Abstract (327/350 words)

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 hypothesised three decades ago that species with the ability to compartmentalise 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 anatomic 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 opportunities related to age-dependent mortality factors. Other areas for

28   further research include the role of modularity in relation to endocrine actions, and the

29   costs of modular anatomies.

30

31   KEYWORDS

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

1 | THE EVITABILITY OF SENESCENCE

Since the late 1970s, three major theories have persisted to explain declines in physiological performance with age, that is *senescence*. These are: (1) Medawar's (1952) theory of mutation accumulation, whereby organisms senesce due to their inability to revert the rate of accumulation of deleterious mutations; (2) 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 (3) 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 center on genetic principles for a discounting of late-life performance (Le Bourg 2001; Promislow et al. 1999; Hamilton 1966). 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 (Turke 2008; Kirkwood and Austad 2000; Kirkwood and Rose 1991; Rose 1991). 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,

62 Hamilton (1966) asserted that actuarial senescence necessarily follows from the

63 exponential growth of population. He mathematically showed that organisms with

64 demographies highly favorable to late-life performance still experience selection bias

65 for early-life performance. Implied here is that populations with non-exponential growth

66 could escape senescence. Williams (1957) described senescence as a transient

67 genetic phenomenon in which genes that present mixed benefits are partially

68 substituted by more adaptive genotypes. According to this theory, variation in the

69 genes and in their influence on one another can generate positive, negligible, and

70 negative senescence. Negative senescence describes a decline in mortality after

71 maturity (*sensu* Vaupel 2004).

72   More recent models of senescence describe how density dependence can

73 accelerate or decelerate senescence as a function of stage-dependent mortality

74 (Abrams 1993). In addition to these models, an important distinction about senescence

75 has been made with respect to cell differentiation. This was implied by Williams' work

76 and has been highlighted by a number of other researchers (Shefferson, Jones, and

77 Salguero-Gómez 2017; Gómez 2010; Turke 2008; Buss 1987). Organisms with

78 undifferentiated cells (thus without soma-germ separation) may not be subject to

79 patterns of senescence assumed under all classical theories for senescence.

80 Surprisingly, the existence of alternative explanations for escaping senescence did not

81 detract from Hamilton's (1966) prediction of universal senescence until recently

82 (Nussey et al. 2013; Baudisch 2005).

83   A systematic review or estimate for how many taxa are accountable to the

84 above-mentioned exceptions does not exist, that we are aware of. We also do not

85 know how the closely proposed mechanisms that release species from senescence

86 relate to particular groups of species. This information can advance our understanding

87   of how much has been explained under existing theory, and can focus future studies.

88   While the conceptual foundations of these exceptions are strong, they have rarely

89   been tested empirically. For example, Vaupel et al. (2004) offer a cogent proof of

90   negative senescence based on growth-related functional performance. They ground

91   theoretical advancements with examples of species that exhibit negligible or negative

92   senescence that are also indeterminate in their growth. Until recently, we were not

93   aware of any work connecting how closely indeterminate growth predicts negligible

94   senescence, nor analysing the existence of confounding influences.

95   Taxonomic bias in early studies of senescence in unitary organisms (e.g.

96   mammals, birds) likely contributed to the notion that senescence is universal (Jones

97   et al. 2014; Monaghan et al. 2008; Shefferson et al. 2017). The idea of universal

98   senescence may also be attractive because cellular ageing, not far afield of

99   senescence, is written about in universal terms (*i.e.*, cellular senescence; noted by

100 Comfort 1979). Statements in recent publications put little distance between cell and

101 organismal finitude: "Organisms wear out, just like machines" (Ricklefs 2008);

102 "[D]amage will accumulate in parallel with cells" (Kirkwood 2005); "Aging and its

103 associated decline in survival and reproductive fitness is an inherent feature of

104 biological systems" (Alper, Bronikowski, and Harper 2015). Studies on ageing focus

105 on a set of cellular and molecular processes that are distinct from those that are the

106 focus of senescence. Ageing processes include telomere shortening (Young 2018);

107 oxidative stress (Monaghan, Metcalfe, and Torres 2009); double-strand break (White

108 and Vijg 2016); and related mechanisms underlying the damage or error theories of

109 aging (Jin 2010; reviewed in connection with senescence by Comfort 1979; Petralia,

110 Mattson, and 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 (Wrycza, Missov, and Baudisch 2015; Wensink, Caswell, and Baudisch 2017). 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 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 up-scaling 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 (Jones et al. 2014; Vaupel et al. 2004; Gardner and Mangel 1997). Many cases of negligible senescence (*i.e.,* mortality [Fig. 1b & e] and/or fertility [the latter not addressed analytically in this review] remaining invariable with age) or even negative senescence (mortality risk decline [Fig. 1e & f] and/or fertility increase with age [again, the latter not addressed in this review, but see Baudisch and Stott 2019] are now known based on reliable data (Pletcher, Houle, and Curtsinger 1998; Finch 2009). 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

136 include Brandt's bat (*Myotis brandtii*; Podlutsky et al. 2005), barn owl (*Tyto alba*;

137 Altwegg, Schaub, and Roulin 2007), greenland shark (*Somniosus microcephalus*;

138 Nielson et al. 2016), rockfishes (Cailliet et al. 2001; Mangel, Kindsvater and Bonsall

139 2007; Munk 2001), ants and termites (Carey 2001), naked mole-rat (*Heterocephalus*

140 *glaber*; Buffenstein 2008), *Hydra* (Schaible et al. 2015; Martínez 1998), bristlecone

141 pine (*Pinus longaeva*; Lanner and Connor 2001), *Borderea pyrenaica* (García,

142 Espadaler, Olesen 2012), and wilcox brush (*Eremophila forrestii*; Erlén and Lehtilä

143 2002).

144   Caleb E. Finch, in his seminal monograph of senescence (1990), brought

145 attention to the few known species that at the time suggested negligible or even

146 negative senescence. In this monograph, Finch posited a novel connection between

147 senescence and anatomy, by probing the question of how modular forms could alter

148 senescence. Finch suggested that if modules age independently or incur injury

149 independently, as physiological units, then they may insulate the composite individual

150 from those effects. Thus, Finch inquired whether modules could lengthen the life of an

151 individual by preventing local injury from translating to injury of the individual. Until

152 recently, testing this hypothesis has been hindered by a dearth of relevant data and

153 substantial literature across many species. Here, we avail of recent advances in

154 histology and physiology to proxy the degree of modularity, and evaluate links between

155 demographic data and actuarial senescence.

