Functional Ecology | BRITISH ECOLOGICAL SOCIETY

Testing Finch's hypothesis: The role of organismal modularity on the escape from actuarial senescence

Connor Bernard[1] | Aldo Compagnoni[2] | Roberto Salguero-Gómez[1,3,4]

[1]Department of Zoology, University of Oxford, Oxford, UK

[2]Martin Luther University Halle-Wittenberg, German Centre for Integrative Biodiversity Research, Leipzig, Germany

[3]Centre for Biodiversity and Conservation Science, University of Queensland, St. Lucia, QLD, Australia

[4]Evolutionary Demography laboratory, Max Plank Institute for Demographic Research, Rostock, Germany

Correspondence
Connor Bernard
Email: connor.bernard@zoo.ox.ax.uk

Abstract

1. Until recently, senescence was assumed to be a universal phenomenon. Evolutionary theories of senescence predict that no organism may escape the physiological decline that results in an increase in mortality risk and/or decline in fertility with age. However, evidence both in animals and plants has emerged in the last decade defying such predictions. Researchers are currently seeking mechanistic explanations for the observed variation in ageing trajectories.

2. We argue that the historical view on the inevitability of senescence is due, in part, to the development of its classical theories, which targeted primarily unitary organisms. In unitary species, the integration of resources and functions is high, and adult size is determined. In contrast, the architecture of modular organisms is indeterminate and built upon repeated modules. The isolation of mortality risk in species like hydra (*Hydra* spp.) or creosote brush *Larrea tridentata* may explain their null or even negative senescence.

3. Caleb Finch hypothesized three decades ago that species with the ability to compartmentalize risk may escape senescence. Here, we first review the evidence on organisms that slow down or even avoid senescence in the context of their architecture, along a continuum of unitarity-modularity. Then, we use open-access databases to comparatively analyse various moments of senescence and link longevity to the degree of anatomical modularity. Our analysis compares the pace of senescence across 138 plants and 151 animals, and the shape of senescence across a subset of these. Our comparative analysis reveals that plant species that are more modular do indeed tend to escape from senescence more often than those that are unitary. The role of modularity in animal senescence is less clear.

4. In light of novel support for Finch's hypothesis across a large diversity of plant species, and with less conclusive findings in animals, we identify new research directions. We highlight the opportunities related to age-dependent mortality factors. Other areas for further research include the role of modularity in relation to endocrine actions, and the costs of modular anatomies.

KEYWORDS
ageing, BIEN database, cavitation, COMADRE animal matrix database, COMPADRE plant matrix database, Keyfitz's entropy, life table, matrix population model

88 | | wileyonlinelibrary.com/journal/fec | *Functional Ecology*. 2020;34:88–106.

1 | THE EVITABILITY OF SENESCENCE

The actinozooid is a living thing which knows no time of youthful vigour, no waxing to a period of adult life, no waning to senility – it knows no age – it practically knows no natural death. *Wood-Jones (1912)*

Since the late 1970s, three major theories have persisted to explain the declines in physiological performance with age, that is *senescence*. These are: (a) Medawar's (1952) theory of mutation accumulation, whereby organisms senesce due to their inability to revert the rate of accumulation of deleterious mutations; (b) Williams' (1957) theory of antagonistic pleiotropy, whereby genes with dual effects (with positive effects of fitness early in life but negative late in life) are selected because 'the young matter more than the old'; and (c) Kirkwood's (1977) disposable soma theory, whereby organisms with a clear physical separation between germ and soma lines senesce because natural selection selects the germ over the maintenance of the soma. Medawar's and Williams' theories centre on genetic principles for a discounting of late-life performance (Hamilton, 1966; Le Bourg, 2001; Promislow, Tatar, Pletcher, & Carey, 1999). Williams' and Kirkwood's theories share ground in staking their respective claims on ties between fundamental biological constituents. These two theories identify links between between-genes' actions (i.e. early vs. late) and between-functional allocations (i.e. maintenance vs. reproduction), respectively (Kirkwood & Austad, 2000; Kirkwood & Rose, 1991; Rose, 1991; Turke, 2008). The three classical theories of senescence aim to identify the forces responsible for shaping patterns of senescence. However, they largely fail to explain how or under what conditions senescence could be postponed or even reverted.

In the decades since the classical theories for senescence were posited, research has qualified the apparent inescapability of senescence. For example, Hamilton (1966) asserted that actuarial senescence necessarily follows from the exponential growth of population. He mathematically showed that organisms with demographies highly favourable to late-life performance still experience selection bias for early life performance. Implied here is that populations with non-exponential growth could escape senescence. Williams (1957) described senescence as a transient genetic phenomenon in which genes that present mixed benefits are partially substituted by more adaptive genotypes. According to this theory, variation in the genes and in their influence on one another can generate positive, negligible and negative senescence. Negative senescence describes a decline in mortality after maturity (sensu Vaupel, 2004).

More recent models of senescence describe how density dependence can accelerate or decelerate the senescence as a function of stage-dependent mortality (Abrams, 1993). In addition to these models, an important distinction about senescence has been made with respect to cell differentiation. This was implied by Williams' work and has been highlighted by a number of other researchers (Buss, 1987; Gómez, 2010; Shefferson, Jones, & Salguero-Gómez, 2017; Turke, 2008). Organisms with undifferentiated cells (thus without soma-germ separation) may not be subject to patterns of senescence assumed under all classical theories for senescence. Surprisingly, the existence of alternative explanations for escaping senescence did not detract from Hamilton's (1966) prediction of universal senescence until recently (Baudisch, 2005; Nussey, Froy, Lemaitre, & Gaillard, 2013).

A systematic review or estimate for how many taxa are accountable to the above-mentioned exceptions does not exist, that we are aware of. We also do not know how the closely proposed mechanisms that release species from senescence relate to particular groups of species. This information can advance our understanding of how much has been explained under existing theory, and can focus future studies. While the conceptual foundations of these exceptions are strong, they have rarely been tested empirically. For example, Vaupel, Baudisch, Dölling, Roach, and Gampe (2004) offer a cogent proof of negative senescence based on the growth-related functional performance. They ground theoretical advancements with examples of species that exhibit negligible or negative senescence that are also indeterminate in their growth. Until recently, we were not aware of any work connecting how closely indeterminate growth predicts negligible senescence, nor analysing the existence of confounding influences.

Taxonomic bias in early studies of senescence in unitary organisms (e.g. mammals, birds) likely contributed to the notion that senescence is universal (Jones et al., 2014; Monaghan, Charmantier, Nussey, & Ricklefs, 2008; Shefferson et al., 2017). The idea of universal senescence may also be attractive because cellular ageing, not far afield of senescence, is written about in universal terms (i.e. cellular senescence; noted by Comfort, 1979). Statements in recent publications put little distance between cell and organismal finitude: 'Organisms wear out, just like machines' (Ricklefs, 2008); '[D]amage will accumulate in parallel with cells' (Kirkwood, 2005); 'Ageing and its associated decline in survival and reproductive fitness is an inherent feature of biological systems' (Alper, Bronikowski, & Harper, 2015). Studies on ageing focus on a set of cellular and molecular processes that are distinct from those that are the focus of senescence. Ageing processes include telomere shortening (Young, 2018); oxidative stress (Monaghan, Metcalfe, & Torres, 2009); double-strand break (White & Vijg, 2016); and related mechanisms underlying the damage or error theories of ageing (Jin, 2010; reviewed in connection with senescence by Comfort, 1979; Petralia, Mattson, & Yao, 2014). Insofar as variations in the ageing processes at the molecular level influence the types and rates of age-dependent physiological degradation, they may be an important dimension to the discourse on senescence.

The extent to which ageing processes should be treated independently of senescence, evolutionarily and mechanistically, is contested. The distinction between the two relates to a distinction in evolutionary theory related to evolutionary constraints (Wensink, Caswell, & Baudisch, 2017; Wrycza, Missov, & Baudisch, 2015). The approach we use, based on Baudisch (2011), negotiates this conflict by separating two central axes of variation – between pace and shape. This approach enables a clean differentiation between the patterns of life expectancy and mortality patterns.

We argue that the inevitability of organismal senescence should hold only so far as molecular declines upscale to the demography of the organism. This upscaling – from cellular degradations to physiological injury – must go through the anatomy (i.e. tissues) and physiology (i.e. organs) of the individual. Considering this upscaling of cellular declines to physiological declines may inform the mechanisms for relieving senescent forces of natural selection.

Recent research has highlighted an important aspect that was absent from whole-organismal senescence with respect to plants and non-unitary animals (Gardner & Mangel, 1997; Jones et al., 2014; Vaupel et al., 2004). Many cases of negligible senescence (i.e. mortality [Figure 1b,e] and/or fertility [the latter not addressed analytically in this review] remaining invariable with age) or even negative senescence (mortality risk decline [Figure 1e,f] and/or fertility increase with age [again, the latter not addressed in this review, but see Baudisch & Stott, 2019] are now known based on reliable data (Finch, 2009; Pletcher, Houle, & Curtsinger, 1998). Perhaps not by coincidence, these patterns occur in organisms not studied (or even discovered!) when the classical theories of the evolution of senescence were developed. Some of these include Brandt's bat *Myotis brandtii* (Podlutsky, Khritankov, Ovodov, & Austad, 2005), barn owl *Tyto alba* (Altwegg, Schaub, & Roulin, 2007), greenland shark *Somniosus microcephalus* (Nielson et al., 2016), rockfishes (Cailliet et al., 2001; Mangel, Kindsvater, & Bonsall, 2007; Munk, 2001), ants and termites (Carey, 2001), naked mole-rat *Heterocephalus glaber* (Buffenstein, 2008), *Hydra* (Martínez, 1998; Schaible et al., 2015), bristlecone pine *Pinus longaeva* (Lanner & Connor, 2001), *Borderea pyrenaica* (García, Espadaler, & Olesen, 2012) and wilcox brush *Eremophila forrestii* (Ehrlén & Lehtilä, 2002).

Caleb E. Finch, in his seminal monograph of senescence (1990), brought attention to the few known species that at the time suggested
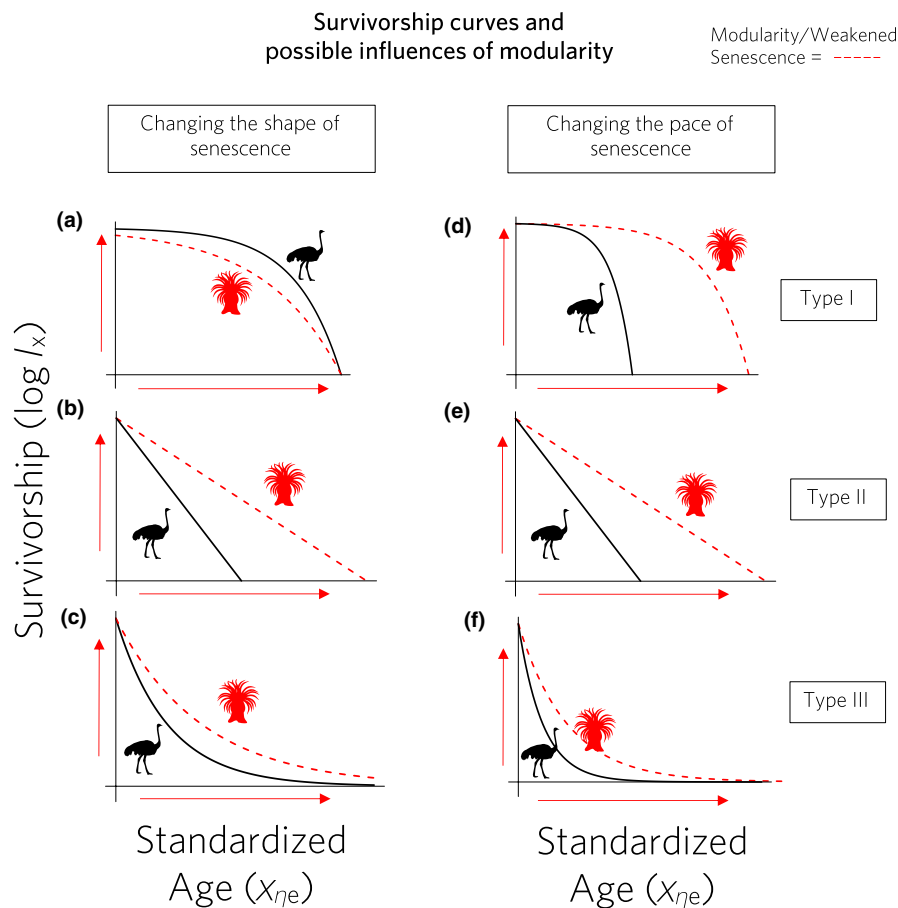

FIGURE 1 A graphic representation of Finch's Hypothesis for actuarial (i.e. mortality) senescence. These figures represent the hypothesized trait–senescence relationship in which modularity will lower the shape (a, b, c) and reduce the pace (d, e, f) of senescence, extending life span and reducing late-life mortality. The black lines labelled with ostrich icons represent a hypothetical unitary organism (with high physiological integration), and red lines labelled with coral icons represent a hypothetical modularized organism (with low physiological integration). Finch's hypothesis predicts that more modularized physiologies (the red lines) should result in reduced senescence (i.e. longer life span and lower late-age/stage mortality) relative to their unitary counterparts (black lines). The survivorship curves are characterized by survivorship type (Type I: a, d; Type II: b, e; and Type III: c, f), reflecting standard groupings of mortality patterns, which is discussed further in Figure 2. Note that changes in the shape of a Type II curve obligates either a Type I (a, d) or III (c, f) curve, but pace can drive a less severe slope, which takes its place in this graphic. Also, note that the difference in shape and pace curves among Type 3 (c, f) curves are more subtle than for Type I (a, d) curves; the difference between panels are most noticeable in how they intercept the *x*-axis. If the effect of modularity on senescence was strong enough, a species or lineage could be predicted to pass from one survivorship type to another—from Type I, to II, to III respectively

negligible or even negative senescence. In this monograph, Finch posited a novel connection between senescence and anatomy, by probing the question of how modular forms could alter senescence. Finch suggested that if modules age independently or incur injury independently, as physiological units, then they may insulate the composite individual from those effects. Thus, Finch inquired whether modules could lengthen the life of an individual by preventing local injury from translating to injury of the individual. Until recently, testing this hypothesis has been hindered by a dearth of relevant data and substantial literature across many species. Here, we avail of recent advances in histology and physiology to proxy the degree of modularity, and evaluate links between demographic data and actuarial senescence.

