

1 Introduction: Wilting Leaves and Rotting Branches

Reconciling Evolutionary Perspectives on Senescence

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*It don't make no difference
'cos I ain't gonna be, easy, easy,
the only time I'm gonna be easy's when I'm
Killed by death* — Motörhead, Killed By Death (1984)

Short Summary

We humans have long wondered about the seemingly inevitable physiological decline that happens after our maturity. This phenomenon, known as ‘senescence’, is recognised as the physiological deterioration that results in increasing age-specific mortality or decreasing age-specific fertility at or beyond some age in maturity. But is this phenomenon universal, and is it always the result of the same processes? Although evolutionary theories of ageing exist that suggest that senescence should be universal, empirical data increasingly suggest that senescence may not necessarily be a ubiquitous feature among multicellular organisms, and where it does occur, it is not clear that it is always the result of the same ultimate or proximate mechanisms. In this contributed book, some of the leading scientists in ageing research offer an in-depth, updated understanding of the mechanisms behind senescence, using cutting-edge approaches and species representing a wide evolutionary diversity of life.

Introduction

We are all aware of the march of time in our lives. We are born, we grow, we (may) reproduce, we advance in age and we die. So enveloping is our awareness of this phenomenon that, at least in the English language, we do not differentiate between ageing as the progress of time (i.e. chronological ageing) and senescence, or the decline in physiological well-being that begins at some point at or beyond our age at reproductive maturity. In this sense, it is often assumed that senescence is such an unshakeable

part of ageing that it is universal – no organism can escape from it, except by dying prematurely from an exogenous factor (e.g. a wild fire, a ferocious predator).

But is senescence truly universal? While ageing is certainly universal in the sense that we cannot escape the march of time, senescence itself is probably not. The test for the universality of senescence is fairly straightforward: there must not exist a single species in which physiological decline with age cannot be documented. A species that escapes from senescence would exhibit the lack of such decline, referred to as ‘negligible senescence’ (Vaupel et al. 2004), or perhaps even physiological improvement with age (i.e. ‘negative senescence’, per Vaupel et al. 2004). Recent analyses suggest that such species exist (Baudisch et al. 2013; Garcia et al. 2011; Jones et al. 2014). However, over much of its history, the study of senescence has been strongly taxonomically biased, focusing heavily on humans, other mammals and birds, and a small number of model organisms including *Drosophila* spp. and *Caenorhabditis elegans*, among others.

This book represents an overdue attempt to examine the evolutionary consequences and mechanisms of senescence in organisms across the Tree of Life and to assess whether senescence is truly universal and why. We paint an evolutionarily broad picture in order to relate what we know to the world’s biodiversity. This is a long-overdue attempt because, since the first evolutionary theory of senescence was advanced by August Weismann over a century ago (Weismann 1892, 1893), we have only recently managed to accumulate sufficient genetic, physiological and demographic data on a diverse enough group of organisms to begin to address this topic rigorously. Biologists studying senescence in different groups of organisms have tended to use disparate methods, met at sub-discipline-specific scientific meetings, and have established their own distinct traditions and terminology. We have attempted to bring these traditions together to look for common trends and answers. For this purpose, this book is divided into a first part focused on the general evolutionary theory of ageing, followed by parts focused on animals (naturally, including humans), plants and microbes. We also keep to some common definitions throughout the book. Most importantly, we and all contributing authors strictly define ‘senescence’ as the process of physiological or biological decay leading to increasing mortality rates and/or decreasing fertility rates with age; we distinguish this from ‘ageing’, which here is viewed simply as the march of time, with no physiological decay implied.

A Short History of the Senescing Universe

The modern evolutionary theory of senescence is rooted in the late nineteenth century. August Weismann postulated that an organism’s cell lines can be decomposed into a germ line and a soma (Weismann 1892, 1893). The germ line constitutes the cells responsible for the organism’s reproduction. The soma constitutes cell lineages created to keep the germ line alive and reproducing. While all cells are ‘born’ and die, the line of germ cells is potentially immortal, and somatic lineages always die when the organism itself dies – but we do note that the picture gets rather quickly complicated in organisms with clonal abilities (see Chapters 11, 13, and 15 to 17). The corollary of Weismann’s

work is that the soma exists to maintain the germ line and that the soma is essentially disposable (Kirkwood 1977). In essence, Weismann suggested that the physiological performance of the soma declines with age by interacting with the environment and buffering the germ line from it. Remarkably, he even briefly supposed that death itself was adaptive because, via death, senescing individuals would alleviate competition for younger individuals with greater reproductive potential, although he quickly abandoned this idea, recognising its inherent flaws.