156   Here, and in line with classical use, we use the term "modular" to describe

157 individual organisms that are constructed of multiple, repeat sub-units that are

158 individually dispensable without injury to survivorship of the individual (Vuorisalo and

159 Tuomi 1986; Vuorisalo and Mutikainen 1999). Within this framework, the individual

160 can be thought of as a grouping of genetically identical cells which share physiological

161 interdependence (extending from criteria distinctions discussed by Pepper and Herron

162 2008; Pradeu 2016; DiFrisco 2017). This usage allows for minor genetic variation that

163 occurs from random, somatic point mutations. Clones are therefore considered parts

164 of a single individual –as are modules– despite their heightened degree of

165 physiological independence and compartmentation. Alternative ways to define

166 individals may be used on the basis of fundamental evolutionary units, specific

167 physiological properties, or other factors. However, our definition facilitates

168 comparison across broad morphological types and taxonomies, while retaining a

169 demographically coherent unit. Our analysis shows that traits that reflect a higher

170 degree of physiological modularity correlate with negative senescence in plants,

171 although not necessarily in animals. These findings therefore provides novel empirical

172 support for a possible explanation for escaping senescence.

173

174 **2 | A COMPARATIVE OVERVIEW OF FINCH'S HYPOTHESIS**

175 We extend Finch's hypothesis by linking its predictions to the *pace* and the

176 *shape* of senescence. The distinction between the pace and shape of senescence

177 emerged long ago (Keyfitz 1977) as a useful framework in ageing research that

178 decouples important metrics of ageing (Keyfitz 1977; Baudisch 2011; Baudisch et al.

179 2013, Wrycza and Baudisch 2014). The pace of senescence represents the

180 characteristic mean life expectancy (η_e) of an organism. For example, the lifespan of

181 moss campion *(Silene acaulis*; routinely >50 yrs, some specimens >300 yrs; Morris

182 and Doak 1998) is greater than that of the invasive cheatgrass (*Bromus tectorum*; <1

183 yr; Compagnoni and Adler 2014). On the other hand, the shape of senescence

184 describes *when* and how intensively mortality hazards change during the *normalised*

185 lifetime (typically over η_e; Keyfitz 1977) of a species (Fig. 2). Because shape is

186   normalised (*i.e.,* age-value divided by mean life expectancy), it allows one to compare

187   the rate of senescence across many species, regardless of differences in η_e across

188   populations and species.

189   Following Finch's hypothesis, we expect greater functional modularity to

190   negatively influence the pace, and, to a more limited degree, the shape of senescence.

191   Modularity should affect the pace of senescence through the individual's ability to

192   minimise damage by shedding damaged modules (Kozlowski 1973; Denley and

193   Metaxas 2016). Examples of modules that could be shed include specific ramets (in

194   clonal organisms), wood vessels, cnidarian polyps, bryozoan polymorphs, and

195   ascidian zooids. In general, we do not expect the shape of senescence of a given

196   species to be influenced by modularity. This is because neither theory nor evidence

197   suggest that localised injury is linked to age-specific mortality any more or any less

198   than other mortality factors. However, modularity might alter shape if the accrual of

199   mutations (*sensu* Medawar 1952) occurs on a modular basis and age-dependent

200   declines are restricted to the module. Because multiple prerequisites underlie this

201   proposition, we consider it a subsidiary hypothesis.

202   The scope of our review encompasses multiple kingdoms and a wide array of

203   taxonomic groups. By approaching the commonalities of modularity broadly, the

204   structural appearance and functional role of modules is diverse across different

205   species. For example, plants can have deeply modularised anatomies where

206   physiological independence among structurally discrete components of an individual

207   is high (e.g., ramets of an aspen grove). In contrast, animals tend to be "unitary"

208   individuals, meaning that they have structural anatomies where physiological

209   integration is high across their body plan. In these organisms, "shedding" part of a

9

210 unitary animal (e.g., loss of an appendage or loss of a segment of the body) tends to

211 be, by definition, physiologically disruptive to the individual.

212   The potential for greatly different physical and functional qualities of modules is

213 unified by a basic characterisation: modules need not be inclusive of the full range of

214 physiological functions carried out by the individual such as water balance or energy

215 regulation. Instead, modules can be recognised as repeat sub-units within just one of

216 those physiological functions (e.g., immune activity, renal activity, respiratory activity).

217 This latter point suggests that we can divide our analysis into kingdoms, separating it

218 between unitary (animals) and non-unitary (some animals like corals and sponges,

219 and plants) organisms.

220   In general, traits that enhance modularity should correlate positively with the

221 pace of senescence (Silvertown, Franco, and Perez-Ishiwara 2001). In plants, we

222 expect increases in clonality, stem sectoriality (Schenk et al. 2008; Ewers et al. 2007),

223 and bark stripping (*i.e.*, the ability of trees to persevere when only a small part of the

224 trunk is surrounded by live cambium, Matthes et al. 2002) to prolong longevity. In

225 animals, we expect the number of polyps within a colony (Denley and Metaxas 2016)

226 and the number of functionally redundant organs to prolong longevity. The number of

227 discrete, redundant pathways for blood or lymph circulation (Seiler 2010; Huang,

228 Lavine, and Randolph 2017 respectively), and the number of parallel endocrine

229 pathways should do the same. We expect that all of these traits should correlate

230 negatively with mortality rates, and therefore increase the pace of senescence (Fig.

231 1a, b & c).

232   On the other hand, the link between modularity traits and the rate of mortality

233 over age (*i.e.*, the shape of senescence) is subtler. In plants, several traits could

234 change age-specific mortality rates, including clonality, epicormic branching in trees,

235 and indeterminate growth. On the other hand, in animals, fewer traits would be

236 expected to change age-specific mortality rates, because growth tends to be definite.

237 A notable exception to this is metazoans (Bodnar 2009). The indirect effect of age-

238 related size-dependent benefits can potentially tap alternative mechanisms for

239 escaping senescence detailed by Vaupel et al. (2004).

240 Modularity is expected to decrease the pace of senescence, with possible

241 indirect effects on the shape of senescence. As discussed above, modularity may

242 indirectly affect the shape of senescence as a result of facilitating the attainment of

243 advanced ages, and size-dependent benefits. More generally, modularity could have

244 a more complex effect on the shape of senescence if localised injuries affect age-

245 specific mortality. Some mortality factors are internally mediated and dependent on

246 lifespan-normalized age; others are external and independent of age. As a result, if

247 modularity facilitates the attainment of more advanced ages, we would expect that the

248 pertinent mortality factors will change. These changes may alter when in the lifespan

249 of an individual mortality is most likely to occur, and thus change the shape of

250 senescence.

251 This relationship is captured in the generalised age-dependent/degenerative

252 term under the Reliability Theory of Senescence (Gavrilov and Gavrilova 2001).

253 Where environmental mortality factors influence mortality rate irrespective of

254 physiological declines, they are mapped as age-independent mortality factors. Under

255 Reliability Theory, modularity will influence the realisation of a system failure (which

256 we define at the scale of the individual), as it constitutes a system component with

257 functional redundancy that, in turn, acts as a redundancy parameter, reducing *failure*

258 *events:system failure* (*i.e.*, local injury:individual mortality).

259

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, and Michler 2012). Epicormic branching has been linked to longevity in long-lived trees, such as the Douglas fir (*Pseudotsuga menziesii*; Ishii et al. 2007). Epicormic branches of the Douglas fir allow old, large trees to maintain foliage (Ishii et al. 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 et al. 2009; Mock et al. 2008) or a Neptune grass clone (*Posidonia oceanica*) found in the Mediterranean Sea (Guerrero-Meseguer, Sanz-Lázaro and Marín 2018).