Here, and in line with classical use, we use the term 'modular' to describe individual organisms that are constructed of multiple, repeat subunits that are individually dispensable without injury to survivorship of the individual (Vuorisalo & Mutikainen, ; Vuorisalo & Tuomi, 1986). Within this framework, the individual can be thought of as a grouping of genetically identical cells which share physiological interdependence (extending from criteria distinctions discussed by DiFrisco, 2018; Pepper & Herron, 2008; Pradeu, 2016). This usage allows for minor genetic variation that occurs from random, somatic point mutations. Clones are therefore considered parts of a single individual – as are modules – despite their heightened degree of physiological independence and compartmentation. Alternative ways to define individuals may be used on the basis of fundamental evolutionary units, specific physiological properties or other factors. However, our definition facilitates comparison across broad morphological types and taxonomies, while retaining a demographically coherent unit. Our analysis shows that traits that reflect a higher degree of physiological modularity correlate with negative senescence in plants, although not necessarily in animals. These findings therefore provide novel empirical support for a possible explanation for escaping senescence.

2 | A COMPARATIVE OVERVIEW OF FINCH'S HYPOTHESIS

We extend Finch's hypothesis by linking its predictions to the *pace* and the *shape* of senescence. The distinction between the pace and shape of senescence emerged long ago (Keyfitz, 1977) as a useful framework in ageing research that decouples important metrics of ageing (Baudisch, 2011; Baudisch et al., 2013; Keyfitz, 1977; Wrycza & Baudisch, 2014). The pace of senescence represents the characteristic mean life expectancy (η_e) of an organism. For example, the life span of moss campion *Silene acaulis* (routinely > 50 years, some specimens > 300 years; Morris & Doak, 1998) is greater than that of the invasive cheatgrass *Bromus tectorum*; <1 year (Compagnoni & Adler, 2014). On the other hand, the shape of the senescence describes *when* and how intensively mortality hazards change during the *normalized* lifetime (typically over η_e; Keyfitz, 1977) of a species (Figure 2). Because shape is normalized (i.e. age-value divided by mean life expectancy), it allows one to compare the rate of senescence across many species, regardless of differences in η_e across populations and species.

Following Finch's hypothesis, we expect greater functional modularity to negatively influence the pace, and, to a more limited degree, the shape of senescence. Modularity should affect the pace of senescence through the individual's ability to minimize the damage by shedding damaged modules (Denley & Metaxas, 2016; Kozlowski, 1973). Examples of modules that could be shed include specific ramets (in clonal organisms), wood vessels, cnidarian polyps, bryozoan polymorphs and ascidian zooids. In general, we do not expect the shape of senescence of a given species to be influenced by modularity. This is because neither the theory nor the evidence suggests that localized injury is linked to age-specific mortality any more or any less than other mortality factors. However, modularity might alter shape if the accrual of mutations (sensu Medawar, 1952) occurs on a modular basis and age-dependent declines are restricted to the module. Because multiple prerequisites underlie this proposition, we consider it a subsidiary hypothesis.

The scope of our review encompasses multiple kingdoms and a wide array of taxonomic groups. By approaching the commonalities of modularity broadly, the structural appearance and functional role of modules is diverse across different species. For example, plants can have deeply modularized anatomies where physiological independence
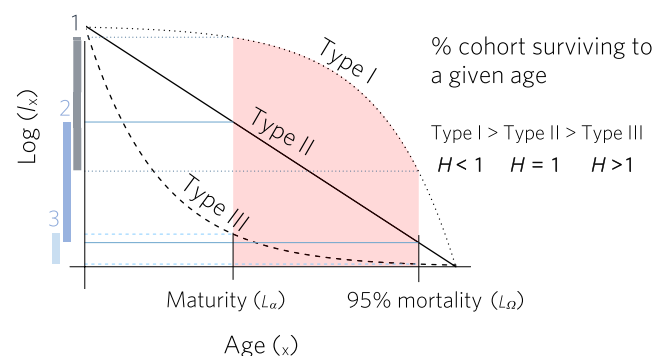
FIGURE 2 A graphic representation of Keyfitz' entropy (*H*). The shape of actuarial senescence can be measured across a wide range of dissimilar actuarial dynamics, using a standardized metric of *H*. Keyfitz' entropy *H* quantifies when mortality occurs along the lifetime of a cohort, from age at maturity (L_α), until – in this case – 95% of a cohort has died. This metric is dimensionless and normalized by the mean life expectancy of the organism, which allows for intra and interspecific comparisons, and is directly linked to the degree of modularity of different organisms (See Figure 4). Survivorship types describe different mortality patterns with age. In Figure 4, which highlights a period from maturity to 95% cohort mortality in red, a larger fraction of total mortality occurs in Type I and Type II mortality than in Type III, as shown by the length of bars on the y-axis, which correspond the colour to the survivorship lines. To a lesser extent, more mortality occurs in Type I than Type II over this same interval. The normalized metric represented by bar length (percent cohort change) indicates the mortality change over the *entire* interval; however, this measure of change can be measured at any age interval within this range. *H* is a metric of whether mortality through time tends to be weighted in early life, nearer to maturity, or late in life, nearer to 95% cohort mortality. Accordingly, Type I survivorship has the greatest *H*, with higher mortality change occurring late in the interval, near L_Ω, and Type III has the lowest *H*, with mortality change occurring late in life, near L_α

among structurally discrete components of an individual is high (e.g. ramets of an aspen grove). In contrast, animals tend to be 'unitary' individuals, meaning that they have structural anatomies where physiological integration is high across their body plan. In these organisms, 'shedding' part of a unitary animal (e.g. loss of an appendage or loss of a segment of the body) tends to be, by definition, physiologically disruptive to the individual.

The potential for greatly different physical and functional qualities of modules is unified by a basic characterization: modules need not be inclusive of the full range of physiological functions carried out by the individual such as water balance or energy regulation. Instead, modules can be recognized as repeat subunits within just one of those physiological functions (e.g. immune activity, renal activity, respiratory activity). This latter point suggests that we can divide our analysis into kingdoms, separating it between unitary (animals) and non-unitary (some animals like corals and sponges, and plants) organisms.

In general, traits that enhance modularity should correlate positively with the pace of senescence (Silvertown, Franco, & Perez-Ishiwara, 2001). In plants, we expect increases in clonality, stem sectoriality (Ewers, Ewers, Jacobsen, & López-Portillo, 2007; Schenk et al., 2008) and bark stripping (i.e. the ability of trees to persevere when only a small part of the trunk is surrounded by live cambium, Matthes, Kelly, Ryan, & Larson, 2002) to prolong longevity. In animals, we expect the number of polyps within a colony (Denley & Metaxas, 2016) and the number of functionally redundant organs to prolong longevity. The number of discrete, redundant pathways for blood or lymph circulation (Huang, Lavine, & Randolph, 2017; Seiler, 2010 respectively), and the number of parallel endocrine pathways should do the same. We expect that all of these traits should correlate negatively with mortality rates, and therefore increase the pace of senescence (Figure 1a–c).

On the one hand, the link between modularity traits and the rate of mortality over age (i.e. the shape of senescence) is subtler. In plants, several traits could change age-specific mortality rates, including clonality, epicormic branching in trees and indeterminate growth. On the other hand, in animals, fewer traits would be expected to change age-specific mortality rates, because growth tends to be definite. A notable exception to this is metazoans (Bodnar, 2009). The indirect effect of age-related size-dependent benefits can potentially tap alternative mechanisms for escaping senescence detailed by Vaupel et al. (2004).

Modularity is expected to decrease the pace of senescence, with possible indirect effects on the shape of senescence. As discussed above, modularity may indirectly affect the shape of senescence as a result of facilitating the attainment of advanced ages and size-dependent benefits. More generally, modularity could have a more complex effect on the shape of senescence if localized injuries affect age-specific mortality. Some mortality factors are internally mediated and dependent on lifespan-normalized age; others are external and independent of age. As a result, if modularity facilitates the attainment of more advanced ages, we would expect that the pertinent mortality factors will change. These changes may alter when in the life span of an individual mortality is most likely to occur, and thus change the shape of senescence.

This relationship is captured in the generalized age-dependent/degenerative term under the Reliability Theory of Senescence (Gavrilov & Gavrilova, 2001). Where environmental mortality factors influence the mortality rate irrespective of physiological declines, they are mapped as age-independent mortality factors. Under Reliability Theory, modularity will influence the realization of a system failure (which we define at the scale of the individual), as it constitutes a system component with functional redundancy that, in turn, acts as a redundancy parameter, reducing *failure events:system failure* (i.e. local injury:individual mortality).

2.1 | Modularity and senescence in plants

Several lines of evidence suggest that structural characteristics of plants that increase their modularity also lengthen their longevity. One line of evidence relates to epicormic branching in trees – the ability to produce branches directly from sections of the trunk (Meier, Saunders, & Michler, 2012). Epicormic branching has been linked to longevity in long-lived trees, such as the Douglas fir *Pseudotsuga menziesii* (Ishii, Ford, & Kennedy, 2007). Epicormic branches of the Douglas fir allow old, large trees to maintain foliage (Ishii, Ford, & Dinnie, 2001), which is crucial to provide the large photosynthetic capacity that allows the trees to maintain their characteristic high growth rates at advanced ages (Sillett et al., 2010). Moreover, modularity clearly promotes large sizes in many clonal species of plants. Some of the largest and oldest individual organisms on earth are clonal plants, such as the Pando aspen clone *Populus tremuloides* (DeWoody, Rowe, Hipkins, & Mock, 2009; Mock, Rowe, Hooten, Dewoody, & Hipkins, 2008) or a Neptune grass clone *Posidonia oceanica* found in the Mediterranean Sea (Guerrero-Meseguer, Sanz-Lázaro, & Marín, 2018).

Demographic effects of physiological injuries also appear to be informed, at least in part, by the structural organization of an organism. Severe, long-term impairments of a physiological system can be fatal for some species and merely locally injurious, and without individual-scale physiological injury, for others (Kozlowski, 1973; Winston, 2010). Whether one is injured can be a function of the degree of integration within the individual. The transport of water in plants is instructive in this regard. Since the 1980s, the formation of air pockets in hydraulic conduits within plant vascular tissues – *air embolism/cavitation* – has been found to be a prevalent cause of mortality in terrestrial plants (Tyree & Sperry, 1989; Zimmerman, 1983). Variation in the morphology that influences embolic risk is expected to have a high demographic impact.

The interaction of emboli with the morphology of plant vascular and vessel morphometry has been the focus of much research (Bailey, 1916; Ewers, 1985; Pitterman et al., 2010; Sperry, Sano, Sikkema, Feild, & Hacke, 2007; Tyree & Sperry, 1989; Venturas, Sperry, & Hacke, 2017). The well-established 'rare pit hypothesis' of trade-offs between efficiency and safety of vessels stems from

this line of questions (Christman, Sperry, & Adler, 2009; Christman, Sperry, & Smith, 2012). Of direct importance to senescence, the safety-efficiency vessel trade-off offers a case example of direct importance to senescence. When an embolism forms in a vessel, the vascular configuration of the plant determines whether and to what extent the initial embolic disruption spreads to the whole organism (i.e. runaway embolism sensu Sperry and Pockman 1993). More sectorial vasculatures can mitigate the risk of runaway embolism by reducing the lateral connections between vessel bundles. If emboli result in runaway embolisms, they would be contained to a few bundles (Orians, Babst, & Zanne, 2005; Zanne et al., 2014).

A series of studies have explored the environmental correlates of this type of sectoriality, and have found higher sectoriality in habitats with higher embolic risk, notably deserts (Carlquist, 1975; Ehleringer, 1980; Ewers et al., 2007; Maherali, Pockman, & Jackson, 2004; Royer, Wilf, Janesko, Kowalski, & Dilcher, 2005; Wright et al., 2004; see, too, related questions addressed by Alder, Sperry, & Pockman, 1996). Indeed, Schenk et al. (2008) showed that sectoriality increases on either hemisphere towards the latitudinal belt of arid zones across 12 woody species.

To test the effect of modularity on senescence in plants, we quantified the correlations between measures of the pace and shape of senescence and proxies to plant modularity. We quantified pace and shape of senescence using the COMPADRE Plant Matrix Database (Salguero-Gómez et al., 2015) and used methods described in Salguero-Gómez (2014) and Jones et al. (2014). We implemented a method described in Baudisch et al. (2013) to compare inter-specifically various moments of senescence across species. This approach isolates the measures of life span from lifespan-standardized metrics of survivorship, and reduces highly variable mortality patterns to select statistic features whose discrete markers reflect key variations in population structure.

We derived, from the resulting 138 plant species in COMPADRE, three metrics of the pace of life, which are used as proxies for measuring the pace of senescence. These metrics consist of mean life expectancy (η_e), maximum longevity (η_{max}) and generation time (T) using the methods developed by Caswell (2011). We used a single metric to characterize the shape of senescence: Keyfitz' entropy (H) (Baudisch, 2011; Keyfitz, 1977; Wrycza et al., 2015), which describes the overall shape of the survivorship curve (Figure 2), such that when $H > 1$, mortality—and thus the rate of actuarial senescence—decreases with age. Thus, under this metric, the decay in survivorship is quantified irrespective of reproduction. An estimate of the pace of senescence for a species can be calculated from an adult survivorship class. Relying upon a single adult stage-class for survivorship estimation, however, implies that there is no shape of senescence for the given species, which in select cases may not be the case. The influence of this assumption as results will not be known until a later time, when more age/stage-specific survivorship estimates are available.

The metric H, as well as those that quantify the pace of life (e.g. η_e, η_{max}, T), rely on age-structured demographic information. The underlying demographic information is often captured in a stage-structured format (as matrix population models). To derive the age-structure from stage-structured models, we used methods from Jones et al., 2014 (based on mathematical proofs by Caswell, 2001). These age-from-stage extraction methods use numerical simulations of the stage-structured matrix to determine a quasi-stable-state of a population distribution for species that have multiple adult age/stage-class survivorship estimates. This, in turn, yields a decaying survivorship l_x curve from which our desired summary metrics can be extracted in an age-structured form. When simulation models converged to a stable stationary population distribution, they were not included in our data. This method and convergence selection criterion were also used on the data for animals (below).