Weismann's germ-soma theory was profoundly influential and continues to inspire researchers to this day. However, his theory predicts only that senescence will occur in all organisms with a strict germ/soma separation and so cannot account for senescence observed in unicellular life, plants, fungi, some animals such as corals, and many microbes. Comfort (1964) even criticised the theory as being somewhat circular for assuming what it is supposed to explain and therefore not really explaining why senescence may evolve in the first place. In addition, Comfort argued that it does not truly relate any particular genetic, physiological or demographic processes to fitness. Although more mechanical, physiological views of senescence most certainly exist (Comfort 1964), some have suggested that the ways in which senescence is thought to evolve preclude any common mechanism of decline (Silvertown 2013).

The mathematical foundations for a synthetic evolutionary theory of senescence were laid by Ronald Fisher. He argued that senescence was likely a result of the accumulation of deleterious age-specific traits that could not be effectively removed by natural selection. The strength of natural selection was related to the reproductive value of a particular age, the remaining number of offspring an individual could expect to produce before death (Fisher 1930). John B. S. Haldane had a similar notion about a decade later (Haldane 1941), but it was Peter Medawar (1952) who succinctly emphasised the consequences for senescence in his essay 'An Unsolved Problem of Biology', where he remarked that, after sexual maturity, '[t]he force of natural selection weakens with increasing age – even in a theoretically immortal population, provided only that it is exposed to real hazards of mortality. If a genetic disaster . . . happens late enough in individual life, its consequences may be completely unimportant.' George Williams extended this argument by pointing out that in addition to the accumulation of deleterious mutations late in life, senescence could also be caused by pleiotropic genetic effects that are positive in early life but negative in late life (Medawar 1952; Williams 1957; see review in Ljubuncic & Reznick 2009). These mechanisms for the evolution of senescence – mutation accumulation and antagonistic pleiotropy – are also potentially universal, but at the time that they were developed, once again, little to no data actually existed on senescence outside humans. Medawar and others even assumed that animals could not live long enough in the wild to experience senescence (Medawar 1952), an assumption that we know now to be false (Jones et al. 2008; Nussey et al. 2013; see also Chapter 7).

Until relatively recently, senescence was viewed as a universal phenomenon, and for senescence to be truly universal, an evolutionary mechanism for its universality must explain genetic, physiological and demographic patterns throughout the life span in all

lineages across the tree of life. In 1966, the *Journal of Theoretical Biology* published a paper in which William Hamilton laid out the mathematics behind a theory, drawing on the ideas of Williams and Medawar, leading to the outcome that ‘senescence is an inevitable outcome of evolution’ (Hamilton 1966). Though others had certainly had a large impact on the development of the field, this was the first seemingly testable theory to explain the evolution of ageing as a universal. Fortunately, it was developed at a time when technological advances (Holliday 1990; Medina 2005; Rabinow 1997) facilitated studies of the underlying genetics (e.g. Hoffman et al. 2014; Moorad & Promislow 2011) and physiology of senescence (e.g. Kumar et al. 2012). It was also done at a time when genetic perspectives began to revolutionise evolutionary biology (Dawkins 1978). Therefore, beginning around the start of the 1970s, senescence research truly started broadening its evolutionary scope.

Since Hamilton’s work, the most prominent theory for the evolution of senescence has been Thomas Kirkwood’s ‘disposable soma theory’ (Kirkwood 1977; see also Chapter 2). According to this theory, an organism’s cells accumulate deleterious mutations with age, and repairing this damage is costly. Since the soma functions merely as a vessel for ensuring the replication/continuation of the immortal germ line, mutations in soma cells are less important to repair than those in the germ line, which are carefully protected to ensure accurate replication from generation to generation. Therefore, his theory suggests that the evolutionary stable strategy (ESS) of the trade-off between resource allocation to processes of somatic maintenance and maintenance of the germ line inescapably favours the germ line, resulting in senescence.

It is noteworthy that these evolutionary theories were developed seemingly with mostly mammals and birds in mind. Other organisms have presented more perplexing mysteries for the development of a universal evolutionary theory of senescence, particularly long-lived plants such as the redwood but also fungi and other microbes. Indeed, although Hamilton considered mortality patterns in extremely long-lived plants (Hamilton 1966), it is doubtful that he accessed the data available on some longer-lived tree species that contemporary evolutionary plant ecologists were working with (Harper 1967; Harper & White 1974).

