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A synthesis of senescence predictions for indeterminate growth, and support from multiple tests in wild lake trout

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Senescence—the deterioration of functionality with age—varies widely across taxa in pattern and rate. Insights into why and how this variation occurs are hindered by the predominance of laboratory-focused research on short-lived model species with determinate growth. We synthesize evolutionary theories of senescence, highlight key information gaps and clarify predictions for species with low mortality and variable degrees of indeterminate growth. Lake trout are an ideal species to evaluate predictions in the wild. We monitored individual males from two populations (1976–2017) longitudinally for changes in adult mortality (actuarial senescence) and body condition (proxy for energy balance). A cross-sectional approach (2017) compared young (ages 4–10 years) and old (18–37 years) adults for (i) phenotypic performance in body condition, and semen quality—which is related to fertility under sperm competition (reproductive senescence)—and (ii) relative telomere length (potential proxy for cellular senescence). Adult growth in these particular populations is constrained by a simplified foodweb, and our data support predictions of negligible senescence when maximum size is only slightly larger than maturation size. Negative senescence (aka reverse senescence) may occur in other lake trout populations where diet shifts allow maximum sizes to greatly exceed maturation size.

1. Introduction

Senescence is a decline in individual biological function with age, and is typically quantified as an increase in adult mortality rate or reduced 'fertility' [1], but can be applied to any decline in phenotypic performance. Tremendous variability exists among species in the shape (direction) and speed (rate) of senescence [2–5], and many authors seek to explain such patterns (e.g. [3,6,7]). The contention that the strength of selection declines with age is a common explanation of senescence [8]. The premise being that few individuals reach old age, and many have already reproduced when younger, therefore selection cannot remove problems that arise only at old age. A hypothesis that 'low adult death rates should be associated with low rates of senescence, and high adult death rates with high rates of senescence' [9], has empirical support. However, the nuances of the hypothesis and its predictions are debated [6,10,11]. Relative rates of adult to juvenile mortality appear to be critical [6], but asymmetry between parent and offspring [7] can differ widely between determinate and indeterminate growers making generalizations problematic. An example with bivalves provides a useful illustration [6, p. 527], which also applies to most fishes.

Our manuscript has three primary goals: (i) synthesize existing senescence theories, highlighting the importance of growth pattern, and emphasizing data needed

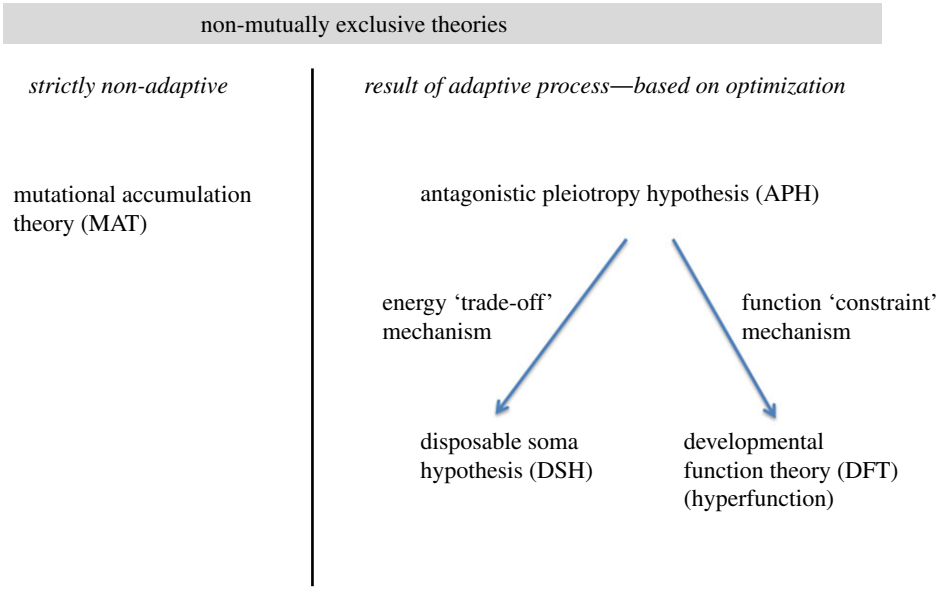

Figure 1. Our hierarchical conceptualization of the main evolutionary theories of senescence. Modified and expanded form Maklakov & Chapman [9]. (Online version in colour.)

to fill key voids, (ii) introduce lake trout (*Salvelinus namaycush*) as an ideal species to address senescence in the wild, and (iii) present a case study of two lake trout populations.

(a) Evolutionary theories of senescence

Attempts to explain senescence are challenged by inconsistencies in terminology and in the hierarchy of how theories are grouped. Complicating things further, the major theories of senescence [7] are not mutually exclusive, and create similar predictions but for different reasons. Our interpretation (figure 1) represents a modification from Maklakov & Chapman [8, fig. 2]. The mutational accumulation theory (figure 1), posits [12] that individuals senesce from the accumulation of deleterious mutations through their lifetime, such that senescence is strictly maladaptive. Other theories consider the notion of fitness optimization or life-history trade-offs, whereby declining performance with age may result from increased performance while young (figure 1); the antagonistic pleiotropy hypothesis (APH) [9] suggests senescence occurs when certain genes have positive effects in early life but negative effects later, and the disposable soma hypothesis (DSH) [13] proposes energy allocated in reproduction is unavailable to maintain the soma, resulting in deterioration. Many present APH and DSH as distinct, but we consider DSH to be a version of APH (figure 1). More recently, optimization of function has been proposed; appearing as developmental function theory (DFT) [8] and hyperfunction [14]. Conceptually, this is similar to DSH but the proposed mechanism varies, being energy allocation for DSH (a trade-off) versus DFT being a consequence of hyperfunctioning genes leading to excessive biosynthesis and molecular turnover in mature individuals, which (unlike [8]) we consider as a putative constraint (*sensu* [15])—as opposed to a plasticity enabled trade-off (figure 1). How DFT might apply to indeterminate growers is unclear, as development never stops.