To test whether functional correlates of physiological modularity inform the pace and shape of senescence, we drew on several open source databases. We collected functional trait data related to the degree of modularity from the FRED (Iversen et al., 2017), BIEN (Enquist, Condit, Peet, Schildhauer, & Thiers, 2009) and TRY databases (Kattge et al., 2011), as shown in Figure 3. These global trait databases are curated repositories of plant traits collected from a wide range of field studies, laboratory experiments and observations. Here, we consider only those quantified under unmanipulated conditions. When multiple functional trait values were available per species, we computed the mean of each species-specific trait value. This was a straightforward mean, which we performed after making sure that identical values of a trait (e.g. root diameter of 0.4996 mm) were not doubles. To do so, we examined several instances of double values in each one of the three databases, and contacted database curators for feedback on the most suspicious cases. We present only results of our analyses where >20 species were present in COMPADRE and in the functional trait databases (Table S2).

Finally, we tested for correlations between species-specific demographic traits and their modularity traits using a battery of phylogenetic generalized least squares (pgls) using the packages APE (Paradis, Claude, & Strimmer, 2004) and CAPER (Orme, 2013) in R (R Core Team, 2012). We used the phylogeny of vascular plants by Zanne et al. (2014). Family-wise errors were corrected using a Bonferroni correction. We used Akaike information criterion (AIC) scores for model selection among a set of competing models with linear and quadratic predictors. Linear models consistently had the lowest AIC and were therefore retained.

2.2 | Modularity and senescence in animals

The literature on senescence has historically been more inclusive of unitary animals than it has been for other groups. Important examples that have advanced our understanding of animal senescence include studies on roe deer *Capreolus capreolus* (Gaillard et al., 1993, Loison, Festa-Bianchet, Gaillard, Jorgenson, & Jullien, 1999); Soay sheep *Ovis aries* (Catchpole, Morgan, & Coulson, 2000); Dall sheep *Ovis dalli* (Deevey, 1947; Kurtén, 1953; Murie, 1944); alpine marmot *Marmota marmota* (Berger et al., 2016); and the short-tailed vole *Microtus agrestis* (Leslie & Ranson, 1940; Leslie, Tener, Vizoso, &
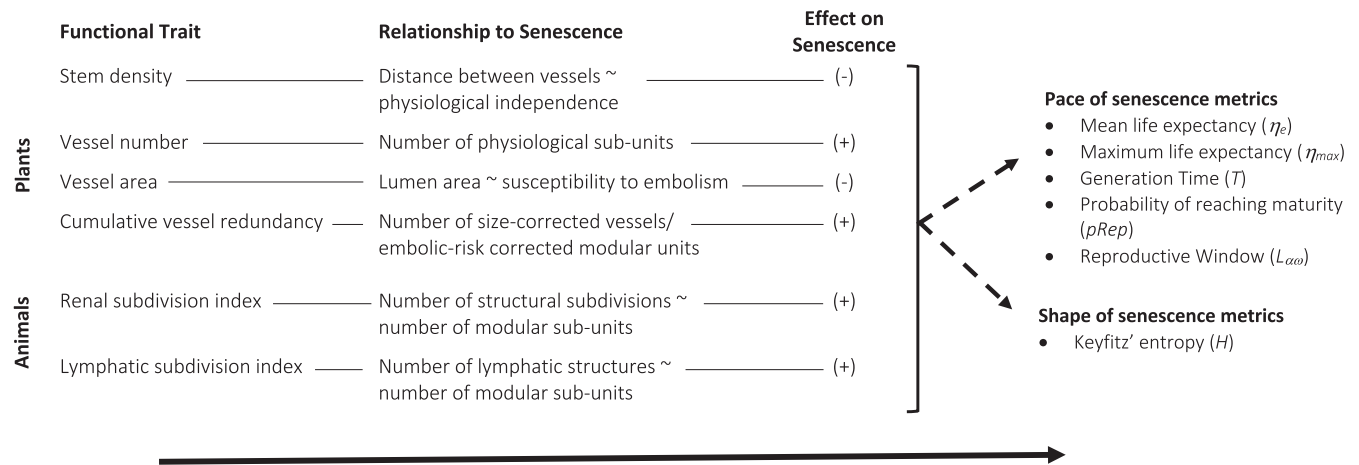
Functional Trait	Relationship to Senescence	Effect on Senescence

Plants

Stem density	Distance between vessels ~ physiological independence	(-)
Vessel number	Number of physiological sub-units	(+)
Vessel area	Lumen area ~ susceptibility to embolism	(-)
Cumulative vessel redundancy	Number of size-corrected vessels/ embolic-risk corrected modular units	(+)

Animals

| Renal subdivision index | Number of structural subdivisions ~ number of modular sub-units | (+) |
| Lymphatic subdivision index | Number of lymphatic structures ~ number of modular sub-units | (+) |

Pace of senescence metrics
- Mean life expectancy (η_e)
- Maximum life expectancy (η_{max})
- Generation Time (T)
- Probability of reaching maturity ($pRep$)
- Reproductive Window ($L_{\alpha\omega}$)

Shape of senescence metrics
- Keyfitz' entropy (H)

FIGURE 3 Hypothesized relationships between modularity and moments of actuarial senescence. Caleb Finch hypothesised that modularity controls the spread of injury from the site of injury to individual-level injury and mortality. We accordingly expect that anatomic proxies of modularity should predict higher values across indicators of longevity. The nexus of modularity and age-specific mortality is also interrogated in this study through proxy of Keyfitz' entropy

Chitty, 1955). However, to date, other lineages across the tree of life have not received much attention.

Research in senescence has only gained momentum in the Plantae Kingdom in the last two decades (Munné-Bosch, 2008; Salguero-Gómez, Shefferson, & Hutchings, 2013; Thomas, 2002, 2013; Vaupel 2004) and it remains even less represented among modular animals (but see Bythell, Brown, & Kirkwood, 2018 and Tanner, 2001). Within plants, researchers have explored the question as to whether ramets senesce faster than genets (Orive, 1995; Salguero-Gómez, 2017). A similar angle on the animal kingdom remains untapped. This may be attributable to the close relationship of modularity to meristem-based growth, plant vasculature and other functional characteristics restricted to the Plantae Kingdom (Clarke, 2011; Vuorisalo & Mutikainen,). Broader application of these scaling relationships may also be hindered by the conflation of modularity with other coexistent functional units. Modules often co-incide with carbon sectors, structural axes, independent physiological units and inflorescences, among other such structures.

Here, we provide a review of animal modularity and its relationship between longevity and senescence. Our goal was to offer a translation of the primarily plant-based term modularity to individual animals for which the integration of resources is high and adult size tends to be determined. In doing so, we take a discrete step beyond previous treatments on the subject (notably Ewers et al., 2007; Harper, 1980; Vuorisalo & Mutikainen,), in that we evaluate specific hierarchies of organ systems across demographic metrics.

At first sight, the term modularity would not be applicable to most animals if defined as repeat subunits of multi-cellular tissue (Chapman, 1981; Finch, 1990; Vuorisalo & Tuomi, 1986). Notable exceptions include corals and sponges, or, using a colony perspective, social animals such as ants (Kramer, Schrempf, Scheuerlein, & Heinze, 2015). Overall, however, the consideration of the Kingdom Animalia through the lens of classical modularity has to date received little attention. There is a corresponding lack of precision in describing the intertaxonomic concepts pertaining to modularity across kingdoms.

The development of a framework that cuts across kingdoms allows us to test urgently needed mechanistic theories across the tree of life (Baudisch & Vaupel, 2012).

A notable exception in this direction is the work by Esteve-Altava (2017), who recently interrogated a number of similarities in modular concepts. This research focused on structural, developmental, functional and topological integration of modules in plants and animals, thus touching upon how we characterize modularity in this paper. However, in stressing the conceptual cross over between plants and animals, Esteve-Altava highlights developmental origins, rather than the nexus of modularity with demography. The relationship between modularity and longevity found in plants (Figure 4a,b) raises the novel question of whether the degree of structural redundancy in animals is relevant to their survival. Specifically, it raises the question of whether its effect on senescence is comparable to that of modules in plants. We tackle this question with a review of animal tissues and a criteria-based approach to qualifying modularity from a histological and functional angle.

The classical model of modularity (White, 1979) describes modules along lines of discrete units of growth, units of reproductive independence (Harper & Bell, 1979), independent functional units (Sprugel, Hinckley, & Schaap, 1991) or independent physiological units (Watson, 1986; Watson & Casper, 1984). This principle works well to describe the plants and colonial invertebrates, since these are comprised of well-defined, repeat structural subunits. In other ways, it is deficient and unduly restrictive.

Modularity is a physiologically defined term whose touchstone is partial independence of different components of an individual. In most unitary animals, modularity does not appear to have ready application, as physiological functions are deeply integrated. In these organisms, the furthest extremities are physiologically linked to the central corpus of the individual. In this way, they are nearly opposite the concept of comprising semi-independent units. There is a critical disconnect, however, regarding this perception. Modules are traditionally thought of as 'individuoids' or 'complete physiological units', compartments
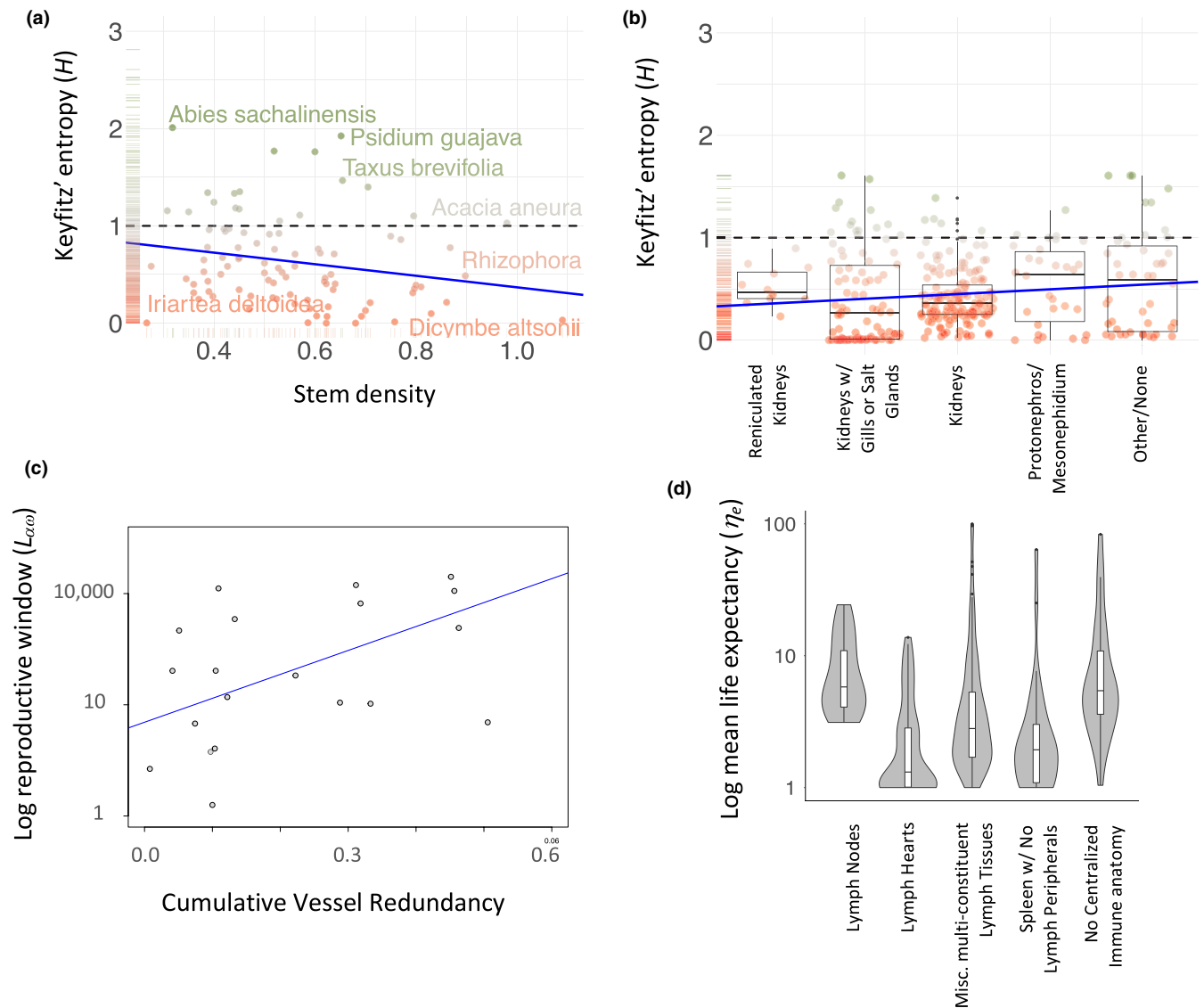
FIGURE 4 Finch's hypothesis accurately predicts the relationships between plant modularity as measured by functional traits and senescence, but fails to explain patterns in unitary animals between organ dividedness and senescence. Blue lines show statistically significant relationships between the shape of senescence (Keyfitz' entropy (H); see Figure 2 for more information on this metric) and (a) stem density, and (b) an index of renal modularization. $H = 1$, signified by the dashed black line, represents negligible or neutral senescence, and is the dividing line between positive and negative senescence. Larger H-values, higher on the y-axis (and colour-weighted by green), represent lower senescence; Smaller H-values, lower on the y-axis (and colour-weighted by red), represent higher senescence. While the stem density effect on the shape of senescence supports Finch's hypothesis, the renal index runs counter to expectation. (c) Cumulative vessel redundancy (lumen area corrected by vessel number) provides a measure of modular vessel units, quantified by number, corrected for vulnerability to embolism related to lumen area, and thereby is expected to be a more effective positive correlate of modularity than vessel number alone. Cumulative vessel redundancy positively correlates with reproductive window ($L_{\alpha\omega}$), consistent with the idea that modularized vessel anatomy slows the pace of senescence. (d) Mean life expectancy (η_e) correlates negatively with an index of immune anatomy subdividedness, counter to the hypothesis that more sub-divided, and thereby more modularized, anatomy would promote longevity. There is wide variation in the distribution characteristics which map out differently among groups, though this is not statistically significant. Some of these characteristics include the lack of short-lived species with lymph nodes, and a tendency for higher representation of species with low life spans in all groups except those with lymph nodes and no centralized immune anatomy. By reorienting the rank order of 'no centralized immune system', a stronger relationship would emerge

which package the full suite of physiological functions of an individual. But this does not need to be the case.