Although some of the most important work in evolutionary biology historically has been done on plants (Clausen et al. 1947), plant life histories themselves were not the subject of truly rigorous study until John Harper invigorated the subject with his classic book that inspired a generation of plant ecologists (Harper 1977). From then on, we have witnessed the gradual development of the field of plant senescence biology (e.g. Roach 1993; Silvertown et al. 2001). Most recently the subject has even covered a special issue of a major journal focused on the ecological consequences and evolutionary origin of or escape from senescence in plants (Salguero-Gómez et al. 2013). Other groups of organisms, particularly microbes, have only recently become major targets of research, mostly due to the much more recent development of methods to enable experimentation on them and to basic challenges in defining what the *individual* is in many organisms (e.g. hyphae of some fungal species may contain many different nuclei (Roper et al. 2011); some forests of quaking aspen (*Populus tremuloides*) are in fact a huge clone of a single individual (DeWoody et al. 2008)).

The State of the Science and Further Challenges

Alex Comfort's classic book on senescence, now over fifty years old, summarised the state of the evolutionary study of senescence as lacking the scientific rigor seen in other disciplines (Comfort 1964). He criticised the field for yielding a number of evolutionary theories but failing to generate sufficient data to test those theories. Implicit in this criticism was the acknowledgement that biologists used a range of incompatible definitions of senescence and that the majority of research was too narrowly focused on humans to evaluate the universality of senescence.

We have come some way since Comfort (1964). We have documented demographic patterns across the life course in a growing number of organisms and have finally produced some comparative research suggesting that senescence may not be universal after all (Jones et al. 2014). However, even Comfort himself with his survey of the limited data available in the 1960s suggested that senescence was unlikely to be universal and that the mechanisms for its evolution may differ across species (Comfort 1964). Yet, he also felt that senescence was a relatively rare phenomenon among species, particularly restricted to some, but not all, metazoa, and that life in the wild was harsh enough to prevent most species from reaching senescent ages.

In many cases, we know now that a lack of senescence is likely a real phenomenon in some groups rather than simply a case of inadequate or insufficient data. It was in the 1990s that Caleb Finch (1990, 1998) gave serious consideration to organisms that exhibit 'negligible senescence' and experience no, or only very small, increases in mortality rate with age. Finch noted that high-quality demographic data were lacking for most species at the time, but his contenders with supporting evidence included sexually reproducing species known to reach advanced age, such as the trees bristlecone pine (*Pinus longaeva*) and yew (*Taxus baccata*), lobsters (e.g. *Homarus* spp.), bivalves such as the quahog (*Arctica islandica*), marine fish including rockfish (*Sebastes* spp.) and halibut (*Hippoglossus* spp.) and the Testudinidae (tortoises) (Finch 1990, 1998). Although we still lack high-quality data on most of these groups, this mode of senescence has recently been confirmed in *Hydra* (Schaible et al. 2015; see also Chapter 12).

As far as we have come in the field of senescence biology, we find nonetheless that a number of important challenges and opportunities exist for further research. We would like to outline five particularly important and promising venues of research, both theoretical and experimental, to develop a unifying theory of the evolution of and escape from senescence.

1. Meaning and Mechanisms

First and, far from sounding trivial, most importantly, we find a great variety of interpretation in the meaning of and assumed mechanisms behind senescence. For some, senescence is unrepaired wear and tear similar to the eventual breakdown of a mechanical system (Comfort 1964). This definition implies that the environment drives senescence and that senescence is caused by unavoidable physical and chemical

processes. In contrast, others view senescence as the natural consequence of the accumulation of genes expressed later in life that are detrimental to health. This view implies that senescence is not fundamentally driven by physical wear and tear but rather by intrinsic processes regulated by the genome and patterns in gene expression (Lundberg et al. 2000). Regardless, senescence is most often interpreted scientifically as actuarial senescence, in which age-specific mortality is measured at the population level, while others argue that other forms of senescence, such as reproductive and physiological senescence, may be just as important to consider and perhaps even easier to measure (see Chapter 7).