Demographic effects of physiological injuries also appear to be informed, at least in part, by the structural organisation 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 and Sperry 1989; Zimmerman 1983). Variation in the morphology that influences embolic risk is expected to have a high demographic impact.

285   The interaction of emboli with the morphology of plant vascular and vessel

286   morphometry has been the focus of much research (Sperry et al. 2007; Venturas,

287   Sperry, and Hacke 2017; Pitterman et al. 2010; Tyree and Sperry 1989; Ewers 1985;

288   Bailey 1916). The well-established "rare pit hypothesis" of trade-offs between

289   efficiency and safety of vessels stems from this line of questions (Christman, Sperry,

290   and Adler 2009; Christman, Sperry, and Smith 2012). Of direct importance to

291   senescence, the safety-efficiency vessel trade-off offers a case example of direct

292   importance to senescence. When an embolism forms in a vessel, the vascular

293   configuration of the plant determines whether and to what extent the initial embolic

294   disruption spreads to the whole organism (*i.e.* runaway embolism *sensu* Sperry and

295   Pockman 1993). More sectorial vasculatures can mitigate the risk of runaway

296   embolism by reducing the lateral connections between vessel bundles. If emboli result

297   in runaway embolisms, they would be contained to a few bundles (Zanne et al. 2014;

298   Orians, Babst, and Zanne 2005).

299   A series of studies have explored the environmental correlates of this type of

300   sectoriality, and have found higher sectoriality in habitats with higher embolic risk,

301   notably deserts (Maherali, Pockman, and Jackson 2004; Wright et al. 2004; Ewers et

302   al. 2007; Royer et al. 2005; Ehleringer 1980; Carlquist 1975; see, too, related

303   questions addressed by Adler, Sperry, Pockman 1996). Indeed, Schenk and

304   collaborators (2008) showed that sectoriality increases on either hemisphere towards

305   the latitudinal belt of arid zones across 12 woody species.

306   To test the effect of modularity on senescence in plants, we quantified the

307   correlations between measures of the pace and shape of senescence and proxies to

308   plant modularity. We quantified pace and shape of senescence using the COMPADRE

309   Plant Matrix Database (Salguero-Gómez et al. 2015) and used methods described in

310   Salguero-Gómez (2016) and Jones et al. (2014). We implemented a method described

311   in Baudisch et al. (2013) to compare inter-specifically various moments of senescence

312   across species. This approach isolates measures of lifespan from lifespan-

313   standardised metrics of survivorship, and reduces highly variable mortality patterns to

314   select statistic features whose discrete markers reflect key variations in population

315   structure.

316   We derived, from the resulting 138 plant species in COMPADRE, three metrics

317   of the pace of life, which are used as proxies for measuring the pace of senescence.

318   These metrics consist of mean life expectancy (η_e), maximum longevity (η_{max}), and

319   generation time (T) using methods developed by Caswell (2011). We used a single

320   metric to characterise the shape of senescence: Keyfitz' entropy (H) (Keyfitz 1977;

321   Baudisch 2011; Wrycza, Missov, and Baudisch 2015), which describes the overall

322   shape of the survivorship curve (Fig. 2), such that when H>1, mortality –and thus the

323   rate of actuarial senescence– decreases with age. Thus, under this metric, the decay

324   in survivorship is quantified irrespective of reproduction. An estimate of the pace of

325   senescence for a species can be calculated from an adult survivorship class. Relying

326   upon a single adult stage-class for survivorship estimation, however, implies that there

327   is no shape of senescence for the given species, which in select cases may not be the

328   case. The influence of this assumption as results will not be known until a later time,

329   when more age/stage-specific survivorship estimates are available.

330   The metric H, as well as those that quantify the pace of life (e.g., η_e, η_{max}, T),

331   rely on age-structured demographic information. The underlying demographic

332   information is often captured in a stage-structured format (as matrix population

333   models). To derive age-structure from stage-structured models, we used methods

334   from Jones et al. 2014 (based on mathematical proofs by Caswell 2001). These age-

335   from-stage extraction methods use numerical simulations of the stage-structured

336   matrix to determine a quasi-stable-state of a population distribution for species that

337   have multiple adult age/stage-class survivorship estimates. This, in turn, yields a

338   decaying survivorship l_x curve from which our desired summary metrics can be

339   extracted in an age-structured form. When simulation models converged to a stable

340   stationary population distribution, they were not included in our data. This method and

341   convergence selection criterion were also used on the data for animals (below).

342   To test whether functional correlates of physiological modularity inform the pace

343   and shape of senescence, we drew on several open source databases. We collected

344   functional trait data related to the degree of modularity from the FRED (Iversen et al.

345   2017), BIEN (Enquist et al. 2009), and TRY databases (Kattge et al. 2011), as shown

346   in Table 1. These global trait databases are curated repositories of plant traits

347   collected from a wide range of field studies, laboratory experiments, and observations.

348   Here, we consider only those quantified under unmanipulated conditions. When

349   multiple functional trait values were available per species, we computed the mean of

350   each species-specific trait value. This was a straightforward mean, which we

351   performed after making sure that identical values of a trait (e.g. root diameter of 0.4996

352   mm) were not doubles. To do so, we examined several instances of double values in

353   each one of the three databases, and contacted database curators for feedback on

354   the most suspicious cases. We present only results of our analyses where >20 species

355   were present in COMPADRE and in the functional trait databases (Table S2).

356   Finally, we tested for correlations between species-specific demographic traits

357   and their modularity traits using a battery of phylogenetic generalised least squares

358   (pgls) using the packages ape (Paradis, Claude, Strimmer 2004) and caper (Orme

359   2013) in R (R Core Team 2019). We used the phylogeny of vascular plants by Zanne

360 et al. (2014). Family-wise errors were corrected using a Bonferroni correction. We

361 used Akaike Information Criterion (AIC) scores for model selection among a set of

362 competing models with linear and quadratic predictors. Linear models consistently had

363 the lowest AIC and were therefore retained.

364

365 **2.2 | Modularity and senescence in animals**

366   The literature on senescence has historically been more inclusive of unitary

367 animals than it has been for other groups. Important examples that have advanced

368 our understanding of animal senescence include studies on roe deer (*Capreolus*

369 *capreolus*; Gaillard et al. 1993, Loison et al. 1999); Soay sheep (*Ovis aries*; Catchpole,

370 Morgan, and Coulson 2000); Dall sheep (*Ovis dalli*; Murie 1944; Deevey 1947; Kurtén

371 1953); alpine marmot (*Marmota marmota*; Berger et al. 2016); and the short-tailed vole

372 (*Microtus agrestis*; Leslie and Ranson 1940; Leslie et al. 1955). However, to date,

373 other lineages across the tree of life have not received much attention.