We can conceive of redundant organs in a subsystem, which might offer independence in function. So long as injury can be localized to a physiological system, one can envision a benefit conferred by

redundancies. Unitization can be within a single subsystem that ultimately affects the individual's fitness. Albeit counterintuitive, there exists a practical translation of the conceptual model of modularity to highly physiologically integrated organisms (i.e. traditional unitary organisms, such as higher vertebrates).

Injury to the physiology of integrated organisms is, almost by definition, of whole-organismal consequence. Importantly, integrated organisms typically relegate reproductive capacity to discrete sexual organs, and are thereby largely unamenable to regional reproductive isolation. This bauplan contrasts with that of plants, corals and sponges, where both somatic and reproductive functions are carried out in unison. In those organisms, the death of a branch or subcolony may not have important consequences. With a unitary animal, the loss of a subpart, such as an appendage or segment, may affect motility in a manner largely absent or irrelevant to plants and colonial invertebrates (Kozlowski, 1973).

It is thus challenging to find a ready and competent gross analogue in most animal species. But as was recognized decades ago, and indicated above, the full or near-full range of individualistic properties exhibited by plant modules is not the qualification for modularity. The bauplan of the individual may not be the appropriate level of assessment (Harper, 1980; Vuorisalo & Mutikainen, 1999). Indeed, the axioms of modularity are multicellularity and multiple *redundant* units (Tuomi & Vuorisalo, 1989). Thus, modularity is not necessarily incompatible with high bodily integration.

Archived information on the number of replicated organs across the tree of animals is surprisingly lacking. Tissues are often similar in organs across different species of animals and play a common functional role. We believe this to be a logical starting point for a systematic inquiry into the potential role of modularity in animal senescence, as posed by Finch (1990). Are any organs divided into major functional compartments that have functionally similar – if not equivalent – roles? Are they functionally related such that the impairment of one would not be indispensible to the individual's survival?

We carried out an exhaustive survey classifying redundant components in 151 animal species. For the two systems – lymphatic and renal – where there appear to be conditions that lend themselves to functional redundancy, we investigate the types and scale of repeated elements in the system and how those are distributed among major groups of organisms. Table S3 of the online supplemental material contains a summary review of other organ systems. To test the effect of organ multiplicity on senescence, we used methods similar to those employed in plants (above). This involves running a series of phylogenetic generalized least square models to quantify the relationship between indices of organ multiples and mean life expectancy (η_e; Figure 3), maximum life expectancy (η_{max}), generation time (T) and Keyfitz' entropy (H). H was analysed for only those species for which survivorship was estimated for at least two adult stage-classes.

2.2.1 | Immune system hierarchies

The lymphatic system is a strong candidate for a modular structure that might influence senescence partly because of the importance of immune integrity to survivorship (Nussey et al., 2013). More specifically, in certain species, the lymphatic system comprises numerous independent subunits that are spatially discrete. Within species that have lymph nodes, we speculate that modularity corresponds with the number of lymph nodes in the body. Thus, species with a greater lymphatic modularity should have lower rates of senescence, as an extension of Finch's thesis (1990).

Lymphatic systems of mammals span some 20-fold variation in lymph node number, from 22 in mice to 450 in humans (Haley, 2016) and several thousands in large ungulates (Nikles & Heath, 1992). However, despite such a vast range of interspecific variation, reliable estimates of lymph node numbers across a large range of animal species do not exist. Indeed, for species lacking lymph nodes, other proxies for the degree of modularity require more finessing. Organs known as lymph hearts promote the movement of lymph through the lymph vascular network of certain species. They are found in lieu of lymph nodes in reptiles (Hedrick, Hillman, Drewes, & Withers, 2013), amphibians (Hedrick et al., 2013) and some bird species (Budras, Hullinger, & Rautenfeld, 1987; Hedrick et al., 2013). These structures can range from several to >200 in an individual (e.g. Caecilians *Caecilia*; Kampmeier, 1969).

Lymph heart function is not equivalent to that of lymph nodes. These organs do, however, share qualities in that they both influence immune system integrity and comprise discrete, highly numerated structures that mediate lymph. Independent of the above structures, temporary or reactive lymphatic structures develop in certain other species. These are variously termed aggregates, nodules or patches, and participate in immune function. As such, they play a role in preventing tissue damage, disease or dysfunction, and their continued functionality thereby reduces mortality. One of the complications with the lymphatic system in regard to isolating injury and compartmentalizing damage is the functional integration of lymph nodes. Depending on the configuration, topology and dynamics of lymph flow, regionalized isolation of injury may be infeasible.

2.2.2 | Renal hierarchies

The kidney presents a good example of a non-conventional morphological unit that would meet the discrete, functional unit criteria of modularity. Kidneys are physically separate and individually dispensible. As reflected in humans, loss of a kidney does not attend serious acute or long-term declines in the integrity of the renal system (Foley & Ibraim, 2010; Kasiske et al., 2013; Wee, Tegzes, & Donker, 1990).

Most vertebrates possess two bilaterally positioned kidneys (Yokota, Kawashima, Ohkubo, & Sasaki, 2008). Among species, there is a wide variation in both function and morphology of kidneys (Bentley, 1971; Beuchat, 1996; Ortiz, 2001; Williams & Worthy, 2002). In bears and cetaceans, for example, the kidney contains numerous lobes (*reniculation*) which exercise partial independence from one another (Maluf & Gassman, 1998). The lobes comprise a physically integrated organ, but sub-regionalization within this organ is high. This represents the highest degree of subdivision within our grading system because the kidney structure can be functionally

divided into more than 100 compartments in some species (Ortiz, 2001). Reniculated kidneys with high structural and functional division may offer more potential for localized damage and tissue death without functional injury to the renal system.

Kidney pathologies are diverse, but mostly internally mediated (Basile, Anderson, & Sutton, 2012). Common drivers of kidney stress, injury and death are high amino acid or nitrogenous waste loads, hypertension and ischaemia/hypoxia (Palm & Nordquist, 2011). External factors such as blunt force trauma or kidney rupture more tightly akin to air embolism from the standpoint of acute, anatomically focused injury, are less common. It is unclear how subdivision affects the resiliency of renal physiology in response to different types of injury and stress.

While other species lack multi-compartmental renal organs, many species have multiple organs that participate in the management of wastes and ion balance. In marine fishes (Evans, 2008, 2010), sharks (Shadwick, Farrell, & Brauner, 2016) and crabs (Towle & Weihrauch, 2001; Weihrauch & O'Donnel, 2015), gills play an important role as an exchange site for ions between the organisms and their environments. In fact, these can be critical to nitrogenous waste excretion (see Wilkie, 2002 and Wright & Wood, 2012 for reviews). Many species of bony fishes are long-lived, including dogfishes, paddlefishes, sturgeon, rock fishes and eels (Patnaik, Mahapatro, & Jena 1994). Similarly, a number of shark species are atypically long-lived (Hamady, Natanson, Skomal, & Thorrold, 2014) and crustaceans, though particularly lacking in demographic studies, are popularly cited for their long life span (Vogt, 2012). Despite this, there is little clade-level synthesis of senescence or negligible senescence for these groups (Patnaik et al., 1994; Vogt, 2012). Attending the lack of research on senescence, links between specific anatomical similarities and longevity have not been directly explored, let alone interrogated in regard to renal anatomy.

Depressed osmoregulatory capacity resulting from injury to either the kidneys or the gills may be partially compensated for by having multiple osmoregulatory organs. Secondary osmoregulatory organs might also enable a higher osmoregulatory capacity and thereby tolerance for renal stress (Kültz, 2015), which could both prevent injury and facilitate recovery (Reimschuessel, 2001) from sublethal renal injury. In marine birds (Schmidt-Nielsen, 1960) and some reptiles (Schmidt-Nielsen & Fange, 1958), salt glands provide a mechanism to shed high salt concentrations. These glands could similarly enhance the redundancy of function (Gutiérrez, 2014; but see Babonis, Miller, & Evans, 2011) without nitrogenous excretion. It is broadly recognized that birds are unusually long-lived (Hickey et al., 2012; Monaghan et al., 2008; Nussey, Coulson, Festa-Bianchet, & Gaillard, 2008; Péron, Gimenez, Charmantier, Gaillard, & Crochet, 2010; Ricklefs, 2008), but research has conspicuously overlooked the role of anatomy in their longevity, and instead focused on cellular (Monaghan et al., 2008) and ecological explanations (Ricklefs, 2008). Reptiles include a number of the longest-lived species, such as tuatara which can live some 90 years (Magalhaes & Costa, 2009). Similar to birds, however, we see an inclination towards metabolic and cell-based explanations and an absence of attention given to organismal physiology and anatomy.

Along with gills, salt glands in accessory to kidneys constitute the second grade of centralization in renal function in animals (Figure 4c and d). They are ranked below reniculated kidneys because lobes of reniculation have the full suite of functional tools for renal control, whereas gills and salt glands are more limited. Gills in certain species of elasmobranchs (e.g. Smith, 1929; Wood, Pärt, & Wright, 1995) and salt glands carry functional weight only as it relates to ionic homeostasis, not nitrogenous wastes control. Recorded variation tracks with environmental stresses, such as seawater ingestion (Williams & Worthy, 2002) and hibernation (Ortiz, 2001), and corresponds in part with acute and chronic kidney injury susceptibility (Stenvinkel et al., 2018).

We note that two-component systems, such as bilateral kidneys, raise special questions under the framework we describe here. These cases elevate questions about alternative explanations for the second organ that may not reflect an adaptation for functional redundancy. There are two particularly important scenarios: alternative selective pressures and non-adaptive tendencies.

As highlighted by Ewers et al. (2007), the function of two eyes is an adept example of how non-redundant traits can drive redundant features. As such, functional benefits unrelated to redundancy can confound the evolution of multiple organs and their potential role in modularity as it relates to senescence. Separate ears present another example of a highly specialized alternative benefit. More generally, symmetrical bauplans may favour bilateral, paired structures. This may be particularly likely where symmetrical division of gross anatomy shapes a developmental or functional pattern in the organism. A candidate for this might be the bilateral lungs found in many terrestrial vertebrates. This issue has not been the subject of focused research.

Evolutionarily, the configuration of developmental control genes may influence the development of accessory organs (Oakley, 2017). There is currently a dearth of research on the relative tendency for singular versus multiple organ duplication resulting from genetic mutations. Regardless, the cost of having multiple organs is likely to factor into whether organs are likely to fix into a population by genetic drift. If we assume that costs are commensurate with organ number, lower numbers of organs would have a higher likelihood of persisting in a population before being removed. Therefore, one should attribute functional purpose to single-duplicate organs with a degree of scepticism.

3 | ANALYSIS OF MODULARITY IN PLANTS AND ANIMALS

Our analysis supports Finch's hypothesis that modularity influences the senescence in plants, but support appears mixed across animals (Figure 4). We found seven relationships between modular traits and measures of senescence (Table 1). The strongest relationships were found between between-cumulative vessel redundancy and the pace of senescence (e.g. cumulative vessel redundancy × reproductive

Demographic measure	Plant functional trait	Slope	p-value	F Statistic
Keyfitz' entropy (H)	Vessel number	−0.048	.299	1.122
	Stem density	−0.594	**.043**	4.225
	Root lignin	−0.005	.515	0.438
	Root diameter	−0.307	.616	0.256
	Vessel lumen area	5.123	.249	1.393
Generation time (T)	Vessel number	−0.121	.415	0.0269
	Stem density	−1.247	.207	0.570
	Root lignin	0.011	.770	0.243
	Root diameter	0.620	.795	0.404
	Vessel lumen area	9.067	.522	0.189
Mean life expectancy (η_{ee})	Vessel number	−0.164	.086	3.186
	Stem density	0.615	.550	0.360
	Root lignin	−0.013	.649	0.213
	Root diameter	−0.833	.716	0.135
	Vessel lumen area	19.341	**.034**	5.028
Maximum life expectancy (η_{max})	Vessel number	−0.305	**.041**	4.642
	Stem density	−0.674	.456	0.559
	Root lignin	−0.045	.170	2.016
	Root diameter	−0.802	.691	0.161
	Vessel lumen area	32.165	**.025**	5.669
Probability of reaching maturity ($pRep$)	Vessel number	−0.020	.508	0.451
	Stem density	0.232	.163	1.976
	Root lignin	0.000	.971	0.001
	Root diameter	−0.238	.490	0.488
	Vessel lumen area	0.579	.847	0.038
Mean age at maturity (L_α)	Vessel number	−0.156	.375	0.813
	Stem density	−0.211	.845	0.039
	Root lignin	−0.004	.909	0.013
	Root diameter	1.717	.423	0.658
	Vessel lumen area	21.611	.201	1.722
Reproductive window ($L_{\alpha\omega}$)	Vessel number	−0.375	.059	4.021
	Stem density	2.526	.159	2.039
	Root lignin	0.014	.791	0.073
	Root diameter	0.894	.865	0.029
	Vessel lumen area	45.652	**.022**	6.221

window (F = 3.9; R^2 = .41; Figure 4b). Other relationships were weaker, but discernable, such as that between vessel density and the shape of senescence (F = 4.2; R^2 = .08; Figure 4a). However, most of the relationships we tested within plants were not statistically significant (Table S4).

In animals, there is evidence both supporting and refuting Finch's hypothesis (Table 2). Our tests support the hypothesis that modularity lengthens the pace of senescence in animals (Figure 4; Table S5). Species with lymphatic organs with greater subdivision into repeat components have higher maximum life spans (95% C.I. [36.4, 73.4 years]) than those with more centralized lymphatic anatomies (95% C.I. [19.0, 59.2 years]). In contrast, evidence refuting Finch's hypothesis was somewhat stronger. Lymphatic anatomies with greater subdivision into repeat components presented lower mean life expectancies than those with lower subdivision (95% C.I. [4.1, 12.1 years] vs. 95% C.I. [7.9, 15.7 years]). When analysing renal anatomies, higher modular indices correlated with lower maximum life spans (95% C.I. [0.3, 13.6 years]) and we found no correlation between modularity index and mean life expectancy. The renal modularity index was negatively correlated with the shape of senescence (95% C.I [0.008, 0.084]). Other relationships were not statistically significant (Table S5).