Ultimately, many of us are interested in senescence because of our own awareness of mortality and our hope that understanding senescence can lead to measures to counter it. However, a less self-centred perspective deserves attention here too: ecology and evolution attempt to understand variation, and why some organisms senesce and others seem to escape from senescence is a fundamental question for how the world works, aside from our own hopes and aspirations to live forever. The wear-and-tear view of its nature has led to research on physiochemical processes that lead to decay, such as oxidative stress (Møller 2007), while the genetic view has led to research on the genes driving physiological decline and increased mortality risk, such as cancer genes (Itahana et al. 2004). These are radically different views of senescence, and they need to be reconciled for a comprehensive evolutionary theory to explain the phenomenon.

2. The Confounding Impact of Life History

Some taxa have proven to be particularly challenging to study demographically, mostly because (1) they are long-lived, (2) their age-related demographic patterns are highly variable or (3) the individual is difficult to define and identify (Abrahamson 1980; see also Chapters 17 and 19). While it may seem strange that life span should be a confounding variable, the life histories of the longest-lived organisms are also among the most difficult to study. Long life spans typically require study commitments that fall outside the horizons of modern-day PhD dissertations and funding agencies and can even be challenged by the life span of the experimenter (e.g. the herbaceous perennial plant *Borძerea pyrenaica*, which can live 300 years) (Garcia et al. 2011). Individuals with long life spans also have a tendency to be either fairly large or to have requirements for growth that prevent experimentation. Both of these challenges would be evident for scientists working with some of the world's long-lived mammals, such as whales and elephants, which are logistically challenging to study in wild conditions and in terms of following a pedigree over several generations, as would be required for a thorough experimental study on the micro-evolutionary context of senescence (though there are some notable exceptions, e.g. Foote 2008; Foster et al. 2012; Hayward et al. 2014). The latter problem is also evident in many long-lived herbaceous perennials, which have stringent germination requirements, long juvenile periods often measuring decades and cryptic life history stages (Gremer et al. 2010; Rasmussen et al. 2015; Shefferson 2009). A corollary of these problems is that they usually prevent the inclusion of the large cohort and sample sizes necessary to ensure sufficient statistical power

to precisely measure traits at advanced (perhaps senescent) ages. For example, in what will no doubt be a classic study on plant senescence, Roach et al. (2009) planted over 30,000 seedlings of the short-lived perennial *Plantago lanceolata* in order to keep statistical power high for those living to old age, finding that over 90 per cent had died by age ten (Shefferson and Roach 2012). An important limitation in the study of senescence in herbs is also that size and age are often decoupled (Salguero-Gómez & Casper 2010) and that anatomic legacies of age (e.g. growth rings) are often difficult to assess (but see Schweingruber & Poschold 2005). Luckily, some new analytical developments may remedy these limitations (Colchero & Schaible 2014).

Clonal plants, clonal animals and hyphal fungi have proven particularly challenging because of their modularity and continual growth, which create strongly size-based patterns in mortality and fecundity that override age-based patterns and create inherent difficulties in delineating individuals (Bierzychudek 1982; Salguero-Gómez et al. 2013). Modularity in plants and clonal animals creates a role for genetic mosaicism in promoting individual fitness, where seemingly ‘somatic’ mutations are nonetheless propagated to future generations because of the lack of a clear germ-soma distinction in clonally growing structures (Gill et al. 1995; see also Chapter 11). This phenomenon is taken to a whole new level in many glomeromycotan, ascomycetous, and basidiomycetous hyphal fungi, which often exchange and keep multiple different nuclei within their physiologically ‘individual’ but genetically diverse mycelia (Roper et al. 2011). These challenges are compounded with empirical difficulties, such as unobservable life stages including vegetative dormancy (Shefferson 2009; Tuomi et al. 2013), or migration (Barthold et al. 2016) and the difficulty in detecting microscopic individuals of fungi in the environment (see Chapter 17).

It is only recently that we have seen the growth of research programmes on senescence in these clonal organisms (Jones et al. 2014; Salguero-Gómez et al. 2013). Yet, pioneering theoretical work suggests that clonal indeterminate growers are precisely the organisms in which we need to understand senescence most, because they are the most likely to have escaped from it altogether (Bidder 1932; Vaupel et al. 2004). Amazingly, not only do we still need to better resolve the difficulties in analysing the demography of clonal plants, clonal animals, and fungi, but we also need to agree on a definition of fitness that can allow further study (although some theory currently exists) (see Orive 1995). In the latter vein, evolutionary biology’s heavy reliance on short-term fitness metrics may confound comparisons between short-lived organisms and long-lived organisms with overlapping generations, such as most – if not all – perennial plant species.

