2008. These range from positive (increased risk of mortality and reduced rate of reproduction with age) to negligible (constant rates with age) to negative (decreased risk of mortality and increased rate of reproduction with age). In this regard, a very well known example is that of fixed and very low rates of mortality in the aquatic metazoan *Hydra*, which suggests a lack of senescence for this species, as first reported in Martínez 1998. Patterns of senescence tend to have some phylogenetic component—for example Baudisch, et al. 2013 finds that the majority of angiosperms appear not to senesce, but also may span across taxa depending on key life history or ecological components. Theoretical treatments from Vaupel, et al. 2004 (cited under Modern Theories of Senescence—Growth Forms) and Caswell and Salguero-Gómez 2013 (cited under Extensions to the Classical Theories—Germ-Soma vs. Parent-Offspring Divide) suggest growth form and size may be key. Explaining the variation in patterns of senescence is the current focus of more modern theories of senescence.

Baudisch, A., R. Salguero-Gómez, O. Jones, et al. 2013. The pace and shape of senescence in angiosperms. *Journal of Ecology* 101.3:596–606.

This is a key comparative study of actuarial senescence rates in natural populations of approximately three hundred plant species, where the authors demonstrate a great deal of variation in senescence rates, but a relatively strong phylogenetic role underpinning this variance. The authors show that the immense majority of study species (93 percent) do not undergo actuarial senescence, and that most gymnosperms do undergo senescence.

Martínez, D. E. 1998. Mortality patterns suggest lack of senescence in hydra. *Experimental Gerontology* 33.3:217–225.

The author provides evidence that senescence, both in terms of survival and reproduction, is negligible in the freshwater metazoans of the genus *Hydra*.

Monaghan, P., A. Charmantier, D. H. Nussey, and R. E. Ricklefs. 2008. The evolutionary ecology of senescence. *Functional Ecology* 22.3:371–378.

This general and accessible review discusses the causes of senescence and claims that environmental mortality can preclude the realization of intrinsic demographic senescence in wild populations.

Nussey, D. H., T. Coulson, M. Festa-Bianchet, and J. M. Gaillard. 2008. Measuring senescence in wild animal populations: Towards a longitudinal approach. *Functional Ecology* 22.3:393–406.

This paper discusses the importance and value of measuring senescence in the wild and the applied challenges of the data that can be collected in the wild.

Roach, D. A., and J. R. Carey. 2014. Population biology of aging in the wild. *Annual Review of Ecology, Evolution, and Systematics* 45:421–443.

This review article on ageing in wild populations of animals evaluates how species are advantaged and disadvantaged by selection against older individuals being retained in wild populations. Unlike much of the literature on the topic, which highlights the extrinsic mortality component of selection, this paper discusses competing forces that would select for retaining older individuals in a population.

Salguero-Gómez, R., R. Shefferson, and R. Hutchings. 2013. Plants do not count . . . or do they? New perspectives on the universality of senescence. *Journal of Ecology* 101.3:545–554.

The authors outline experimental approaches to discern the roles of various sources of mortality on the age-based demographic trajectories of plants.

Data Biases

The study of senescence has remained mostly a human-centric discipline since its inception. This is quite understandable—in principle—since how fast we lose vitality has important aspects on our own health, economy, and social interactions. However, the classical (and some modern) theories on the evolution of senescence were developed at a time where data from only a small section of the tree of life were available to test them: primarily mammals and to a certain extent birds. Our understanding of the lack of universality of senescence, contrary to predictions in Hamilton 1996, has been supported recently by more extensive, long-term datasets on natural populations in Jones, et al. 2014 and Roper, et al. 2021 (animals and plants), Zajitschek, et al. 2020 (insects), and Maas, et al. 2017 (fungi). However, the data still remain significantly biased toward charismatic species that morphologically look like humans (e.g., tetrapods), as highlighted in Conde, et al. 2019 and Troudet, et al. 2017, and on ecoregions of the planet that contain a higher density of research institutions, as revealed in Salguero-Gómez, et al. 2016. From this angle, research on age-based vitality "in the farthest reaches of almost any bizarre universe" has shown that the Hamiltonian predictions on the generality of senescence were actually

incorrect.

Conde, D. A., J. Staerk, F. Colchero, et al. 2019. Data gaps and opportunities for comparative and conservation biology. *Proceedings of the National Academy of Sciences* 116.9:9658–9664.