TABLE 2 Full results of linear models of indices of anatomic modularity relating to metrics of senescence and longevity in animals. Statistically significant relationships occur in bold

Demographic measure	Animal functional trait	Slope	p-Value
Keyfitz' entropy (H)	Renal modularity index	0.046	**.019**
Keyfitz' entropy (H)	Lymphatic modularity index	0. 001	.967
Mean life expectancy (η_e)	Renal modularity index	1.636	.274
Mean life expectancy (η_e)	Lymphatic modularity index	1.474	**<.001**
Maximum life expectancy (η_{max})	Renal modularity index	6.706	.061
Maximum life expectancy (η_{max})	Lymphatic modularity index	−6.595	**.002**
Generation time	Renal modularity index	2.343	.304
Generation time	Lymphatic modularity index	0.113	.910

4 | DISCUSSION AND FUTURE DIRECTIONS

In this study, we tested Finch's hypothesis (1990) that more modular organisms should be more likely to livelonger because of a decoupling of local injury from individual mortality. We situated Finch's hypothesis in a broad, comparative framework to look at a complete spectrum of degrees of physiological integration within the context of the most recent research on senescence (Cohen, 2018; Jones et al., 2014; Vaupel et al., 2004). To test our hypotheses in a fully comparative framework, we used functional traits to explore plant senescence and propose measures of modularity in unitary animals, a group of organisms thus far neglected in the comparative study of modularity. We found support for Finch's hypothesis in plants, but mixed support in animals.

Our results suggest that, in plants, modularity may in fact prevent or moderate the potential for localized injuries to depress demographic rates (i.e. reproduction and survival). Modularity may decouple localized injuries from individual fitness. Our work offers a general test of this hypothesis using functional traits and demographic data, and highlights the need for further work to uncover the underlying processes.

We tap a similar angle for two animal traits for which our results are less conclusive. In the two physiological systems we studied in animals, the effect of modularized systems on the pace and shape of senescence may be limited because of (a) the types of mortality factors they interact with (Baudisch & Vaupel, 2010; Caswell, 2007; Williams, Day, Fletcher, & Rowe, 2006); (b) the potentially confounding influence of other unitary physiological systems to which they are linked (physiological causality, and thus relationships, are not well understood; Nussey et al., 2013 and Ricklefs, 2008); or (c) because their preliminary classification of subdivision does not best represent functional divisions (trait-based approaches based on organ morphology are few).

4.1 | Plants

In plants, the examination of this empirical support suggests that modularity can affect senescence in a wide variety of ways. In particular, modularity could affect senescence by changing average mortality throughout the life cycle of organisms, by decreasing age-specific mortality rates. Modularity could also affect senescence by increasing the likelihood of reaching maturity and the reproductive window with age (Caswell, 2007).

We found a positive correlation between modularity in plants and the pace and shape of senescence. This suggests that the degree of modularity may affect the life cycle by interacting with age-independent and age-specific mortality rates. A consistent pattern in the slowed pace of senescence between modularity and clonality supports the view that there is a similarity in demographic process. Extended life spans that we report for modular plants add to, and generalize from, a tradition of studies that relate clonal forms to longevity (Orive, 1995; Sköld & Obst, 2011; de Witte & Stöcklin, 2010). Modularity can be thought of, in limited part, as a downscaled, localized and weaker form of semi-clonality (White, 1979). This trait enables larger, composite organisms to access a new ecological and evolutionary space that is inaccessible to less unitary plants (Mackie, 1986). Today, the spectrum of modularity adds a complex layer to the picture of demography, by grading a key distinction regarding senescence between clonal and non-clonal species. Unlike clonality, which lies at the high end of the physiological independence spectrum, many other modular species tend to have an intermediate degree of physiological integration. Modular species therefore present a valuable opportunity to explore the senescence and the mechanism which relieve it.

Moreover, the measures of modularity that correlate with measures of senescence further support the adaptive value of hydraulic integration as suggested by Schenk et al. (2008). Although promising, we cannot, with the data available at present, attribute the general relationships in our results to the causality of air embolism and related injury. Moreover, the potential adaptive aspects of modularity may not speak to the origins of its evolution, or if it is incidental to another adaptive feature in plants.

The correlations between stem density/cumulative vessel redundancy and mean life expectancy (a measure of the pace of senescence; Figure 2) suggest that modularity decreases the average mortality rate. However, the correlation between the same measures of modularity and a less strong shape of senescence (Keyfitz' entropy; Keyfitz, 1977) could reflect an alleviation of age-specific risk factors. Our initial expectation was that modularity would not influence the shape of senescence, but for a specific caveat related to how effects of age-specific deleterious genes might be minimized by modularity (consistent with the three classical theories for senescence).

Our expectation implied that the effect of modularity would not change based on the age of an organism. A correlation between modularity and the shape of senescence suggests that modularity can affect age-specific mortality factors. Such decrease in age-specific mortality rates could be a direct or indirect effect of modularity. Our measures

of modularity – including stem density and cumulative vessel redundancy – might plausibly decrease age-specific mortality directly. This direct decrease could occur because the risk of stem cavitation should increase with the height, and therefore age, of individual plants (Koch, Sillett, Jennings, & Davis, 2004). However, other modularity traits could decrease age-specific mortality rates indirectly. For example, epicormic branching should increase the capacity for long-term growth (Ishii et al., 2001) and therefore fertility (Ishii et al., 2007) of trees. In this case, modularity could decrease age-specific mortality rates indirectly, by increasing size.

4.2 | Animals

In animals, our analyses partly support the hypothesis that modularity should decrease the pace of senescence in animals. In particular, we found that immune anatomies with greater organ system subdivision have higher life spans than those with higher centralization. However, we found an opposite (though weaker) trend when looking at mean life span.

The nexus of immune anatomy and immune function is an area that would benefit from further study, particularly because of the role of localization and metastatic processes. Moreover, there is a dearth of studies on the functional property differences between the lymph nodes and lymph hearts (Hendrick et al., 2013). In particular, there is little available information on interspecific lymph node variation. But despite having effectively no information on lymph node variation, the category has a narrow distribution of rates of senescence (H) and mean life expectancies (η_e). *H-values* reflect that not only most organisms exhibited similarly lengthed life spans, but that mortality patterns were similar among species. Lymph nodes are almost exclusive to class Mammalia, for which the overall number of samples in the database is not high – and thus this may be partly an artefact of sample size. This topic remains of high interest because of growing interest in immunosenescence and how lymphatics intermediate immune integrity comparative physiology of lymph (Aw, Silva, & Palmer, 2007).

In animals, the shape of senescence was not consistently predicted by our index of modularity. One possible explanation for these results may reside in the way we classified basal vertebrate and invertebrate systems as 'centralized' or non-modular. There is also latitude to interpret which sets and subsets of organs are appropriate for comparative analysis, which could prove material to the results. Given the extreme range of anatomies and distributed nature of available data on anatomical biometrics, there is ample room, apt timing and increasing value (Griffith, Salguero-Gómez, Merow, & Mahon, 2016; Weiss & Ray, 2019) in a more refined approach to defining functional and anatomical trait groups for comparison.

An opposite trend between modularity and longevity in renal anatomy is notable. In addition to caveats noted at the outset of this section, this result may be explained partly by variation in the renal stresses experienced by the organisms evaluated. For example, renal stress is greater for species which ingest high amounts of

salt and for those that exchange ions with hypertonic environments (Hildebrandt, 2001). Similar influences may be responsible for the unexpected pattern we identified for age-specific mortality differences in renal anatomy.

In both plants and animals, there was a lack of detection of many significant relationships between measures of modularity and measures of senescence. The statistically significant relationships might reflect spurious correlations or true, reproducible patterns. Prior knowledge suggests that the correlations we detected likely reflect the reproducible patterns, at least in the case of plants. We expect these patterns to be reproducible because our significant correlations include measures of modularity that are considered adaptive (Schenk et al., 2008). Future studies should clarify the strength of the evidence here presented, especially because the sample sizes of freely available demographic and functional trait data are increasing exponentially with time (Enquist et al., 2009; Iversen et al., 2017; Kattge et al., 2011; Salguero-Gómez et al., 2015, 2016).

In species where reproduction is limited by actuarial senescence, we would expect modularity to change the shape of reproductive senescence (Lemaître & Gaillard, 2017; Ricklefs, 2008). Specifically, modularity that promotes, directly or indirectly, the attainment of large organismal size should increase age-class-dependent reproduction (Stearns, 1992). A relative increase in an organism's maximum organismal size at first reproduction should do the same (Lacey, 1986). We do not analytically test this in our review due to data constraints. We note, however, that contrary to our a priori expectation, the literature provides evidence of reproductive senescence in clonal species (e.g. Ally, Ritland, & Otto, 2010) and actuarial senescence in asexual species (Martinez & Levinton, 1992).

We do not test reproductive senescence in this monograph because there are more involved procedures required to ascertain changes in reproductive patterns. These are not supported by the current data available, but may be in the near future. Nevertheless, we predict that modularity could affect reproductive senescence if it facilitates the attainment of large sizes, to which fertility is often proportional (Weiner, Campbell, Pino, & Echarte, 2009). In addition, at large sizes, the patterns of selection for fertility and mortality can become decoupled from age (Caswell & Salguero-Gómez, 2013; Roper, Capdevila, & Salguero-Gómez, 2019). Forthcoming work lays a conceptual foundation for statistically decomposing fertility patterns similarly to how we approach actuarial patterns here (Baudisch & Stott, 2019). This approach uses pace and shape metrics in a slightly different application, offering a promising next step to this subject.

Our analyses of plant and animal data show that greater degrees of modularity correlate with the pace and shape of senescence, at least in plants. Recent developments in research on hydraulic integration provide strong inference for why we would expect to see the patterns we report. Despite this, one cannot eliminate the possibility that correlated, yet to be identified benefits are responsible for driving this pattern. Moreover, current mechanisms do not necessarily give insight to the evolutionary origins of the trait, which will require addition study. These results offer a foundation from which to work and enhance the urgency for this line of research.

4.3 | Future directions

Our findings and review point to three avenues of potentially fruitful future research:

4.3.1 | Age-specific mortality factors influenced by modularity

We have long understood that modularity, at least in the form of clones, could relieve mortality forces for the greater individual (Cook, 1979; Salguero-Gómez et al., 2013; Williams, 1957). That less-physiologically independent modules could achieve a similar outcome and play a role in determining the shape of senescence is a more recent area of thought (Vaupel et al., 2004). While we are left with more questions than answers in our results, our finding that modularity may act on age-specific mortality rates suggests value in a research agenda focusing on age-specific mortality factors as a fitness benefit of modularity.

4.3.2 | Role of dysregulation and physiological modules in the endocrine system

The field would benefit from additional investigation into the role of dysregulation and physiological modules in the endocrine system. A growing body of research describes a close connection between endocrine dysregulation and ageing in humans (Cohen et al., 2013; Li et al., 2015; López-Otín, Blasco, Partridge, Serrano, & Kroemer, 2013). This work provides an important and potentially widely applicable mechanism for ageing, explaining patterns of physiological senescence, based on the analysis of biomarkers. Dysregulation research advances are at the forefront of understanding intermediating factors in physiological declines. Theory in this area suggests that dysregulation does not necessarily rely upon the three prevailing theories for the evolution of senescence (Cohen, 2016). Rather, this phenomenon is likely linked to emergent properties of complex systems. Understanding the role of this mechanism, its origination, and its interactions with related theory – including the modularity interface – will be a major step in senescence research.

4.3.3 | Specific limitation on the fitness advantage of modularity

Ageing biologists would benefit from a research programme that probes the specific limitations on the fitness advantage conferred by modularity. Welch and Waxman (2003) offer a strong example of the type of work to be probed at this interface from an evolutionary angle. We have found reduced mortality rates associated with highly modularized architectures in plants. A now relevant question is whether there are costs and specific factors which curtail such a benefit to plants and animals. Some candidates for inquiry include

environmental stresses that could be exacerbated by branched morphologies (in climes where snow, wind, cold temperatures, or other physical stresses exert routine injury), energetic costs of higher surface area (bark-to-vascular tissue), vulnerability to disease and light competition factors.

For decades, senescence was believed to be a universal phenomenon. Evidence now exists that the ageing process is not inevitable in all species. Mechanisms remain elusive, but this paper takes a modest step forward in testing Finch's prediction of one mechanism based on the modular architecture of plants and animals. Future research is needed to more deeply explore relevant modular traits and their influence on specific mortality factors in plant and animal species.

AUTHORS' CONTRIBUTIONS
R.S.-G. conceived the ideas and designed methodology with input from C.B. and A.C. R.S.-G. quantified life-history traits. A.C. collated information on plant functional traits. C.B. obtained indicators of modularity in animals. R.S.-G. and C.B. ran the phylogenetic analyses. C.B. led the writing of the manuscript with input from A.C. and R.S.-G.

DATA AVAILABILITY STATEMENT
Data deposited in the Dryad Digital Repository https://doi.org/10.5061/dryad.7pvmcvdpk (Bernard, Compagnoni, & Salguero-Gómez, 2019). The demographic information is permanently, open-access archived in www.compadre-db.org.

ORCID
Connor Bernard (iD) https://orcid.org/0000-0003-2175-5712
Aldo Compagnoni (iD) https://orcid.org/0000-0001-8302-7492
Roberto Salguero-Gómez (iD) https://orcid.org/0000-0002-6085-4433

REFERENCES
Abrams, P. A. (1993). Does increased mortality favor the evolution of more rapid senescence? *Evolution*, *47*(3), 877–887. https://doi.org/10.1111/j.1558-5646.1993.tb01241.x
Alder, N. N., Sperry, J. S., & Pockman, W. T. (1996). Root and stem xylem embolism, stomatal conductance, and leaf turgor in Acer grandidentatum populations along a soil moisture gradient. *Oecologia*, *105*(3), 293–301.
Ally, D., Ritland, K., & Otto, S. P. (2010). Aging in a long-lived clonal tree. *PLoS Biology*, *8*(8), e1000454.