3. Controlled Conditions versus the Wild

A further challenge lies in the collection of empirical data on senescence. Analyses of observational, wild-collected data rarely seem to match predictions originating from classical evolutionary theory, except perhaps in large animals (Bronikowski & Promislow 2005; Jones et al. 2014; Reznick et al. 2004;). In long-lived plants, for example, observational approaches have certainly yielded many interesting patterns

that yield far more questions than answers (Garcia et al. 2011; Jones et al. 2014; Martínez 1998). In this book, some authors argue that the wild is not the place to look for senescence; rather, a controlled and safe environment in which organisms actually have the potential to age even perhaps beyond reproduction is necessary to observe predicted patterns (see Chapter 3). Others here argue that wild-collected data often show evidence of senescence (see Chapter 7), and the real challenge is not to test the theory under controlled conditions but instead to build a theory that incorporates ecological context better (see Chapters 6, 15, 17 and Chapter 19). A further viewpoint notes that many animal species tend to show senescence even in the wild (Nussey et al. 2013; Reznick et al. 2006) but that experimentation in controlled conditions may yield more consistent results (see Chapter 9). We argue that both controlled experiments and in situ observational approaches need to be pursued simply because there is so little agreement on how senescence actually evolves.

4. The Role of Environment

We further argue that the role of the environment in senescence may be more complicated than is currently acknowledged. Plant and wildlife demographers, who are used to having access to long-term, high-resolution data with pedigree and causes of mortality, generally view mortality risk as being derived from either intrinsic (e.g. disease genes) or extrinsic (e.g. climatic catastrophes) sources (e.g. Abrams 1993; Ricklefs 2000). In reality, we argue, the separation of intrinsic versus extrinsic causes of mortality is more complicated than this dichotomy, and these are more likely to take place as interactions rather than as simple additive effects. For example, a tree that has been attacked by a fungus is more likely to succumb to an ‘internal’ cause of mortality, whatever that may be, than an un-attacked tree is likely to succumb to that same internal cause of mortality.

Evolutionary demographers have been strongly influenced by W. Hamilton, who viewed senescence as primarily an intrinsically driven phenomenon caused by the cumulative impact of deleterious genes (Hamilton 1966), though with the ability to evolve depending on patterns in age-specific extrinsic mortality (Caswell 2007; see also Chapters 4 and 9). Intrinsic factors can make individuals more susceptible to extrinsic sources of mortality. However, evolutionary biologists have long noted that many genes exhibit differential expression patterns under different environmental contexts, a phenomenon known as ‘phenotypic plasticity’ (West-Eberhard 2003). Because trait expression depends on cellular sensing of differentially expressed or concentrated gene products in the immediate environment, many biologists now argue that most, if not all, traits are actually plastic (Pigliucci 2005; West-Eberhard 2003). In evolutionary demography, a small subset of researchers has similarly asked if patterns of senescence might be strongly driven by both intrinsic and extrinsic factors and has suggested useful frameworks for investigating the dependence of senescence-related patterns in mortality, fertility and physiological traits as a function of environmental variation (Hammers et al. 2012; Koons et al. 2014; Ricklefs 2000; Shefferson & Roach 2013).

Along similar lines, some authors have noted that senescence is strongly influenced by ecological context. For example, predation can shape senescence by imposing selection against the senescent, provided that the organisms are still able to reproduce and that predation is higher upon older-aged individuals (see Chapter 9). Predation and other causes of mortality also continually weed out the weakest individuals from the population, yielding a ‘disappearing fraction’, or heterogeneity effect (Vaupel & Yashin 1985), that removes senescent individuals and may result in the measurement of senescence-related traits in only the most physiologically fit individuals (Bennington & McGraw 1995; Nussey et al. 2011; see also Chapter 8).

Among the most important consequences of the relatively poor consideration of the complexity of sources of mortality and environmental influences in general is the somewhat simplistic view of trade-offs in much of the evolutionary literature on senescence. Trade-offs are often thought of as dichotomous splits in resource allocation between two traits, but the truth is far more complicated. Though trade-offs are always assumed to operate and so prevent the evolution of Darwinian demons (i.e. organisms with infinite fitness, per Law 1979), environmental influences on the expression of related traits can lead to trade-offs disappearing altogether or becoming positive relationships (Reznick et al. 2000; Spitze 1991). Trade-offs are further complicated by the order of resource allocation among traits (see Chapter 5), which can lead to many traits becoming positively related (de Jong and van Noordwijk 1992); by relationships among traits via commonly used gene expression pathways (Stearns and Magwene 2003); and by the influence of past trade-offs (Stearns 1992). Such complexities are not formally included in any model of evolutionary senescence, and indeed the most commonly used models posit dichotomous relationships between fitness components such as survival and reproduction that may or may not hold in much of the Tree of Life.