In this paper the authors review the availability of demographic data to evaluate, among other aspects, actuarial and fertility senescence across all tetrapod species. The paper highlights that there is a significant lack of knowledge of demographic information even for groups that *a priori* should be well represented, and calls for the integration of wild and captive population data to provide a more comprehensive view of the demography of hundreds of species.

Hamilton, W. D. 1996. *Narrow roads of gene land: The collected papers of W. D. Hamilton*. Vol. 1. New York: Freeman.

This is a summary of contributions on evolutionary biology, including senescence, where Hamilton made the formal claim that senescence is universal.

Jones, O. R., A. Scheuerlein, R. Salguero-Gómez, et al. 2014. Diversity of ageing across the tree of life. *Nature* 505:169–173.

The authors examine the age-specific patterns of senescence on mortality risk and reproduction in eleven mammals, twelve other vertebrates, ten invertebrates, twelve vascular plants, and a green alga, showing a great deal of variation in senescence trajectories that are not explained by any of the classical theories of senescence.

Maas, F. P. M., A. J. M. Debets, B. J. Zwaan, et al. 2017. Why some fungi senesce and others do not: an evolutionary perspective on fungal senescence. In *The Evolution of Senescence in the Tree of Life*. Edited by R. Shefferson, O. Jones, and R. Salguero-Gómez. Cambridge, UK: Cambridge Univ. Press.

The authors discuss the molecular and physiological perspectives for why some fungal species do undergo senescence, while for others there is no evidence of it. This is one of the few rigorous existing treaties of senescence in fungi.

Salguero-Gómez, R., O. R. Jones, C. R. Archer, et al. 2016. COMADRE: a global database of animal demography. *Journal of Animal Ecology* 85.2:371–384.

The authors discuss taxonomic biases in the first version of this matrix population database. Forty-five percent of models were Mammals (relative to 8 percent of vertebrate animals); 6 percent were reptiles (relative to ~15 percent of vertebrates); and <1 percent were amphibians (relative to 11 percent of vertebrates). Other over- and underrepresented groups are discussed regarding their relative contributions in the overall dataset. Additional biases (e.g., temporal and geographic bias) are discussed with regard to the existing range of structural population models.

Roper, M., P. Capdevila, and R. Salguero-Gómez. 2021. Senescence: Why and where selection gradients might not decline with age. *Proceedings of the Royal Society B* 288:20210851.

The authors present the most up-to-date and taxonomic complete evidence that senescence trajectories for mortality and fertility are wildly variable across plants, algae and plant. The present conclusive evidence that taxonomic and phylogenetic correlates do not predict senescence outcomes, and instead provide a series of arguments as to why selection gradients need not decline with age for every species.

Troudet, J., P. Grandcolas, A. Blin, R. Vignes-Lebbe, and F. Legendre. 2017. Taxonomic bias in biodiversity data and societal preferences. *Scientific Reports* 7.9132:1–14.

preferences. *Scientific Reports* 7.1:1–14.

The authors evaluate widespread biases in occupancy data recorded in the Global Biodiversity Information Facility (GBIF) dataset. Birds are highly overrepresented whereas insects are highly underrepresented in the database. For most taxonomic groups, there are appreciable biases in the representation of certain groups. The authors note that certain biases are becoming increasingly severe with time, with bird records increasing and amphibian, reptile, and algal records stagnating or decreasing with time.

Zajitschek, F., S. Zajitschek, and R. Bonduriansky. 2020. Senescence in wild insects: Key questions and challenges. *Functional Ecology* 34.1:26–37.

This work focuses on the contributions of invertebrates to the evolution of senescence. The authors discuss the widespread demographic biases underlying existing demographic data, as well as the general kinds of taxonomic biases and some of the reasons. The authors discuss possible remedies for the barriers to creating more taxonomically balanced studies by focusing on solutions to applied challenges in field data collection and key questions that have yet to be resolved.