Alper, S. J., Bronikowski, A. M., & Harper, J. M. (2015). Comparative cellular biogerontology: Where do we stand? *Experimental Gerontology*, *71*, 109–117. https://doi.org/10.1016/j.exger.2015.08.018

Altwegg, R., Schaub, M., & Roulin, A. (2007). Age-specific fitness components and their temporal variation in the barn owl. *The American Naturalist*, *169*(1), 47. https://doi.org/10.2307/4122263

Aw, D., Silva, A. B., & Palmer, D. B. (2007). Immunosenescence: Emerging challenges for an ageing population. *Immunology*, *120*, 435–446. https://doi.org/10.1111/j.1365-2567.2007.02555.x

Babonis, L. S., Miller, S. N., & Evans, D. H. (2011). Renal responses to salinity change in snakes with and without salt glands. *The Journal of Experimental Biology*, *214*, 2140–2156. https://doi.org/10.1242/jeb.052852

Bailey, I. W. (1916). The structure of the bordered pits of conifers and its bearing upon the tension hypothesis of the ascent of sap in plants. *Botanical Gazette*, *62*(2), 133–142. https://doi.org/10.1086/331877

Basile, D. P., Anderson, M. D., & Sutton, T. A. (2012). Pathophysiology of acute kidney injury. *Comparative Physiology*, *2*(2), 1303–1353. https://doi.org/10.1002/cphy.c110041

Baudisch, A. (2005). Hamilton's indicators of the force of selection. *Proceedings of the National Academy of Sciences of the United States of America*, *102*(23), 8263–8268. https://doi.org/10.1073/pnas.0502155102

Baudisch, A. (2011). The pace and shape of ageing. *Methods in Ecology and Evolution*, *2*(4), 375–382. https://doi.org/10.1111/j.2041-210X.2010.00087.x

Baudisch, A., Salguero-Gómez, R., Jones, O. R., Wrycza, T., Mbeau-Ache, C., Franco, M., & Colchero, F. (2013). The pace and shape of senescence in angiosperms. *Journal of Ecology*, *101*(3), 596–606. https://doi.org/10.1111/1365-2745.12084

Baudisch, A., & Stott, I. (2019). Pace and shape perspective on fertility. *Methods in Ecology and Evolution*, *10*, 1941–1951. https://doi.org/10.1111/2041-210X.13289

Baudisch, A., & Vaupel, J. W. (2012). Getting to the root of ageing. *Science*, *338*(6107), 618–619. https://doi.org/10.1126/science.12264467

Bentley, P. J. (1971). *Endocrines and osmoregulation: A comparative account in vertebrates*. Heidelberg, Germany: Springer.

Berger, V., Lemaître, J., Dupont, P., Allainé, D., Gaillard, J., & CohasBeuchat, A. (2016). Age-specific survival in the socially monogamous alpine marmot (*Marmota marmota*): Evidence of senescence. *Journal of Mammology*, *271*(3), 992–1000. https://doi.org/10.1093/jmammal/gyw028

Bernard, C., Compagnoni, A., & Salguero-Gómez, R. (2019). Data from: Testing Finch's hypothesis: The role of organismal modularity on the escape from actuarial senescence. *Dryad Digital Repository*, https://doi.org/10.5061/dryad.7pvmcvdpk

Beuchat, C. A. (1996). Structure and concentrating ability of the mammalian kidney: Correlations with habitat. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, *271*(1), R157–R179.

Bodnar, A. G. (2009). Marine invertebrates as models for aging research. *Experimental Gerontology*, *44*(8), 477–484. https://doi.org/10.1016/j.exger.2009.05.001

Budisch, A., & Vaupel, J. W. (2010) Senescence vs. sustenance: Evolutionary-demographic models of aging. *Demographic Research*, *23*, 655–668. https://doi.org/10.4054/DemRes.2010.23.23

Budras, K. D., Hullinger, R. L., & Rautenfeld, D. B. V. (1987). Lymph heart musculature in birds. *Journal of Morphology*, *191*(1), 77–87. https://doi.org/10.1002/jmor.1051910108

Buffenstein, R. (2008). Negligible senescence in the longest living rodent, the naked mole-rat: Insights from a successfully aging species. *Journal of Comparative Physiology B*, *178*(4), 439–445. https://doi.org/10.1007/s00360-007-0237-5

Buss, L. G. (1987). *The evolution of individuality*. Princeton, NJ: Princeton University Press.

Bythell, J. C., Brown, B. E., & Kirkwood, T. B. L. (2018). Do reef corals age? *Biological Reviews*, *93*(2), 1192–1202. https://doi.org/10.1111/brv.12391

Cailliet, G. M., Andrews, A. H., Burton, E. J., Watters, D. L., Kline, D. E., & Ferry-Graham, L. A. (2001). Age determination and validation studies of marine fishes: Do deep-dwellers live longer? *Experimental Gerontology*, *36*(4–6), 739–764. https://doi.org/10.1016/S0531-5565(00)00239-4

Carey, J. R. (2001). Demographic mechanisms for the evolution of long life in social insects. *Experimental Gerontology*, *36*(4–6), 713–722. https://doi.org/10.1016/S0531-5565(00)00237-0

Carlquist, S. (1975). *Ecological strategies of xylem evolution*. Berkeley, CA: University of California Press.

Caswell, H. (2007). Extrinsic mortality and the evolution of senescence. *Trends in Ecology & Evolution*, *22*(4), 173–174. https://doi.org/10.1016/j.tree.2007.01.006

Caswell, H. (2001). *Matrix population models*. Sunderland, Massachusetts: Sinauer Associates.

Caswell, H. (2011). *Matrix population models: Construction, analysis, and interpretation* (2nd ed.). Sunderland, UK: Sinauer Associates Inc.

Caswell, H., & Salguero-Gómez, R. (2013). Age, stage and senescence in plants. *Journal of Ecology*, *101*(3), 585–595. https://doi.org/10.1111/1365-2745.12088

Catchpole, E. A., Morgan, B. J. T., Coulson, T. N., Freeman, S. N., & Albon, S. D. (2000). Factors influencing soay sheep survival. *Journal of the Royal Statistical Society. Series C (Applied Statistics)*, *49*(4), 453–472. https://doi.org/10.1111/1467-9876.00205

Chapman, G. (1981). Individuality and modular organisms. *Biological Journal of the Linnean Society*, *15*, 177–183. https://doi.org/10.1111/j.1095-8312.1981.tb00757.x

Christman, M. A., Sperry, J. S., & Adler, F. R. (2009). Testing the "rare pit" hypothesis for xylem cavitation resistance in three species of acer. *New Phytologist*, *182*(3), 664–674. https://doi.org/10.1111/j.1469-8137.2009.02776.x

Christman, M. A., Sperry, J. S., & Smith, D. D. (2012). Rare pits, large vessels and extreme vulnerability to cavitation in a ring-porous tree species. *New Phytologist*, *193*(3), 713–720. https://doi.org/10.1111/j.1469-8137.2011.03984.x

Clarke, E. (2011). Plant individuality and multilevel selection theory. In K. Sterelny, & B. Calcott (Eds.), In the major transitions revisited. Cambridge, MA: MIT Press. https://doi.org/10.7551/mitpress/9780262015240.003.0012

Cohen, A. A. (2018). Aging across the tree of life: The importance of a comparative perspective for the use of animal models in aging. *Biochimica Et Biophysica Acta - Molecular Basis of Disease*, *1864*(9), 2680–2689. https://doi.org/10.1016/j.bbadis.2017.05.028

Cohen, A. A. (2016). Complex systems dynamics in aging: New evidence, continuing questions. *Biogerontology*, *17*(1), 205–220.

Cohen, A., Milot, E., Yong, J., Seplaki, C. L., Fulop, T., Bandeen-Roche, K., & Fried, L. (2013). A novel statistical approach shows evidence for multi-system physiological dysregulation during aging. *Mechanisms of Ageing and Development*, *134*(3–4), 110–117. https://doi.org/10.1016/j.mad.2013.01.004

Comfort, A. (1979). *The biology of senescence* (3rd ed.). Edinburgh and London: Churchill Livingstone.

Compagnoni, A., & Adler, P. B. (2014). Warming, soil moisture, and loss of snow increase *Bromus tectorum*'s population growth rate. *Elementa: Science of the Anthropocene*, *2*, 000020. https://doi.org/10.12952/journal.elementa.000020

Cook, R. E. (1979). Asexual reproduction: A further consideration. *The American Naturalist*, *113*, 769–772. https://doi.org/10.1086/283435

de Witte, L. C., & Stöcklin, J. (2010). Longevity of clonal plants: Why it matters and how to measure it. *Annals of Botony*, *106*, 859–870. https://doi.org/10.1093/aob/mcq191

Deevey, E. S. Jr. (1947). Life tables for natural populations of animals. *Quarterly Review of Biology*, *22*, 283–314.

Denley, D., & Metaxas, A. (2016). Quantifying mortality of modular organisms: A comparison of partial and whole-colony mortality in a colonial bryozoan. *Ecosphere*, *7*(10), e01483. https://doi.org/10.1002/ecs2.1483

DeWoody, J., Rowe, C. A., Hipkins, V. D., & Mock, K. E. (2009). "Pando" lives: Molecular genetic evidence of a giant aspen clone in central Utah. *Western North American Naturalist*, *68*(4), 493–497. https://doi.org/10.3398/1527-0904-68.4.493

DiFrisco, J. (2018). Token physicalism and functional individuation. *European Journal for Philosophy of Science*, *8*(3), 309–329.

Ehleringer, J. R. (1980). *Adaptations of plants to water and high temperature stress* (N. C. Turner & P. J. Kramer, Eds.). New York, NY: Wiley-Interscience.

Ehrlén, J., & Lehtilä, K. (2002). How perennial are perennial plants? *Oikos*, *98*(2), 308–322. https://doi.org/10.1034/j.1600-0706.2002.980212.x

Enquist, B. J., Condit, R., Peet, B., Schildhauer, M., Thiers, B., & BIEN, Working Group.(2009). Cyberinfrastructure for an integrated botanical information network to investigate the ecological impacts of global climate change on plant biodiversity. 1–32. Retrieved from www.iplantcollaborative.org/sites/default/files/BIEN_White_Paper.pdf

Esteve-Altava, B. (2017). In search of morphological modules: A systematic review. *Biological Reviews*, *92*(3), 1332–1347. https://doi.org/10.1111/brv.12284

Evans, D. H. (2008) Teleost fish osmoregulation: What have we learned since August Krogh, Homer Smith, and Ancel Keys. *American Journal of Physiology – Regulatory, Integrative and Comparative Physiology*, *295*(2), 704–713; erratum 295(4), 1359. https://doi.org/10.1152/ajpregu.90337.2008

Evans, D. H. (2010). A brief history of the study of fish osmoregulation: The central role of the Mt. Desert Island Biological Laboratory. *Frontiers in Physiology*, *1*(13), 1–10. https://doi.org/10.3389/fphys.2010.00013

Ewers, F. W. (1985). Xylem structure and water conduction in conifer trees, dicot trees, and liana. *IAWA Journal*, *6*, 309–317.

Ewers, F. W., Ewers, J., Jacobsen, A. L., & López-Portillo, J. (2007). Vessel redundancy: Modeling safety in numbers. *IAWA Journal*, *28*(4), 373–388. https://doi.org/10.1163/22941932-90001650

Finch, C. E. (1990). *Longevity, senescence, and the genome*. Chicago, IL: University of Chicago Press.

Finch, C. E. (2009). Update on slow aging and negligible senescence – A mini-review. *Gerontology*, *55*(3), 307–313. https://doi.org/10.1159/000215589

Foley, R. N., & Ibrahim, H. N. (2010). Long-term outcomes of kidney donors. *Nephrology and Hypertension*, *19*(2), 129–133. https://doi.org/10.1097/MNH.0b013e328335f9f4

Gaillard, J. M., Delorme, D., Boutin, J. M., VanLaere, G., Boisaubert, B., & Pradel, R. (1993). Roe deer survival patterns – A comparative analysis of contrasting populations. *Journal of Animal Ecology*, *62*(4), 778–791.

Gardner, S. N., & Mangel, M. (1997). When can a clonal organism escape senescence? *The American Naturalist*, *150*(4), 462–490. https://doi.org/10.1086/286076

García, M. B., Espadaler, X., & Olesen, J. M. (2012). Extreme reproduction and survival of a true cliffhanger: The endangered plant Borderea chouardii (Dioscoreaceae). *PLoS ONE*, *7*(9), e44657.

Gavrilov, L. A., & Gavrilova, N. S. (2001). The reliability theory of aging and longevity. *Journal of Theoretical Biology*, *213*(4), 527–545. https://doi.org/10.1006/jtbi.2001.2430

Gómez, J. M. (2010). Aging in bacteria, immortality or not-a critical review. *Current Aging Science*, *3*(3), 198–218. https://doi.org/10.2174/1874609811003030198

Griffith, A. B., Salguero-Gómez, R., Merow, C., & Mahon, S. (2016). Demography beyond the population. *Journal of Ecology*, *104*, 271–280. https://doi.org/10.1111/1365-2745.12547

Guerrero-Meseguer, L., Sanz-Lázaro, C., & Marín, A. (2018). Understanding the sexual recruitment of one of the oldest and largest organisms on Earth, the seagrass *Posidonia oceanica*. *PLoS ONE*, *13*(11), e0207345. https://doi.org/10.1371/journal.pone.0207345

Gutiérrez, J. S. (2014). Living in environments with contrasting salinities: A review of physiological and behavioural responses in waterbirds. *Ardeola*, *61*(2), 233–256. https://doi.org/10.13157/arla.61.2.2014.233

Haley, P. J. (2016). The lymphoid system: A review of species differences. *Journal of Toxicologic Pathology*, *30*(2), 111–123. https://doi.org/10.1293/tox.2016-0075

Hamady, L. L., Natanson, L. J., Skomal, G. B., & Thorrold, S. R. (2014). Vertebral bomb radiocarbon suggests extreme longevity in white sharks. *PLoS ONE*, *9*(1), e84006.

Hamilton, W. D. (1966). The moulding of senescence by natural selection. *Journal of Theoretical Biology*, *12*(1), 12–45. https://doi.org/10.1016/0022-5193(66)90184-6

Harper, J. L. (1980). Plant demography and ecological theory. *Oikos*, *35*(2), 244–253. https://doi.org/10.2307/3544432

Harper, J. L., & Bell, A. D. (1979). The population dynamics of growth form in organisms with modular construction. In R. M. Anderson, B. D. Turner, & L. R. Taylor (Eds.), *"Population dynamics" 20th symposium British ecological society* (pp. 29–52). Oxford, UK: Blackwell Publishing.