374   Research in senescence has only gained momentum in the Plantae Kingdom

375 in the last two decades (Salguero-Gómez, Shefferson, and Hutchings 2013; Thomas

376 2013; Munné-Bosch 2008; Vaupel 2004; Thomas 2002) and it remains even less

377 represented among modular animals (but see Bythell, Brown, and Kirkwood 2018 and

378 Tanner 2001). Within plants, researchers have explored the question as to whether

379 ramets senesce faster than genets (Salguero-Gómez 2017; Orive 1995). A similar

380 angle on the animal kingdom remains untapped. This may be attributable to the close

381 relationship of modularity to meristem-based growth, plant vasculature, and other

382 functional characteristics restricted to the Plantae Kingdom (Vuorisalo and Mutikainen

383 1999; Clarke 2011). Broader application of these scaling relationships may also be

384 hindered by the conflation of modularity with other coexistent functional units. Modules

385 often coincide with carbon sectors, structural axes, independent physiological units,

386 and inflorescences, among other such structures.

387 Here, we provide a review of animal modularity and its relationship to longevity

388 and senescence. Our goal is to offer a translation of the primarily plant-based term

389 modularity to individual animals for which the integration of resources is high and adult

390 size tends to be determined. In doing so, we take a discrete step beyond previous

391 treatments on the subject (notably Ewers et al. 2007; Vuorisalo and Mutikainen 1999;

392 Harper 1980), in that we evaluate specific hierarchies of organ systems across

393 demographic metrics.

394 At first sight, the term modularity would not be applicable to most animals if

395 defined as repeat sub-units of multi-cellular tissue (Chapman 1981; Vuorisalo and

396 Tuomi 1986; Finch 1990). Notable exceptions include corals and sponges, or, using a

397 colony perspective, social animals such as ants (Kramer et al. 2015). Overall,

398 however, the consideration of the Kingdom Animalia through the lens of classical

399 modularity has to date received little attention. There is a corresponding lack of

400 precision in describing the intertaxonomic concepts pertaining to modularity across

401 kingdoms. The development of a framework that cuts across kingdoms allows us to

402 test urgently needed mechanistic theories across the tree of life (Baudisch & Vaupel

403 2012).

404 A notable exception in this direction is the work by Esteve-Altava (2017), who

405 recently interrogated a number of similarities in modular concepts. This research

406 focused on structural, developmental, functional, and topological integration of

407 modules in plants and animals, thus touching upon how we characterise modularity in

408 this paper. However, in stressing the conceptual cross-over between plants and

409 animals, Esteve-Altava highlights developmental origins, rather than the nexus of

410   modularity with demography. The relationship between modularity and longevity found

411   in plants (Fig. 3 a & b) raises the novel question of whether the degree of structural

412   redundancy in animals is relevant to their survival. Specifically, it raises the question

413   of whether its effect on senescence is comparable to that of modules in plants. We

414   tackle this question with a review of animal tissues and a criteria-based approach to

415   qualifying modularity from a histological and functional angle.

416   The classical model of modularity (White 1979) describes modules along lines

417   of discrete units of growth, units of reproductive independence (Harper and Bell 1979),

418   independent functional units (Sprugel, Hinckley, and Schaap 1991), or independent

419   physiological units (Watson 1986; Watson and Casper 1986). This principle works well

420   to describe plants and colonial invertebrates, since these are comprised of well-

421   defined, repeat structural sub-units. In other ways, it is deficient and unduly restrictive.

422   Modularity is a physiologically defined term whose touchstone is partial

423   independence of different components of an individual. In most unitary animals,

424   modularity does not appear to have ready application, as physiological functions are

425   deeply integrated. In these organisms, the furthest extremities are physiologically

426   linked to the central corpus of the individual. In this way, they are nearly opposite the

427   concept of comprising semi-independent units. There is a critical disconnect, however,

428   regarding this perception. Modules are traditionally thought of as "individuoids" or

429   "complete physiological units," compartments which package the full suite of

430   physiological functions of an individual. But this does not need to be the case.

431   We can conceive of redundant organs in a subsystem, which might offer

432   independence in function. So long as injury can be localised to a physiological system,

433   one can envision a benefit conferred by redundancies. Unitisation can be within a

434   single sub-system 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 sub-colony 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 recognised 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 (Tuomi and Mutikainen 1999; Harper 1980). Indeed, the axioms of modularity are multicellularity and multiple *redundant* units (Tuomi and 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

460   functionally related such that the impairment of one would not be indispensible to the

461   individual's survival?

462   We carried out an exhaustive survey classifying redundant components in 151

463   animal species. For the two systems – lymphatic and renal– where there appear to be

464   conditions that lend themselves to functional redundancy, we investigate the types

465   and scale of repeated elements in the system and how those are distributed among

466   major groups of organisms. Table S3 of the online supplemental material contains a

467   summary review of other organ systems. To test the effect of organ multiplicity on

468   senescence, we used methods similar to those employed in plants (above). This

469   involves running a series of phylogenetic generalised least square models to quantify

470   the relationship between indices of organ multiples and mean life expectancy (η_e;

471   Table 1), maximum life expectancy (η_{max}), Generation time (T), and Keyfitz' entropy

472   (H). H was analysed for only those species for which survivorship was estimated for

473   at least two adult stage-classes.

474   **2.2.1 | Immune system hierarchies**

475   The lymphatic system is a strong candidate for a modular structure that might

476   influence senescence partly because of the importance of immune integrity to

477   survivorship (Nussey et al. 2013). More specifically, in certain species, the lymphatic

478   system comprises numerous independent sub-units that are spatially discrete. Within

479   species that have lymph nodes, we speculate that modularity corresponds with the

480   number of lymph nodes in the body. Thus, species with a greater lymphatic modularity

481   should have lower rates of senescence, as an extension of Finch's thesis (1990).

482   Lymphatic systems of mammals span some twenty-fold variation in lymph node

483   number, from 22 in mice to 450 in humans (Haley 2016) and several thousands in

484   large ungulates (Nikles and Heath 1992). However, despite such a vast range of

485   interspecific variation, reliable estimates of lymph node numbers across a large range

486   of animal species do not exist. Indeed, for species lacking lymph nodes, other proxies

487   for the degree of modularity require more finessing. Organs known as lymph hearts

488   promote the movement of lymph through the lymph vascular network of certain

489   species. They are found *in lieu* of lymph nodes in reptiles (Hedrick et al. 2013),

490   amphibians (Hedrick et al. 2013), and some bird species (Hedrick et al. 2013; Budras,

491   Hullinger, and Rautenfeld 1987). These structures can range from several to >200 in

492   an individual (e.g. Caecilians, *Caecilia*; Kampmeier 1969).

493   Lymph heart function is not equivalent to that of lymph nodes. These organs

494   do, however, share qualities in that they both influence immune system integrity and

495   comprise discrete, highly numerated structures that mediate lymph. Independent of

496   the above structures, temporary or reactive lymphatic structures develop in certain

497   other species. These are variously termed aggregates, nodules, or patches, and

498   participate in immune function. As such, they play a role in preventing tissue damage,

499   disease, or dysfunction, and their continued functionality thereby reduces mortality.