Modern Theories of Senescence—Growth Forms

The growth form of an organism can influence the resulting pattern of senescence by controlling how the risk of mortality is transmitted. Rates of senescence may be partly controlled by growth indeterminacy and growth modularity, which often overlap. *Growth indeterminacy*: Since the 1990s, a growing list of species have challenged the notion that senescence is universal. Until the mid-2000s, explanations for the nonuniversality of senescence fell into the interstices of “positive” theories of senescence discussed under Classical Theories of Senescence. For example, some have argued that certain theoretical models necessitate exponential growth to remain valid (e.g., Hamilton 1996 [cited under Data Biases]). No systematic surveys have evaluated whether these claims hold against the empirical data. In 2004, a theory emerged that provided an explanation of why demographic senescence might not to apply to certain organisms. Vaupel, et al. 2004 argues that, because reproduction increases in relation to certain traits such as size (i.e., reproductive allometry), if reproduction-correlated traits increase over time indefinitely, then reproduction increases indeterminately over time. Depending on the costs associated with age-related morphological and functional changes, natural selection could select for limitless survivorship—liberating species from positive senescence. *Growth modularity*: One of the earliest systematic arguments about growth form and ageing arises from the seminal book Finch 1990 (cited under Cellular, Physiological, and Demographic Senescence), which discusses how modularity might mediate longevity. Finch argues that species whose bauplan is comprised of not just a singular, functionally integrated form, but instead separate, highly dispersed functional systems, are less susceptible to extrinsic mortality. Organisms with this growth form could also be less susceptible to intrinsic mortality if some modules of the soma are younger than other parts of the soma. Bernard, et al. 2020 provides support for this hypothesis in plants, but not in animals. A related set of ideas about growth form and longevity comes from Gavrilov and Gavrilova 2001. These authors developed a line of research that applies a generalized systems biology concept to characterize systems with respect to maintenance costs, redundancy, and replaceability. The concept of modules as redundant, semireplaceable components fits within this framework.

Bernard, C., A. Compagnoni, and R. Salguero-Gómez. 2020. Testing Finch’s hypothesis: the role of organismal modularity on the escape from actuarial senescence. *Functional Ecology* 34.1:88–106.

This contemporary review article tests predictions of Finch’s hypothesis. This paper overviews the literature at the interface of architectural constraints and the evolution of senescence and shows that there are certain correlations between more modular species and measures of greater longevity and weaker strengths of senescence.

Gavrilov, L. A., and N. S. Gavrilova. 2001. The reliability theory of aging and longevity. *Journal of Theoretical Biology* 213.4:527–545.

This paper details the predictions of the reliability theory of senescence, coined by Gavrilov and Gavrilova, which is a systems-based approach to ageing. The authors base predictions on complex regulatory systems, rather than any one or few specific biological mechanisms for ageing and senescence.

Vaupel, J. W., A. Baudisch, M. Dölling, D. A. Roach, and J. Gampe. 2004. The case for negative senescence. *Theoretical Population Biology* 65.4:339–351.

This benchmark paper explains the first formal theory for negative senescence. The introduction and conclusion of the paper are accessible to a more general audience, but the balance of the paper delves into more detailed discussion of the mathematical implications and subcomponents of the basic model. The essential argument is that reproduction, insofar as it is tied to longevity in the soma, can promote selection for survivorship over time (i.e., negligible and negative senescence), liberating species from theories for positive senescence.

Modern Theories of Senescence—Sociality and Senescence

Studies of senescence, both empirical and theoretical, have come to diversify their approach to tackling the variation in ageing patterns across the tree of life. With the maturation of the field, sociality has emerged as an underexplored yet potentially important predictor in explaining this variation. As reviewed in Lucas and Keller 2020, sociality is likely to affect aging patterns for three non-mutually-exclusive reasons. First, sociality is likely to alter the rate of extrinsic mortality experienced by individuals, shifting the relative contributions of different age classes to future generations. Second, sociality may alter a species' pace of life, altering the rate of development and age at first reproduction. Third, reviewed more extensively in Bourke 2007, if individuals in social groups are kin, and aging patterns of mortality and fertility affect the fitness of these kin, then senescence will be under kin selection. This topic has received some theoretical attention in Lee 2003. Empirical studies are now beginning to tease apart the variation in longevity and senescence across social systems, addressing these hypotheses.

Bourke, A. F. G. 2007. Kin selection and the evolutionary theory of aging. *Annual Review of Ecology, Evolution, and Systematics* 38:103–128.

In this review, Bourke summarizes the current (at the time) field of senescence in social species, classifying work into different categories depending on the effect of ageing patterns on a focal individual and individuals with which it interacts (references within).

Lee, R. D. 2003. Rethinking the evolutionary theory of aging: Transfers, not births, shape senescence in social species. *Proceedings of the National Academy of Sciences of the United States of America*. 100.16:9637–9642.