Hedrick, M. S., Hillman, S. S., Drewes, R. C., & Withers, P. C. (2013). Lymphatic regulation in nonmammalian vertebrates. *Journal of Applied Physiology*, *115*(3), 297–308. https://doi.org/10.1152/japplphysiol.00201.2013

Hickey, A. J., Juellig, M., Aitken, J., Loomes, K., Hauber, M. E., & Phillips, A. R. (2012). Birds and longevity: Does flight driven aerobicity provide an oxidative sink? *Ageing Research Reviews*, *11*(2), 242–253.

Hildebrandt, J.-P. (2001). Coping with excess salt: Adaptive functions of extrarenalosmoregulatory organs in vertebrates. *Zoology*, *104*(3), 209–220. https://doi.org/10.1078/0944-2006-00026

Huang, L. H., Lavine, K. J., & Randolph, G. J. (2017). Cardiac lymphatic vessels, transport, and healing of the infarcted heart. *JACC: Basic to Translational Science*, *2*(4), 477–483. https://doi.org/10.1016/j.jacbts.2017.02.005

Ishii, H., Ford, D. E., & Dinnie, C. E. (2001). The role of epicormic shoot production in maintaining foliage in old *Pseudotsuga menziesii* (Douglas-fir) trees II. Basal reiteration from older branch axes. *Canadian Journal of Botany*, *80*(9), 916–926. https://doi.org/10.1139/b02-080

Ishii, H. T., Ford, E. D., & Kennedy, M. C. (2007). Physiological and ecological implications of adaptive reiteration as a mechanism for crown maintenance and longevity. *Tree Physiology*, *27*(3), 455–462. https://doi.org/10.1093/treephys/27.3.455

Iversen, C. M., McCormack, M. L., Powell, A. S., Blackwood, C. B., Freschet, G. T., Kattge, J., … Violle, C. (2017). A global fine-root ecology database to address below-ground challenges in plant ecology. *The New Phytologist*, *215*(1), 15–26. https://doi.org/10.1111/nph.14486

Jin, K. (2010). Modern biological theories of aging. *Aging and Disease*, *1*(2), 72–74.

Jones, O. R., Scheuerlein, A., Salguero-Gómez, R., Camarda, C. G., Schaible, R., Casper, B. B., … Vaupel, J. W. (2014). Diversity of ageing across the tree of life. *Nature*, *505*(7482), 169–173. https://doi.org/10.1038/nature12789

Kampmeier, O. F. (1969). *Evolution and comparative morphology of the lymphatic system*. Springfield, IL: Charles C. Thomas.

Kasiske, B. L., Anderson-Haag, T., Ibrahim, H. N., Pesavento, T. E., Weir, M. R., Nogueira, J. M., … Steffes, M. W. (2013). A prospective controlled study of kidney donors: Baseline and 6-month follow-up. *American Journal of Kidney Diseases*, *62*(3), 577–586. https://doi.org/10.1053/j.ajkd.2013.01.027.A

Kattge, J., Díaz, S., Lavorel, S., Prentice, I. C., Leadley, P., Bönisch, G., … Wirth, C. (2011). TRY – A global database of plant traits. *Global Change Biology*, *17*(9), 2905–2935. https://doi.org/10.1111/j.1365-2486.2011.02451.x

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

Kirkwood, T. B. L. (1977). Evolution of ageing. *Nature*, *270*(5635), 301–304. https://doi.org/10.1038/270301a0

Kirkwood, T. B. L. (2005). Understanding the odd science of aging. *Cell*, *120*(4), 437–447. https://doi.org/10.1016/j.cell.2005.01.027

Kirkwood, T. B. L., & Austad, S. (2000). Why do we age? *Nature*, *408*(6809), 233–238. https://doi.org/10.1038/35041682

Kirkwood, T. B. L., & Rose, M. R. (1991). Evolution of senescence – Late survival sacrificed for reproduction. *Cell*, *332*(1262), 15–24. https://doi.org/10.1098/rstb.1991.0028

Koch, G. W., Sillett, S. C., Jennings, G. M., & Davis, S. D. (2004). The limits to tree height. *Nature*, *428*(6985), 851–854. https://doi.org/10.1038/nature02417

Kozlowski, T. T. (1973). *Shedding of plant parts*. New York, NY: Academic Press.

Kramer, B. H., Schrempf, A., Scheuerlein, A., & Heinze, J. (2015). Ant colonies do not trade-off reproduction against maintenance. *PLoS ONE*, *10*(9), 1–13. https://doi.org/10.1371/journal.pone.0137969

Kültz, D. (2015). Physiological mechanisms used by fish to cope with salinity stress. *Journal of Experimental Biology*, *218*(12), 1907–1914. https://doi.org/10.1242/jeb.118695

Kurtén, B. (1953). On the variation and population dynamics of fossil and recent mammal populations. *Acta Zoologica Fennica*, *76*, 1–122.

Lacey, E. P. (1986). Onset of reproduction in plants: Size-versus age-dependency. *Trends in Ecology and Evolution*, *1*(3), 72–75. https://doi.org/10.1016/0169-5347(86)90021-2

Lanner, R. M., & Connor, K. F. (2001). Does bristlecone pine senesce? *Experimental Gerontology*, *36*(4–6), 675–685. https://doi.org/10.1016/S0531-5565(00)00234-5

Le Bourg, E. (2001). A mini-review of the evolutionary theories of aging. *Demographic Research*, *4*(1), 1–28. https://doi.org/10.4054/demres.2001.4.1

Lemaître, J., & Gaillard, J. (2017). Reproductive senescence: New perspectives in the wild. *Biological Reviews*, *92*, 2182–2199. https://doi.org/10.1111/brv.12328

Leslie, P. H., & Ranson, R. M. (1940). The mortality, fertility and rate of natural increase of the vole (*Microtus agrestis*) as observed in the laboratory. *Journal of Animal Ecology*, *9*, 27–52.

Leslie, P. H., Tener, T. S., Vizoso, M., & Chitty, H. (1955). The longevity and fertility of the Orkney vole, *Microtus orcadensis*, as observed in the laboratory. *Proceedings of the Zoological Society of London.*, *125*, 115–125.

Li, Q., Wang, S., Milot, E., Bergeron, P., Ferrucci, L., Fried, L. P., & Cohen, A. A. (2015). Homeostatic dysregulation proceeds in parallel in multiple physiological systems. *Aging Cell*, *14*(6), 1103–1112. https://doi.org/10.1111/acel.12402

Loison, A., Festa-Bianchet, M., Gaillard, J. M., Jorgenson, J. T., & Jullien, J. M. (1999). Age-specific survival in five populations of ungulates: Evidence of senescence. *Ecology*, *80*(8), 2539–2554.

López-Otín, C., Blasco, M. A., Partridge, L., Serrano, M., & Kroemer, G. (2013). The hallmarks of ageing. *Cell*, *153*, 1194–1217. https://doi.org/10.1016/j.cell.2013.05.039

Mackie, G. O. (1986). From aggregates to integrates: Physiological aspects of modularity in colonial animals. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, *113*(1159), 175–196.

Magalhaes, J. D., & Costa, J. (2009). A database of vertebrate longevity records and their relation to other life-history traits. *Journal of Evolutionary Biology*, *22*, 1770–1774. https://doi.org/10.1111/j.1420-9101.2009.01783.x

Maherali, H., Pockman, W. T., & Jackson, R. B. (2004). Adaptive variation in the vulnerability of woody plants to xylem cavitation. *Ecology*, *85*(8), 2184–2199. https://doi.org/10.1890/02-0538

Maluf, N. S. R., & Gassman, J. J. (1998). Kidneys of the killerwhale and significance of reniculism. *Anatomical Record*, *250*(1), 34–44. https://doi.org/10.1002/(SICI)1097-0185(199801)250:1<34:AID-AR4>3.0.CO;2-E

Mangel, M., Kindsvater, H. K., & Bonsall, M. B. (2007). Evolutionary analysis of life span, competition, and adaptive radiation, motivated by the pacific rockfishes (Sebastes). *Evolution*, *61*(5), 1208–1224. https://doi.org/10.1111/j.1558-5646.2007.00094.x

Martínez, D. E. (1998). Mortality patterns suggest lack of senescence in hydra. *Experimental Gerontology*, *33*(3), 217–225. https://doi.org/10.1016/S0531-5565(97)00113-7

Martínez, D. E., & Levinton, J. S. (1992). Asexual metazoans undergo senescence. *Proceedings of the National Academy of Sciences of the United States of America*, *89*, 9920–9923. https://doi.org/10.1073/pnas.89.20.9920

Matthes, U., Kelly, P., Ryan, C. E., & Larson, D. W. (2002). The formation and possible ecological function of stem strips in *Thuja occidentalis*. *International Journal of Plant Sciences*, *163*(6), 949–958.

Medawar, P. B. (1952). *An unsolved problem in biology*. London, UK: H.K. Lewis.

Meier, A. R., Saunders, M. R., & Michler, C. H. (2012). Epicormic buds in trees: A review of bud establishment, development and dormancy release. *Tree Physiology*, *32*(5), 565–584. https://doi.org/10.1093/treephys/tps040

Mock, K. E., Rowe, C. A., Hooten, M. B., Dewoody, J., & Hipkins, V. D. (2008). Clonal dynamics in western North American aspen (*Populus tremuloides*). *Molecular Ecology*, *17*(22), 4827–4844. https://doi.org/10.1111/j.1365-294X.2008.03963.x

Monaghan, P., Charmantier, A., Nussey, D. H., & Ricklefs, R. E. (2008). The evolutionary ecology of senescence. *Functional Ecology*, *22*(3), 371–378. https://doi.org/10.1111/j.1365-2435.2008.01418.x

Monaghan, P., Metcalfe, N. B., & Torres, R. (2009). Oxidative stress as a mediator of life history trade-offs: Mechanisms, measurements and interpretation. *Ecology Letters*, *12*(1), 75–92. https://doi.org/10.1111/j.1461-0248.2008.01258.x

Morris, W. F., & Doak, D. F. (1998). Life history of the long-lived gynodioecious cushion plant *Silene acaulis* (Caryophyllaceae), inferred from size-based population projection matrices. *American Journal of Botany*, *85*(6), 784–793. https://doi.org/10.2307/2446413

Munk, K. M. (2001). Maximum ages of groundfishes in waters off Alaska and British Columbia and considerations of age determination. Reprinted from the Alaska Fishery Research Bulletin, 8(1), Retrieved from http://www.state.ak.us/adfg/geninfo/pubs/afrb/afrbhome.htm

Munné-Bosch, S. (2008). Do perennials really senesce? *Trends in Plant Science*, *13*(5), 216–220. https://doi.org/10.1016/j.tplants.2008.02.002

Murie, A. (1944). The wolves of Mount McKinley (p. 5).Fauna National Parks U.S., Fauna Series 5.

Nielsen, J., Hedeholm, R. B., Heinemeier, J., Bushnell, P. G., Christiansen, J. S., Olsen, J., … Steffensen, J. F. (2016). Eye lens radiocarbon reveals centuries of longevity in the Greenland shark (*Somniosus microcephalus*). *Science*, *353*(6300), 702–704. https://doi.org/10.1126/science.aaf1703

Nikles, S. A., & Heath, T. J. (1992). Pathways of lymph flow through intestinal lymph nodes in the horse. *The Anatomical Record*, *229*(4), 521–524.

Nussey, D. H., Coulson, T., Festa-Bianchet, M., & Gaillard, J. M. (2008). Measuring senescence in wild animal populations: Towards a longitudinal approach. *Functional Ecology*, *22*(3), 393–406. https://doi.org/10.1111/j.1365-2435.2008.01408.x

Nussey, D. H., Froy, H., Lemaitre, J., & Gaillard, J. (2013). Senescence in natural populations of animals: Widespread evidence and its implications for bio-gerontology. *Ageing Research Reviews.*, *12*(1), 214–225. https://doi.org/10.1016/j.arr.2012.07.004.Senescence

Oakley, T. H. (2017). Furcation and fusion: The phylogenetics of evolutionary novelty. *Developmental Biology*, *431*(1), 69–76.

Orians, C. M., Babst, B., & Zanne, A. E. (2005). Vascular constraints and long distance transport in dicots. In *Vascular transport in plants* (pp. 355–371). Cambridge, Massachusetts: Academic Press.

Orive, M. E. (1995). Senescence in organisms with clonal reproduction and complex life histories. *The American Naturalist*, *145*(1), 90–108. https://doi.org/10.1086/285729

Orme, D. (2013). The caper package: Comparative analysis of phylogenetics and evolution in R.

Ortiz, R. M. (2001). Osmoregulation in marine mammals. *The Journal of Experimental Biology*, *204*(11), 1831–1844. https://doi.org/10.1002/JMOR.1051670103

Palm, F., & Nordquist, L. (2011). Renal oxidative stress, oxygenation, and hypertension. *American Journal of Physiology-Regulatory, Integrative*

and Comparative Physiology, 301(5), 1229–1241. https://doi.org/10.1152/ajpregu.00720.2010

Paradis, E., Claude, J., & Strimmer, K. (2004). APE: Analyses of phylogenetics and evolution in R language. Bioinformatics, 20(2), 289–290. https://doi.org/10.1093/bioinformatics/btg412

Patnaik, B. K., Mahapatro, N., & Jena, B. S. (1994). Ageing in fishes. Gerontology, 40(24), 113–132.

Pepper, J. W., & Herron, M. D. (2008). Does biology need an organism concept. Biological Reviews, 83(4), 621–627. https://doi.org/10.1111/j.1469-185X.2008.00057.x

Péron, G., Gimenez, O., Charmantier, A., Gaillard, J.-M., & Crochet, P.-A. (2010). Age at the onset of senescence in birds and mammals is predicted by early-life performance. Proceedings of the Royal Society B: Biological Sciences, 277(1695), 2849–2856. https://doi.org/10.1098/rspb.2010.0530

Petralia, R. S., Mattson, M. P., & Yao, P. J. (2014). Immortality. Ageing Research Reviews, 66–82, https://doi.org/10.1016/j.arr.2014.05.003

Pittermann, J., Lynn, L., Choat, B., Dawson, T. E., Stuart, S. A., & Jansen, S. (2010). The relationships between xylem safety and hydraulic efficiency in the Cupressaceae: The evolution of pit membrane form and function. Plant Physiology, 153(4), 1919–1931. https://doi.org/10.1104/pp.110.158824

Pletcher, S. D., Houle, D., & Curtsinger, J. W. (1998). Age-specific properties of spontaneous mutations affecting mortality in Drosophila melanogaster. Genetics, 148(1), 287–303.