500   One of the complications with the lymphatic system in regard to isolating injury and

501   compartmentalising damage is the functional integration of lymph nodes. Depending

502   on the configuration, topology, and dynamics of lymph flow, regionalised isolation of

503   injury may be infeasible.

504   **2.2.2. Renal hierarchies**

505   The kidney presents a good example of a non-conventional morphological unit

506   that would meet the discrete, functional unit criteria of modularity. Kidneys are

507   physically separate and individually dispensable. As reflected in humans, loss of a

508   kidney does not attend serious acute or long-term declines in the integrity of the renal

509 system (Foley and Ibraim 2010; Wee, Tegzes, and Donker 1990 and Kasiske et al.

510 2013).

511   Most vertebrates possess two bilaterally positioned kidneys (Yokota et al.

512 2008). Among species, there is wide variation in both function and morphology of

513 kidneys (Worthy and Williams 2002; Ortiz 2001; Beuchat 1996; Bentley 1971). In bears

514 and cetaceans, for example, the kidney contains numerous lobes (*reniculation*) which

515 exercise partial independence from one another (Maluf and Gassman 1998). The

516 lobes comprise a physically integrated organ, but sub-regionalisation within this organ

517 is high. This represents the highest degree of subdivision within our grading system

518 because the kidney structure can be functionally divided into more than one-hundred

519 compartments in some species (Ortiz 2001). Reniculated kidneys with high structural

520 and functional division may offer more potential for localised damage and tissue death

521 without functional injury to the renal system.

522   Kidney pathologies are diverse, but mostly internally mediated (Basile,

523 Anderson, and Sutton 2012). Common drivers of kidney stress, injury, and death are

524 high amino acid or nitrogenous waste loads, hypertension, and ischemia/hypoxia

525 (Palm and Nordquist 2011). External factors such as blunt force trauma or kidney

526 rupture more tightly akin to air embolism from the standpoint of acute, anatomically

527 focused injury, are less common. It is unclear how subdivision affects the resiliency of

528 renal physiology in response to different types of injury and stress.

529   While other species lack multi-compartmental renal organs, many species have

530 multiple organs that participate in the management of wastes and ion balance. In

531 marine fishes (Evans 2008, 2010), sharks (Shadwick, Farrell, and Brauner 2016), and

532 crabs (Towle and Weihrauch 2001; Weihrauch and O'Donnel 2015), gills play an

533 important role as an exchange site for ions between the organisms and their

534 environments. In fact, these can be critical for nitrogenous waste excretion (see Wilkie

535 2002 and Wright and Wood 2012 for reviews). Many species of bony fishes are long

536 lived, including dogfishes, paddlefishes, sturgeon, rock fishes, and eels (Patnaik,

537 Mahapatro, Jena 1994). Similarly, a number of shark species are atypically long-lived

538 (Hamady et al. 2014) and crustaceans, though particularly lacking in demographic

539 studies, are popularly cited for their long lifespan (Vogt 2012). Despite this, there is

540 little clade-level synthesis of senescence or negligible senescence for these groups

541 (Patnaik, Mahapatro, Jena 1994; Vogt 2012). Attending the lack of research on

542 senescence, links between specific anatomic similarities and longevity have not been

543 directly explored, let alone interrogated in regard to renal anatomy.

544   Depressed osmoregulatory capacity resulting from injury to either the kidneys

545 or the gills may be partially compensated for by having multiple osmoregulatory

546 organs. Secondary osmoregulatory organs might also enable a higher osmoregulatory

547 capacity and thereby tolerance for renal stress (Kültz 2015), which could both prevent

548 injury and facilitate recovery (Reimschuessel 2001) from sub-lethal renal injury. In

549 marine birds (Schmidt-Nielsen 1960) and some reptiles (Schmidt-Nielsen and Fange

550 1958), salt glands provide a mechanism to shed high salt concentrations. These

551 glands could similarly enhance redundancy of function (Gutiérrez 2014; but see

552 Babonis, Miller, and Evans 2011) without nitrogenous excretion. It is broadly

553 recognised that birds are unusually long-lived (Hickey et al. 2012; Péron et al. 2010;

554 Nussey et al. 2008; Monaghan et al. 2008; Ricklefs 2008), but research has

555 conspicuously overlooked the role of anatomy in their longevity, and instead focused

556 on cellular (Monaghan et al. 2008) and ecological explanations (Ricklefs 2008).

557 Reptiles include a number of the longest-lived species, such as tuatara which can live

558 some 90 years (Magalhaes and Costa 2009). Similar to birds, however, we see an

559 inclination toward metabolic and cell-based explanations and an absence of attention

560 given to organismal physiology and anatomy.

561   Along with gills, salt glands in accessory to kidneys constitute the second grade

562 of centralisation in in renal function in animals (Fig 3c & d). They are ranked below

563 reniculated kidneys because lobes of reniculation have the full suite of functional tools

564 for renal control, whereas gills and salt glands are more limited. Gills in certain species

565 of elasmobranchs (e.g. Wood, Pärt, and Wright 1995, Smith 1929) and salt-glands

566 carry functional weight only as it relates to ionic homeostasis, not nitrogenous wastes

567 control. Recorded variation tracks with environmental stresses, such as seawater

568 ingestion (Worthy and Williams 2002) and hibernation (Ortiz 2001), and corresponds

569 in part with acute and chronic kidney injury susceptibility (Stenvinkel et al. 2018).

570   We note that two-component systems, such as bilateral kidneys, raise special

571 questions under the framework we describe here. These cases elevate questions

572 about alternative explanations for the second organ that may not reflect an adaptation

573 for functional redundancy. There are two particularly important scenarios: alternative

574 selective pressures and non-adaptive tendencies.

575   As highlighted by Ewers and colleagues (2007), the function of two eyes is an

576 adept example of how non-redundant traits can drive redundant features. As such,

577 functional benefits unrelated to redundancy can confound the evolution of multiple

578 organs and their potential role in modularity as it relates to senescence. Separate ears

579 present another example of a highly specialised alternative benefit. More generally,

580 symmetrical bauplans may favor bilateral, paired structures. This may be particularly

581 likely where symmetrical division of gross anatomy shapes a developmental or

582 functional pattern in the organism. A candidate for this might be the bilateral lungs

24

583 found in many terrestrial vertebrates. This issue has not been the subject of focused

584 research.

585   Evolutionarily, the configuration of developmental control genes may influence

586 the development of accessory organs (Oakley 2007). There is currently a dearth of

587 research on the relative tendency for singular versus multiple organ duplication

588 resulting from genetic mutations. Regardless, the cost of having multiple organs is

589 likely to factor into whether organs are likely to fix into a population by genetic drift. If

590 we assume that costs are commensurate with organ number, lower numbers of organs

591 would have a higher likelihood of persisting in a population before being removed.

592 Therefore, one should attribute functional purpose to single-duplicate organs with a

593 degree of skepticism.