Lee showed how the force of selection on age-specific mortality is not only proportional to remaining fecundity, as Hamilton 1996 (cited under Data Biases) displayed, but also on a "transfer" effect. The model postulated that individuals can give and receive resources throughout their lifespan that can alter their own and others' fitness. Juveniles, receiving more resources than they give out, reduce mortality risk. This explains the often-seen reduced risk of mortality with juvenile age—something the classical theories fail to explain.

Lucas, E. R., and L. Keller. 2020. The co-evolution of longevity and social life. *Functional Ecology* 34.1:76–87.

In this more recent review, Lucas and Keller extend the potential effects of sociality on ageing patterns from kin groups to general group-living species. The authors consider the bidirectional relationship between longevity and sociality, exploring ways aging patterns can affect sociality, and how sociality can affect aging patterns. The authors initially consider evolutionary explanations before integrating their arguments with hypothesized proximate mechanisms.

Modern Theories of Senescence—Proximate Mechanistic Theories

The last few decades have witnessed a greater focus on the mechanisms of demographic senescence. These are too many to cover here, and so we focus primarily on calorie restriction (CR) and mention some key additional ones. CR theory predicts that senescence

is delayed and longevity prolonged due to the ultimate effects of restricted resource intake without malnutrition (Weindruch and Walford 1988). This increase in longevity is mediated by lowering molecular oxidative damage and reducing free radical–induced cellular damage. McCay, et al. 1935 showed that restricting food intake rates in rats fed *ad libitum* resulted in a significantly extended lifespan. Weindruch and Walford 1988 provides a synthesis of the influence of food restriction on ageing and disease. Since then, CR has received a significant amount of attention with the predicted positive effects of CR observed in several species across several taxa from yeast to invertebrates and mammals. However, results of CR are not as consistent and ubiquitous as previously thought. Inconsistencies have been observed since the 1980s, with Harrison and Archer 1987 showing that genetic differences in mice result in increased and decreased longevity under CR. Subsequently, several studies have shown that the effects of CR appear inconsistent across species, including on rhesus monkeys in outcomes of two studies—one from the Wisconsin National Primate Research Center in the form of Colman, et al. 2009, and Mattison, et al. 2012 from the National Institute on Aging—showing contradicting results. Lee, et al. 2008 is based on the realization that the impact on longevity may be driven more than just by restriction of calories and the work provides new insights into the relationship of nutrition and lifespan, highlighting that nutrient content, and nutrient ratios, may be more important than caloric intake in impacting longevity. The importance of macronutrients has been tested on invertebrates but tested little in other groups. The taxonomic bias toward studying macronutrients in invertebrates rather than vertebrates is because, to some extent, it is easier to tease apart the effects of calories from macronutrients in invertebrates than vertebrates. However, Moatt, et al. 2019 teases apart these effects in the stickleback fish. CR is of course not the only mechanistic theory for senescence, with other key proposed and well-established proximate links to decreases in vitality with age. In telomere shortening, normal cell activities are compromised with every cell division, which results in the shortening of telomeres (i.e., nucleoprotein structures at the ends of eukaryotic chromosomes that are essential to protect chromosome ends from DNA degradation and enhance DNA repair activities), as shown in, for instance, Muñoz-Lorente, et al. 2019. Another key proposed mechanism is the collapse of tightly linked physiological regulatory networks that would otherwise provide bodily homeostasis, as reviewed in Cohen 2016. This idea indeed summarizes quite a few of the over three hundred theories of ageing proposed by Medvedev back in 1990 and holds promise in linking modularity (both anatomic and physiological) to the ability to compartmentalize risk (see Modern Theories of Senescence—Growth Forms). Further key proximate mechanisms for senescence in the wild are reviewed in Gaillard and Lemaître 2020.

Cohen, A. 2016. Complex systems dynamics in aging: New evidence, continuing questions. *Biogerontology* 17:205–220.

The author presents evidence that ageing is related to complex dynamics of physiological mechanisms that provide homeostasis.

Colman, R. J., R. M. Anderson, S. C. Johnson, et al. 2009. Caloric restriction delays disease onset and mortality in Rhesus monkeys. *Science* 325.5937:201–204.

This paper presents the first findings of increased longevity as a result of calorie restriction in a nonhuman primate, thereby filling a gap in aging research.