Podlutsky, A. J., Khritankov, A. M., Ovodov, N. D., & Austad, S. N. (2005). A new field record for bat longevity. Library, 60(11), 1366–1368. https://doi.org/10.1093/gerona/60.11.1366

Pradeu, T. (2016). Organisms or biological individuals? Combining physiological and evolutionary individuality. Biology & Philosophy, 31(6), 797–817. https://doi.org/10.1007/s10539-016-9551-1

Promislow, D. E., Tatar, M., Pletcher, S., & Carey, J. R. (1999). Below-threshold mortality: Implications for studies in evolution, ecology and demography. Journal of Evolutionary Biology, 12(2), 314–328. https://doi.org/10.1046/j.1420-9101.1999.00037.x

R Core Team. (2012). R: A language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing. Retrieved from http://www.R-project.org/. ISBN 3-900051-07-0.

Reimschuessel, R. (2001). A fish model of renal regeneration and development. ILAR Journal, 42(4), 285–291. https://doi.org/10.1093/ilar.42.4.285

Ricklefs, R. E. (2008). The evolution of senescence from a comparative perspective. Functional Ecology, 22(3), 379–392. https://doi.org/10.1111/j.1365-2435.2008.01420.x

Roper, M., Capdevila, P., & Salguero-Gómez, R. (2019). Senescence: still an unsolved problem of biology. https://doi.org/10.1101/739730

Rose, M. R. (1991). Evolutionary biology of aging. New York, NY: Oxford University Press.

Royer, D. L., Wilf, P., Janesko, D. A., Kowalski, E. A., & Dilcher, D. L. (2005). Correlations of climate and plant ecology to leaf size and shape: Potential proxies for the fossil record. American Journal of Botany, 92(7), 1141–1151. https://doi.org/10.3732/ajb.92.7.1141

Salguero-Gómez, R. (2014). The COMPADRE plant matrix database: An open online repository for plant demography. Journal of Ecology, 103(1), 202–218. https://doi.org/10.1111/1365-2745.12334

Salguero-Gómez, R. (2017). Implications of clonality for ageing research. Evolutionary Ecology, 32(1), 9–28. https://doi.org/10.1007/s10682-017-9923-2

Salguero-Gómez, R., Jones, O. R., Archer, C. R., Bein, C., de Buhr, H., Farack, C., … Vaupel, J. W. (2016). COMADRE: A global database of animal demography. Journal of Animal Ecology, 85(2), 371–384. https://doi.org/10.1111/1365-2656.12482

Salguero-Gómez, R., Jones, O. R., Archer, C. A., Buckley, Y. M., Che-Castaldo, J., Caswell, C., … Vaupel, J. W. (2015). The COMPADRE plant matrix database: An online repository for plant population dynamics. Journal of Ecology, 103(1), 202–218. https://doi.org/10.1111/1365-2745.12334

Salguero-Gómez, R., Shefferson, R. P., & Hutchings, M. J. (2013). Plants do not count… or do they? New perspectives on the universality of senescence. Journal of Ecology, 101(3), 545–554. https://doi.org/10.1111/1365-2745.12089

Schaible, R., Scheuerlein, A., Dańko, M. J., Gampe, J., Vaupel, J. W., & Vaupel, J. W. (2015). Constant mortality and fertility over age in Hydra. Proceedings of the National Academy of Sciences of the United States of America, 112(51), 201521002. https://doi.org/10.1073/pnas.1521002112

Schenk, H. J., Espino, S., Goedhart, C. M., Nordenstahl, M., Cabrera, H. I., & Jones, C. S. (2008). Hydraulic integration and shrub growth form linked across continental aridity gradients. Proceedings of the National Academy of Sciences of the United States of America, 105(32), 11248–11253. https://doi.org/10.1073/pnas.0804294105

Schmidt-Nielsen, K. (1960). The salt-secreting gland of marine birds. Circulation, 21(5), 955–967. https://doi.org/10.1161/01.CIR.21.5.955

Schmidt-Nielsen, K., & Fange, R. (1958). Salt glands in marine reptiles. Nature, 182(4638), 783–785.

Seiler, C. (2010). The human coronary collateral circulation. European Journal of Clinical Investigation, 40(5), 465–476. https://doi.org/10.1111/j.1365-2362.2010.02282.x

Shadwick, R. E., Farrell, A. P., & Brauner, C. J. (2016). Physiology of elasmobranch fishes: Internal processes. (A. Farrell & C. Brauner, Eds.). London, UK: Elsevier.

Shefferson, R. P., Jones, O. R., & Salguero-Gómez, R. (2017). The evolution of senescence in the tree of life. Cambridge, UK: Cambridge University Press.

Sillett, S. C., Van Pelt, R., Koch, G. W., Ambrose, A. R., Carroll, A. L., Antoine, M. E., & Mifsud, B. M. (2010). Increasing wood production through old age in tall trees. Forest Ecology and Management, 259(5), 976–994. https://doi.org/10.1016/j.foreco.2009.12.003

Silvertown, J., Franco, M., & Perez-Ishiwara, R. (2001). Evolution of senescence in iteroparous perennial plants. Evolutionary Ecology Research, 3(4), 393–412.

Sköld, H. N., & Obst, M. (2011). Potential for clonal animals in longevity and ageing studies. Biogerontology, 12(5), 387–396. https://doi.org/10.1007/s10522-011-9333-8

Smith, H. S. (1929). The composition of the body fluids of elasmobranchs. Journal of Biological Chemistry, 81, 407–419.

Sperry, J. S., Sano, Y., Sikkema, E. H., Feild, T. S., & Hacke, U. G. (2007). Hydraulic consequences of vessel evolution in angiosperms. International Journal of Plant Sciences, 168(8), 1127–1139. https://doi.org/10.1086/520726

Sprugel, D. G., Hinckley, T. M., & Schaap, W. (1991). The theory and practice of branch autonomy. Annual Review of Ecology, Evolution, and Systematics., 22, 309–334.

Stearns, S. C. (1992). The evolution of life histories. New York, New York: Oxford University Press.

Stenvinkel, P., Painer, J., Kuro-o, M., Lanaspa, M., Arnold, W., Ruf, T., … Johnson, R. J. (2018). Novel treatment strategies for chronic kidney disease: Insights from the animal kingdom. Nature Reviews Nephrology, 14(4), 265–284. https://doi.org/10.1038/nrneph.2017.169

Tanner, J. E. (2001). The influence of clonality on demography: Patterns in expected longevity and survivorship. Ecology, 82(7), 1971–1981. https://doi.org/10.1890/0012-9658(2001)082[1971:TIOCOD]2.0.CO;2

Thomas, H. (2002). Ageing in plants. Mechanisms of Ageing and Development, 123, 747–753.

Thomas, H. (2013). Senescence, ageing and death of the whole plant. New Phytologist, 197, 696–711. https://doi.org/10.1111/nph.12047

Towle, D. W., & Weihrauch, D. (2001). Osmoregulation by gills of euryhaline crabs. American Zoologist, 780, 770–780.

Tuomi, J., & Vuorisalo, T. (1989). What are the units of selection in modular organisms? T. Wiley on Behalf of Nordic Society Oikos, 54(2), 227–233. https://doi.org/10.2307/3565271

Turke, P. W. (2008). William's theory of senescence. The Quarterly Review of Biology, 83(3), 243–256.

Tyree, M. T., & Sperry, J. S. (1989). Vulnerability of xylem to cavitation and embolism. *Annual Review of Plant Physiology and Plant Molecular Biology*, *40*, 19–36.

Vaupel, J. W., Baudisch, A., Dölling, M., Roach, D. A., & Gampe, J. (2004). The case for negative senescence. *Theoretical Population Biology*, *65*(4), 339–351. https://doi.org/10.1016/j.tpb.2003.12.003

Venturas, M. D., Sperry, J. S., & Hacke, U. G. (2017). Plant xylem hydraulics: What we understand, current research, and future challenges. *Journal of Integrative Plant Biology*, *59*(6), 356–389. https://doi.org/10.1111/jipb.12534

Vuorisalo, T. O., & Mutikainen, P. (Eds.) (1999). *Life history evolution in plants*. Berlin, Germany: Springer Science & Business Media.

Vuorisalo, T., Tuomi, J., & Turku, S. (1986). Criteria for ecological division. *Wiley on Behalf of Nordic Society Oikos*, *47*(3), 382–385. https://www.jstor.org/stable/3565452

Watson, M. A. (1986). Integrated physiological units in plants. *Trends in Ecology and Evolution*, *1*(5), 119–123. https://doi.org/10.1016/0169-5347(86)90005-4

Watson, M. A., & Casper, B. B. (1984). Morphogenetic constraints on patterns of carbon distribution in plants. *Annual Review of Ecology, Evolution, and Systematics*, *15*, 233–258. https://doi.org/10.1146/annurev.es.15.110184.001313

Weihrauch, D., & O'Donnell, M. J. (2015). Links between osmoregulation and nitrogen-excretion in insects and crustaceans. *Integrative and Comparative Biology*, *55*(5), 816–829. https://doi.org/10.1093/icb/icv013

Wee, P. T., Tegzes, A., & Donker, A. J. M. (1990). Renal reserve filtration capacity before and after kidney donation. *Journal of Internal Medicine*, *228*(4), 393–399.

Weiner, J., Campbell, L. G., Pino, J., & Echarte, L. (2009). The allometry of reproduction within plant populations. *Journal of Ecology*, *97*(6), 1220–1233. https://doi.org/10.1111/j.1365-2745.2009.01559.x

Weiss, K. C. B., & Ray, C. A. (2019). Unifying functional trait approaches to understand the assemblage of ecological communities: Synthesizing taxonomic divides. *Ecography*, *42*, 1–9. https://doi.org/10.1111/ecog.04387

Welch, J. J., & Waxman, D. (2003). Modularity and the cost of complexity. *Evolution*, *57*(8), 1723–1734.

Wensink, M. J., Caswell, H., & Baudisch, A. (2017). The rarity of survival to old age does not drive the evolution of senescence. *Evolutionary Biology*, *44*(1), 5–10. https://doi.org/10.1007/s11692-016-9385-4

White, J. (1979). The plant as a metapopulation. *Annual Review of Ecology and Systematics*, *10*, 109–145.

White, R. R., & Vijg, J. (2016). Do DNA double-strand breaks drive aging? *Molecular Cell Journal*, *63*(5), 729–738. https://doi.org/10.1016/j.molcel.2016.08.004

Wilkie, M. P. (2002). Ammonia excretion and urea handling by fish gills: Present understanding and future research challenges. *Journal of Experimental Zoology*, *293*(3), 284–301. https://doi.org/10.1002/jez.10123

Williams, G. C. (1957). Pleiotropy, natural selection, and the evolution of senescence. *Evolution*, *11*(4), 398–411. https://doi.org/10.1038/mp.2011.138

Williams, P. D., Day, T., Fletcher, Q., & Rowe, L. (2006). The shaping of senescence in the wild. *Trends in Ecology and Evolution*, *21*(8), 458–463. https://doi.org/10.1016/j.tree.2006.05.008

Williams, R. T. M., & Worthy, G. A. J. (2002). Anatomy and physiology: The challenge of aquatic living. In A. Rus Hoelzel (Ed.), *Marine mammal biology: An evolutionary approach, Chap. 3*. Oxford, UK: Blackwell Science Ltd.

Winston, J. E. (2010). Life in the colonies: Learning the alien ways of colonial organisms. *Integrative and Comparative Biology*, *50*(6), 919–933. https://doi.org/10.1093/icb/icq146

Wood, C. M., Pärt, P., & Wright, P. A. (1995). Ammonia and urea metabolism in relation to gill function and acid-base balance in a marine elasmobranch, the spiny dogfish (*Squalus acanthias*). *The Journal of Experimental Biology*, *198*, 1545–1558. https://doi.org/10.1242/jeb.00510

Wood-Jones, F. (1912). *Corals and Atolls: A history and description of the Keeling-Cocos Islands, with an account of their Fauna and Flora, and a discussion of the method of development and transformation of coral structures in general*. London, UK: Lovell Reeve & Co. https://doi.org/10.5962/bhl.title.46944. Retrieved from https://archive.org/details/coralatollshisto00jone/page/n15

Wright, I. J., Reich, P. B., Westoby, M., Ackerly, D. D., Baruch, Z., Bongers, F., … Villar, R. (2004). The worldwide leaf economics spectrum. *Nature*, *428*(6985), 821–827.

Wright, P. A., & Wood, C. M. (2012). Seven things fish know about ammonia and we don't. *Respiratory Physiology and Neurobiology*, *184*(3), 231–240. https://doi.org/10.1016/j.resp.2012.07.003

Wrycza, T. F., & Baudisch, A. (2014). The pace of aging: Intrinsic time scales in demography. *Demographic Research*, *30*(1), 1571–1590. https://doi.org/10.4054/DemRes.2014.30.57

Wrycza, T. F., Missov, T. I., & Baudisch, A. (2015). Quantifying the shape of aging. *PLoS ONE*, *10*(3), 1–18. https://doi.org/10.1371/journal.pone.0119163

Yokota, E., Kawashima, T., Ohkubo, F., & Sasaki, H. (2008). Comparative anatomical study of the kidney position in amniotes using the origin of the renal artery as a landmark. *Okajimas Folia Anatomica Japonica*, *81*(6), 135–142. https://doi.org/10.2535/ofaj.81.135

Young, A. J. (2018). The role of telomeres in the mechanisms and evolution of life-history trade-offs and ageing. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *373*(1741), 20160452. https://doi.org/10.1098/rstb.2016.0452

Zanne, A. E., Tank, D. C., Cornwell, W. K., Eastman, J. M., Smith, S. A., FitzJohn, R. G., … Beaulieu, J. M. (2014). Three keys to the radiation of angiosperms into freezing environments. *Nature*, *506*(7486), 89–92. https://doi.org/10.1038/nature12872

Zimmerman, M. H. (1983). *Xylem structure and the ascent of Sap*. Berlin, Germany: Springer-Verlag.

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.