594

595 **3 | ANALYSIS OF MODULARITY IN PLANTS AND ANIMALS**

596   Our analysis supports Finch's hypothesis that modularity influences

597 senescence in plants, but support appears mixed across animals (Fig 3). We found

598 seven relationships between modular traits and measures of senescence. The

599 strongest relationships were found between between cumulative vessel redundancy

600 and the pace of senescence (e.g., cumulative vessel redundancy $\times$ reproductive

601 window (F=3.9; R^2=0.41; Fig 3b). Other relationships were weaker, but discernable,

602 such as that between vessel density and the shape of senescence (F=4.2; R^2=0.08;

603 Fig 3a). However, most of the relationships we tested within plants were not

604 statistically significant (Table S4).

605   In animals, there is evidence both supporting and refuting Finch's hypothesis.

606 Our tests support the hypothesis that modularity lengthens the pace of senescence in

607 animals (Fig 3; Table S5). Species with lymphatic organs with greater subdivision into

608 repeat components have higher maximum lifespans (95% C.I. [36.4, 73.4 years]) than

609 those with more centralised lymphatic anatomies (95% C.I. [19.0, 59.2 years]). On the

610 other hand, evidence refuting Finch's hypothesis was somewhat stronger. Lymphatic

611 anatomies with greater subdivision into repeat components presented lower mean life

612 expectancies than those with lower subdivision (95% C.I. [4.1, 12.1 years] *vs.* 95%

613 C.I. [7.9, 15.7 years]). When analysing renal anatomies, higher modular indices

614 correlated with lower maximum lifespans (95% C.I. [0.3, 13.6 years]) and we found no

615 correlation between modularity index and mean life expectancy. The renal modularity

616 index was negatively correlated with the shape of senescence (95% C.I [0.008,

617 0.084]). Other relationships were not statistically significant (Table S5).

618

619 **4 | DISCUSSION AND FUTURE DIRECTIONS**

620   In this study, we tested Finch's hypothesis (1990) that more modular organisms

621 should be more likely to live longer because of a decoupling of local injury from

622 individual mortality. We situated Finch's hypothesis in a broad, comparative framework

623 to look at a complete spectrum of degrees of physiological integration within the

624 context of the most recent research on senescence (Vaupel et al. 2004; Jones et al.

625 2014; Cohen 2018). To test our hypotheses in a fully comparative framework, we used

626 functional traits to explore plant senescence and propose measures of modularity in

627 unitary animals, a group of organisms thus far neglected in the comparative study of

628 modularity. We found support for Finch's hypothesis in plants, but mixed support in

629 animals.

630   Our results suggest that, in plants, modularity may in fact prevent or moderate

631 the potential for localised injuries to depress demographic rates (*i.e.,* reproduction and

632 survival). Modularity may decouple localised 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 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 modularised systems on the pace and shape of senescence may be limited because of (i) the types of mortality factors they interact with (Williams et al. 2006; Caswell 2007; Baudisch and Vaupel 2010); (ii) 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 Rickefs 2008); or (iii) because their preliminary classification of subdivision does not best represent functional divisions (trait-based approaches based on organ morphology are few).

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 lifecycle 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 similarity in demographic process. Extended lifespans that we report for modular plants adds to, and generalises from, a tradition of studies that relate clonal forms to longevity (Orive 1995; de Witte and Stöcklin 2010; Sköld

658   and Obst 2011). Modularity can be thought of, in limited part, as a downscaled,

659   localized, and weaker form of semi-clonality (White 1979). This trait enables larger,

660   composite organisms to access a new ecological and evolutionary space that is

661   inaccessible to less unitary plants (Mackie 1986). Today, the spectrum of modularity

662   adds a complex layer to the picture of demography, by grading a key distinction

663   regarding senescence between clonal and non-clonal species. Unlike clonality, which

664   lies at the high end of the physiological independence spectrum, many other modular

665   species tend to have an intermediate degree of physiological integration. Modular

666   species therefore present a valuable opportunity to explore senescence and the

667   mechanism which relieve it.

668   Moreover, the measures of modularity that correlate with measures of

669   senescence further support the adaptive value of hydraulic integration as suggested

670   by Schenk and colleagues (2008). Though promising, we cannot, with the data

671   available at present, attribute the general relationships in our results to the causality

672   of air embolism and related injury. Moreover, the potential adaptive aspects of

673   modularity may not speak to the origins of its evolution, or if it is incidental to another

674   adaptive feature in plants.

675   The correlations between stem density/cumulative vessel redundancy and

676   mean life expectancy (a measure of the pace of senescence; Fig. 2) suggest that

677   modularity decreases the average mortality rate. However, the correlation between

678   the same measures of modularity and a less strong shape of senescence (Keyfitz'

679   entropy; Keyfitz 1977) could reflect an alleviation of age-specific risk factors. Our initial

680   expectation was that modularity would not influence the shape of senescence, but for

681   a specific caveat related to how effects of age-specific deleterious genes might be

682   minimized by modularity (consistent with the three classical theories for senescence).

683   Our expectation implied that the effect of modularity would not change based

684   on the age of an organism. A correlation between modularity and the shape of

685   senescence suggests that modularity can affect age-specific mortality factors. Such

686   decrease in age-specific mortality rates could be a direct or indirect effect of

687   modularity. Our measures of modularity –including stem density and cumulative vessel

688   redundancy– might plausibly decrease age-specific mortality directly. This direct

689   decrease could occur because the risk of stem cavitation should increase with the

690   height, and therefore age, of individual plants (Koch et al. 2004). However, other

691   modularity traits could decrease age-specific mortality rates indirectly. For example,

692   epicormic branching should increase the capacity for long-term growth (Ishii et al.

693   2001) and therefore fertility (Ishii et al. 2007) of trees. In this case, modularity could

694   decrease age-specific mortality rates indirectly, by increasing size.

695   *Animals*

696   In animals, our analyses partly support the hypothesis that modularity should

697   decrease the pace of senescence in animals. In particular, we found that immune

698   anatomies with greater organ system subdivision have higher max lifespans than

699   those with higher centralisation. However, we found an opposite (though weaker) trend

700   when looking at mean lifespan.

701   The nexus of immune anatomy and immune function is an area that would

702   benefit from further study, particularly because of the role of localization and metastatic

703   processes. Moreover, there is a dearth of studies on the functional property differences

704   between lymph nodes and lymph hearts (Hendrick et al. 2013). In particular, there is

705   little available information on interspecific lymph node variation. But despite having

706   effectively no information on lymph node variation, the category has a narrow

707   distribution of rates of senescence (*H)* and mean life expectancies (η_e). *H-values*

708   reflect that not only most organisms exhibited similarly lengthed lifespans, but that

709   mortality patterns were similar among species. Lymph nodes are almost exclusive to

710   class Mammalia, for which the overall number of samples in the data base is not high

711   – and thus this may be partly an artifact of sample size. This topic remains of high

712   interest because of growing interest in immunosenescence and how lymphatics

713   intermediate immune integrity comparative physiology of lymph (Aw, Silva, and Palmer

714   2007).