Gaillard, J-M., and J-F. Lemaître, eds. 2020. An integrative view of senescence in nature. In *Special issue*: *Functional Ecology* 34.1:4–16.

This is the editorial in a recent special feature on senescence, which includes some of the most cutting-edge research on proximate mechanisms for senescence in animals and plants.

Harrison, D. E., and J. R. Archer. 1987. Genetic differences in effects of food restriction on aging mice. *The Journal of Nutrition* 117.2:376–382

This paper tested the predictions of calorie restriction on genetically different mice using the same set of experiments. The authors' key finding was that genetic differences can impact the effects of food restriction; two genotypes showed longevity benefits of calorie restriction while one showed reduced longevity, highlighting the impact that genetic diversity can have on any beneficial effects of calorie restriction.

Lee, K. P., S. J. Simpson, F. J. Clissold, et al. 2008. Lifespan and reproduction in *Drosophila*: New insights from nutritional geometry. *Proceedings of the National Academy of Sciences* 105.7:2498–2503.

The paper uses the geometric-framework approach to address several issues that hampered the ability to identify the roles of energy and specific macronutrients in ageing. The authors are able to partition the effects of macronutrients and energy and show that flies are able to regulate protein and carbohydrate intake separately and select a diet composition that maximizes their fitness.

Mattison, J. A., G. S. Roth, T. M. Beasley, et al. 2012. Impact of calorie restriction on health and survival in rhesus monkeys: The NIA study. *Nature* 489.7415:318–321.

This paper presents findings that contradict earlier findings in Colman, et al. 2009 that calorie restriction increases longevity in a nonhuman primate (the rhesus monkey), thereby calling into question the benefits of calorie restriction in such primates. The authors highlight that the different approaches in study design, husbandry, and diet composition taken in their study, compared to the previous study Colman, et al. 2009, strongly impacted the effect of calorie restriction.

McCay, C. M., M. F. Crowell, and L. A. Maynard. 1935. The effect of retarded growth upon the length of life span and upon the ultimate body size: One figure. *The Journal of Nutrition* 10.1:63–79.

This paper is the first well-executed study clearly showing that restricted food intake extended longevity in rats. Several studies before this also claimed to show extended longevity when food intake was restricted; however the outcomes were not considered to be reliable.

Medvedev, Z. 1990. An attempt to rational classification of theories of ageing. *Biological Reviews* 65.3:375–398.

The author provides an exhaustive classification of most existing theories of senescence based on ageing, damage, genetic programming, tissue-specific ageing, mathematical modeling, and evolutionary theories.

Moatt, J. P., M. A. Fyfe, E. Heap, et al. 2019. Reconciling nutritional geometry with classical dietary restriction: Effects of nutrient intake, not calories, on survival and reproduction. *Aging Cell* 18.1:e12868.

This paper is one of the few that is able to separate the effects of calories and nutrients in a vertebrate species (stickleback fish). The authors use the geometric-framework of nutrition approach along with the standard approach of calorie restriction which allows the authors to make a comparison between the two. The findings show that macronutrient intake rather than calories determines lifespan in the stickleback fish.

Moatt, J. P., E. Savola, J. C. Regan, D. H. Nussey, and C. A. Walling. 2020. Lifespan extension via dietary restriction: Time to reconsider the evolutionary mechanisms?. *BioEssays* 42.8:1900241.

This paper describes the current evolutionary explanations of the lifespan response to dietary restriction and provides a synthesis of the application of the theories to the emerging evidence on the importance of macronutrients rather than calories. The authors also highlight that, as the terms dietary restriction and calorie restriction were largely interpreted as synonymous terms, the predominant evolutionary explanations of the dietary restriction effect were developed to explain responses to calorie restriction and not macronutrient availability.

Muñoz-Lorente, M. A., A. C. Cano-Martin, and M. A. Blasco. 2019. Mice with hyper-long telomeres show less metabolic aging and longer lifespans. *Nature Communications* 10.4723.

The authors demonstrate a direct link between telomere length and rate of actuarial senescence and longevity in mice.

Weindruch, R., and R. L. Walford. 1988. *The retardation of aging and disease by dietary restriction*. Springfield, IL: Charles C Thomas.

This book is the first comprehensive review of the evidence of the benefits to longevity of calorie restriction. Also highlighted and discussed in this work are the various hypotheses at the time that may explain the benefits of calorie restriction. Broad claims about the benefits of calorie restriction on life expectancy are made in this paper.

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