With the need for a better understanding of the influence of ecological context on senescence, we argue that the distinction between intrinsic and extrinsic mortality in senescence is not helpful and indeed that many factors typically considered to be one or the other actually have elements of both (see Chapter 6). The field may grow most productively through its incorporation of viewpoints and advances in the fields of epigenetics and developmental biology (Hoffman et al. 2014; Zhu et al. 2015), as well as the inclusion of ecological context (see also Chapters 6 and 9), in a rigorous theoretical framework that can yield strong, testable predictions. It would also grow through the inclusion of further ‘forensic’ data into demographic analysis, for example, on the actual causes of mortality. The latter is something that has allowed studies on human senescence to make important contributions and that studies of the rest of the animal and plant kingdoms could do with.

5. A Robust Phylogenetic Comparative Framework

Although even some of the very earliest work on senescence had a comparative component, a lack of rate-of-ageing data (as opposed to maximum-life-span data) for many groups and the nascent state of phylogenetic comparative methods itself hampered analyses until around fifty years ago. The macro-evolutionary tradition in senescence

biology goes back longer, being influenced by the development of comparative demography primarily in the United Kingdom and the foundation of formal phylogenetic analysis in the 1950s and 1960s with Willi Hennig's seminal contributions (Austad & Fischer 1991; Botkin & Miller 1974; Franco & Silvertown 1997; Grime 1974; Hennig 1966; Lack 1954; Pianka 1970). The first analyses made use of available data, which included sparse coverage of the Tree of Life. Over time, these analyses have generally found support for the idea that senescence varies across organisms, with the most sophisticated recent analyses suggesting that existing theory does not account for the diversity of mortality and fecundity patterns in the world's organisms (e.g. Jones et al. 2014).

Now that we know that senescence varies across the Tree of Life, we need to know how (what are its predictors?) and why (what are its mechanisms for and against it?). We have already made the case for expanding existing theory to account for ecological context, which likely causes severe deviations from expectations in at least some cases (see also Chapters 5, 6 and 8). Evolutionary history may also be relevant to understanding patterns in senescence, particularly since the physiology involved in senescence, or its escape, is probably based on genetic predispositions that are themselves shared by closely related lineages. Do sister species senesce (or not) in similar ways?

Phylogenetic hypotheses have not been explored in any systematic way within the field of senescence biology. However, some clear hypotheses are suggested, even by the dominant theories of senescence evolution. For example, if senescence is the result of a declining force of natural selection with age (Hamilton 1966; Medawar 1952), then age-related mortality and fecundity patterns, and the physiological traits that lead to senescence, may evolve slowly over macro-evolutionary time in ways similar to Brownian motion models of evolution. If so, senescence may exhibit phylogenetic signal, which would lead to a very specific phylogenetic pattern: more closely related species should exhibit more similar demographic patterns than more distantly related ones (Blomberg et al. 2003). Indeed, some groups may be particularly prone to a particular physiological path in senescence, just as some orchid lineages are genetically predisposed to mutate into non-photosynthetic plants, even within otherwise photosynthetic populations (Julou et al. 2005; Motomura et al. 2010). Further analyses can also lead to a better understanding of the evolution of specific demographic patterns and senescence-related traits via phylogenetic contrasts (Felsenstein 1985). For example, although some have suggested that clonal plants may escape senescence because clonality may yield particularly strong benefits to fitness under some circumstances (Vaupel et al. 2004), as far as we know, a systematic macro-evolutionary comparison between sister plant species differing in this trait has never been performed, even though large data sets exist that may allow such analyses (Klimešová & De Bello 2009; Salguero-Gómez et al. 2015).

Phylogenetic analyses are under-utilised in senescence biology and can advance our understanding of senescence immeasurably. However, they require a perspective that can fit the different paradigms of evolutionary demography and phylogenetics together meaningfully. Importantly, as phylogenetics has developed into a strongly quantitative and computationally intensive field, it has developed rigorous tests of assumptions