715   In animals, the shape of senescence was not consistently predicted by our

716   index of modularity. One possible explanation for these results may reside in the way

717   we classified basal vertebrate and invertebrate systems as "centralised" or non-

718   modular. There is also latitude to interpret which sets and subsets of organs are

719   appropriate for comparative analysis, which could prove material to the results. Given

720   the extreme range of anatomies and distributed nature of available data on anatomical

721   biometrics, there is ample room, apt timing, and increasing value (Griffith et al. 2016;

722   Weiss and Ray 2019) in a more refined approach to defining functional and anatomical

723   trait groups for comparison.

724   An opposite trend between modularity and longevity in renal anatomy is

725   notable. In addition to caveats noted at the outset of this section, this result may be

726   explained partly by variation in the renal stresses experienced by the organisms

727   evaluated. For example, renal stress is greater for species which ingest high amounts

728   of salt and for those that exchange ions with hypertonic environments

729   (Hildebrandt 2001). Similar influences may be responsible for the unexpected pattern

730   we identified for age-specific mortality differences in renal anatomy.

731   In both plants and animals, there was a lack of detection of many significant

732   relationships between measures of modularity and measures of senescence. The

733   statistically significant relationships might reflect spurious correlations or true,

734   reproducible patterns. Prior knowledge suggests that the correlations we detected

735   likely reflect reproducible patterns, at least in the case of plants. We expect these

736   patterns to be reproducible because our significant correlations include measures of

737   modularity that are considered adaptive (Schenk et al. 2008). Future studies should

738   clarify the strength of the evidence here presented, especially because the sample

739   sizes of freely available demographic and functional trait data are increasing

740   exponentially with time (Enquist et al. 2009, Kattge et al. 2011, Salguero-Gómez et al.

741   2015, 2016, Iversen et al. 2017).

742   In species where reproduction is limited by actuarial senescence, we would

743   expect modularity to change the shape of reproductive senescence (Ricklefs 2008;

744   Lemaître and Gaillard 2017). Specifically, modularity that promotes, directly or

745   indirectly, the attainment of large organismal size should increase age-class

746   dependent reproduction (Stearns 1992). A relative increase in an organism's

747   maximum organismal size at first reproduction should do the same (Lacey 1986). We

748   do not analytically test this in our review due to data constraints. We note, however,

749   that contrary to our *a priori* expectation, the literature provides evidence of

750   reproductive senescence in clonal species (e.g. Ally et al. 2010) and actuarial

751   senescence in asexual species (Martinez and Levinton 1992).

752   We do not test reproductive senescence in this monograph because there are

753   more involved procedures required to ascertain changes in reproductive patterns.

754   These are not supported by the current data available, but may be in the near future.

755   Nevertheless, we predict that modularity could affect reproductive senescence if it

756   facilitates the attainment of large sizes, to which fertility is often proportional (Weiner

757   et al. 2009). In addition, at large sizes, the patterns of selection for fertility and mortality

758 can become decoupled from age (Caswell and Salguero-Gómez 2013; Roper,

759 Capdevila, and Salguero-Gómez 2019). Forthcoming work lays a conceptual

760 foundation for statistically decomposing fertility patterns similarly to how we approach

761 actuarial patterns here (Baudisch and Stott 2019). This approach uses pace and

762 shape metrics in a slightly different application, offering a promising next step to this

763 subject.

764   Our analyses of plant and animal data shows that greater degrees of modularity

765 correlate with the pace and shape of senescence, at least in plants. Recent

766 developments in research on hydraulic integration provide strong inference for why we

767 would expect to see the patterns we report. Despite this, one cannot eliminate the

768 possibility that correlated, yet to be identified benefits are responsible for driving this

769 pattern. Moreover, current mechanisms do not necessarily give insight to the

770 evolutionary origins of the trait, which will require addition study. These results offer a

771 foundation from which to work and enhance the urgency for this line of research.

772

773 ***Future directions***

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

775 research:

776   *Age-specific mortality factors influenced by modularity*: We have long

777 understood that modularity, at least in the form of clones, could relieve mortality forces

778 for the greater individual (Williams 1957; Cook 1979; Salguero-Gómez et al. 2013).

779 That less-physiologically independent modules could achieve a similar outcome and

780 play a role in determining the shape of senescence, is a more recent area of thought

781 (Vaupel et al. 2004). While we are left with more questions than answers in our results,

782 our finding that modularity may act on age-specific mortality rates suggests value in a

783 research agenda focusing on age-specific mortality factors as a fitness benefit of

784 modularity.

785   *Role of dysregulation and physiological modules in the endocrine system*: The

786 field would benefit from additional investigation into the role of dysregulation and

787 physiological modules in the endocrine system. A growing body of research describes

788 a close connection between endocrine dysregulation and ageing in humans (Li et al.

789 2015; Cohen et al. 2013; López-Otín et al. 2013). This work provides an important and

790 potentially widely-applicable mechanism for ageing, explaining patterns of

791 physiological senescence, based on the analysis of biomarkers. Dysregulation

792 research advances are at the forefront of understanding intermediating factors in

793 physiological declines. Theory in this area suggests that dysregulation does not

794 necessarily rely upon the three prevailing theories for the evolution of senescence

795 (Cohen et al. 2016). Rather, this phenomenon is likely linked to emergent properties

796 of complex systems. Understanding the role of this mechanism, its origination, and its

797 interactions with related theory –including the modularity inferface– will be a major

798 step in senescence research.

799   *Specific limitation on the fitness advantage of modularity*: Ageing biologists

800 would benefit from a research programme that probes the specific limitations on the

801 fitness advantage conferred by modularity. Welch and Waxman (2003) offer a strong

802 example of the type of work to be probed at this interface from an evolutionary angle.

803 We have found reduced mortality rates associated with highly modularised

804 architectures in plants. A now relevant question is whether there are costs and specific

805 factors which curtail such a benefit to plants and animals. Some candidates for inquiry

806 include environmental stresses that could be exacerbated by branched morphologies

807 (in climes where snow, wind, cold temperatures, or other physical stresses exert

808   routine injury), energetic costs of higher surface area (bark-to-vascular tissue),

809   vulnerability to disease, and light competition factors.

810   For decades, senescence was believed to be a universal phenomenon.

811   Evidence now exists that the ageing process is not inevitable in all species.

812   Mechanisms remain elusive, but this paper takes a modest step forward in testing

813   Finch's prediction of one mechanism based on the modular architecture of plants and

814   animals. Future research is needed to more deeply explore relevant modular traits and

815   their influence on specific mortality factors in plant and animal species.

816

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833

834 **AUTHORS' CONTRIBUTIONS**

835 RSG conceived the ideas and designed methodology with input from CB and AC. RSG

836 quantified life history traits. AC collated information on plant functional traits. CB

837 obtained indicators of modularity in animals. RSG and CB ran the phylogenetic

838 analyses. CB led the writing of the manuscript with input from AC and RSG.

839

840 **DATA ACCESSIBILITY**

841 Data used in this manuscript will be deposit open access in DRYAD when the work is

842 accepted. The demographic information is permanently, open-access archived in

843 www.compadre-db.org.

844

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1360

Table 1. Full results of linear models of functional traits relating to metrics of
1362 senescence and longevity in plants. Statistically significant relationships occur in bold.

Demographic measure	Plant functional trait	Slope	P-value	F Statistic
Keyfitz' Entropy (H)	Vessel number	-0.048	0.299	1.122
	Stem density	-0.594	**0.043**	4.225
	Root lignin	-0.005	0.515	0.438
	Root diameter	-0.307	0.616	0.256
	Vessel lumen area	5.123	0.249	1.393
Generation Time (T)	Vessel number	-0.121	0.415	0.0269
	Stem density	-1.247	0.207	0.570
	Root lignin	0.011	0.770	0.243
	Root diameter	0.620	0.795	0.404
	Vessel lumen area	9.067	0.522	0.189
Mean Life Expectancy (η_{e_s})	Vessel number	-0.164	0.086	3.186
	Stem density	0.615	0.550	0.360
	Root lignin	-0.013	0.649	0.213
	Root diameter	-0.833	0.716	0.135
	Vessel lumen area	19.341	**0.034**	5.028
Maximum Life Expectancy (η_{max})	Vessel number	-0.305	**0.041**	4.642
	Stem density	-0.674	0.456	0.559
	Root lignin	-0.045	0.170	2.016
	Root diameter	-0.802	0.691	0.161
	Vessel lumen area	32.165	**0.025**	5.669
Probability of Reaching Maturity ($pRep$)	Vessel number	-0.020	0.508	0.451
	Stem density	0.232	0.163	1.976
	Root lignin	0.000	0.971	0.001
	Root diameter	-0.238	0.490	0.488
	Vessel lumen area	0.579	0.847	0.038
Mean Age at Maturity (L_a)	Vessel number	-0.156	0.375	0.813
	Stem density	-0.211	0.845	0.039
	Root lignin	-0.004	0.909	0.013
	Root diameter	1.717	0.423	0.658
	Vessel lumen area	21.611	0.201	1.722
Reproductive Window ($L_{\alpha\omega}$)	Vessel number	-0.375	0.059	4.021
	Stem density	2.526	0.159	2.039
	Root lignin	0.014	0.791	0.073
	Root diameter	0.894	0.865	0.029
	Vessel lumen area	45.652	**0.022**	6.221

1363

1364 **Table 2**. Full results of linear models of indices of anatomic modularity relating to

1365 metrics of senescence and longevity in animals. Statistically significant relationships

1366 occur in bold.

Demographic measure	Animal functional trait	Slope	P-Value
Keyfitz' Entropy (H)	Renal Modularity Index	0.046	**0.019**
Keyfitz' Entropy (H)	Lymphatic Modularity Index	0. 001	0.967
Mean Life Expectancy (η_e)	Renal Modularity Index	1.636	0.274
Mean Life Expectancy (η_e)	Lymphatic Modularity Index	1.474	**<0.001**
Maximum Life Expectancy (η_{max})	Renal Modularity Index	6.706	0.061
Maximum Life Expectancy (η_{max})	Lymphatic Modularity Index	-6.595	**0.002**
Generation Time	Renal Modularity Index	2.343	0.304
Generation Time	Lymphatic Modularity Index	0.113	0.910

1367

1368

1369 Figure 1. A graphic representation of Finch's Hypothesis for actuarial (*i.e.,* mortality)

1370 senescence. These figures represent the hypothesised trait-senescence relationship

1371 in which modularity will lower the shape (a, b, c) and reduce the pace (d, e, f) of

1372 senescence, extending life span and reducing late-life mortality. The black lines

1373 labeled with ostrich icons represent a hypothetical unitary organism (with high

1374 physiological integration), and red lines labeled with coral icons represent a

1375 hypothetical modularised organism (with low physiological integration). Finch's

1376 hypothesis predicts that more modularised physiologies (the red lines) should result in

1377 reduced senescence (*i.e.,* longer lifespan and lower late-age/stage mortality) relative

1378 to their unitary counterparts (black lines). The survivorship curves are characterised

1379 by survivorship type (Type I: a, d; Type II: b, e; and Type III: c, f), reflecting standard

1380 groupings of mortality patterns, which is discussed further in Figure 2. Note that

1381 changes in the shape of a Type II curve obligates either a Type I (a, d) or III (c, f) curve,

1382 but pace can drive a less severe slope, which takes its place in this graphic. Also, note

1383 that the difference in shape and pace curves among Type 3 (c, f) curves are more

1384 subtle than for Type I (a, d) curves; the difference between panels are most noticible

1385 in how they intercept the x-axis. If the effect of modularity on senescence were strong

1386 enough, a species or lineage could be predicted to pass from one survivorship type to

1387 another – from Type I, to II, to III, respectively.

1388

1389 Figure 2. A graphic representation of Keyfitz' entropy (*H*). The shape of actuarial

1390 senescence can be measured across a wide range of dissimilar actuarial dynamics,

1391 using a standardised metric of *H*. Keyfitz' entropy *H* quantifies when mortality occurs

1392 along the lifetime of a cohort, from age at maturity (L_α), until –in this case– 95% of a

1393 cohort has died. This metric is dimensionless and normalised by the mean life

1394 expectancy of the organism, which allows for intra- and inter-specific comparisons,

1395 and is directly linked to the degree of modularity of different organisms (See Figure 3).

1396 Survivorship types describe different mortality patterns with age. In Figure 3, which

1397 highlights a period from maturity to 95% cohort mortality in red, a larger fraction of total

1398 mortality occurs in Type I and type II mortality than in Type III, as shown by the length

1399 of bars on the y-axis, which correspond by color to the survivorship lines. To a lesser

1400 extent, more mortality occurs in type I than type II over this same interval. The

1401 normalised metric represented by bar length (percent cohort change) indicates the

1402 mortality change over the *entire* interval; however, this measure of change can be

1403 measured at any age interval within this range. *H* is a metric of whether mortality

1404 through time tends to be weighted in early life, nearer to maturity, or late in life, nearer

1405 to 95% cohort mortality. Accordingly, Type I survivorship has the greatest *H*, with

1406 higher mortality change occurring late in the interval, near L_Ω, and Type III has the

1407 lowest *H*, with mortality change occurring late in life, near L_α.

1408

1409 <u>Figure 3</u>. Finch's hypothesis accurately predicts relationships between plant

1410 modularity as measured by functional traits and senescence, but fails to explain

1411 patterns in unitary animals between organ dividedness and senescence. Blue lines

1412 show statistically significant relationships between the shape of senescence (Keyfitz'

1413 entropy (*H*); see Figure 2 for more information on this metric) and (a) stem density,

1414 and (b) an index of renal modularisation. *H*=1, signified by the dashed black line,

1415 represents negligible or neutral senescence, and is the dividing line between positive

and negative senescence. Larger *H*-values, higher on the y-axis (and color-weighted by green), represent lower senescence; Smaller *H*-values, lower on the y-axis (and color-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 modularised 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 modularised, 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 lifespans in all groups except those with lymph nodes and no centralised immune anatomy. By reorienting the rank order of "no centralised immune system," a stronger relationship would emerge.