(b) Atypical patterns of senescence

Most empirical work on senescence has been framed in support of DSH (e.g. [8,15,16]). However, recent work questions this [8,15,17], and new research that addresses some key gaps may be revealing. Examining unusual patterns of senescence [3,7] may help illuminate why and how it occurs [5] (figure 1). Negligible senescence describes species with little or no deterioration with age [2,18,19], while negative (reverse) senescence [20] may occur when biological function increases with age. The tenet of this argument is that in all species, mature individuals have offspring that are smaller than themselves. As offspring grow, their ability to reproduce increases and their probability of mortality can decline. In species with determinate growth, this pattern stops at maturity. Indeterminate growers however continue to increase in size after maturity. If mortality declines and fertility increases with size (age), then there is increased selection against senescence in indeterminate versus determinate growers.

Across different conditions, an optimization model [20] concluded that the intrinsic growth pattern (determinate versus indeterminate) influences the shape (direction) of senescence, while mortality determines its rate. Predictions can be summarized as: (i) senescent conditions (classical ageing) occur when the size at maturity is close to the maximum size (determinate growth) with little scope for increasing fertility with age (e.g. mammals, birds, insects); (ii) negative (reverse) senescence should occur when size at maturity is much less than maximum size (some indeterminate growers), and reproductive capacity increases with size; (iii) negligible senescence (little ageing) is an arbitrary middle ground along this continuum and should occur when size at maturity is somewhat less than maximum size, but reproductive capacity increases with size (age). Support for this framework appears in a recent review [7].

(c) Desirable study systems to fill key voids

Studies of senescence are heavily skewed towards a narrow range of conditions. A synthesis of the repeated calls (e.g. [16]) to address knowledge gaps includes:

(1) A critical need for research focusing on species with indeterminate growth [1,20–22] (e.g. in certain plants [23],

reptiles [24] and fishes [18,19]). Most work on senescence has considered determinate growers (mammals, birds, insects), which likely biases our view of ageing.

(2) The examination of senescence in wild populations [1,25,26], which better encapsulate natural processes and influences of potential environmental covariates on senescence not easily replicated in laboratory settings.

(3) Research that combines both longitudinal and cross-sectional comparisons of age (e.g. [27]). Comparisons in fitness-related traits can be made among age classes (cross-sectional) or by following individuals through time (longitudinal [23]). Because long-lived individuals may have inherent higher quality, their presence may bias cross-sectional comparisons, making longitudinal studies desirable [26,28]. However, longitudinal studies are subject to other confounding variables (e.g. directional environmental change), and it can take decades to track new metrics if following future cohorts. Thus, studies reporting consistent conclusions across combined approaches may provide more robust tests of hypotheses.

(4) Examinations of wild populations not subject to confounding variables [29], such as immigration/emigration (which may influence estimates of adult mortality), anthropogenic effects (e.g. recent changes in mortality adding novel selective pressure), or adult diet shifts with increasing body size, which can have dramatic effects on reproduction (e.g. gape limited carnivorous reptiles shift diet and are a problem, filter feeding bivalves are not).

(5) Research using recognized cellular indices associated with senescence [26,30,31], like relative telomere length [32] and the influence of reactive oxidative species and their potential for oxidative stress or cellular damage [29], particularly in wild ectotherms. Evolutionary literature on senescence ponders what happens (patterns), why it happens (or does not), but rarely addresses how it happens [8,28,30,33]. Laboratory and model organism-based studies on the biochemical mechanisms, or at least correlates associated with ageing and senescence, provide a framework to study senescence in the wild.

(6) Research focusing on reproductive senescence [24,26,34]. Most studies [26] of senescence quantify it as change in adult mortality rates (actuarial senescence), yet invoking mortality as an explanation is circular [26,35] being both a cause and consequence of senescence. Measures of reproductive senescence and other phenotypic traits are free of this problem.

(7) Senescence research that considers male individuals. Females have been the historical focus [33,34], but in most cases, males should senesce faster [8,33,36–40] thus offering larger effect sizes and greater power to answer key questions. This is especially true in species with intense sexual selection [37,41] as increased reproductive effort may come at a cost to tissue maintenance, and mortality can be higher in males due to conspicuous displays.

(i) The special problem of sperm senescence

Reproductive senescence includes senescence on the adult individual (e.g. ability to attract a mate), but additionally on gametes [30,42–44]. Gamete senescence affects the fitness of the individual, but also its mate and offspring [44,45]. However, separating effects among parents, gametes and offspring is difficult, especially in internal fertilizers. Egg senescence is rarely measured [34], but sperm senescence has gained interest [44,46]. Sperm senescence can be considered in two phases [43,46]: pre-meiotic (how the age of the male influences sperm) and post-meiotic (both before and after ejaculation). Sperm are particularly vulnerable to oxidative damage [30], and the male mutational bias [43] has led to interest in human fertility and paternal effects. Male fitness is a function of mating opportunities, sperm performance and offspring viability [34,45], which can be separated under experimental conditions (e.g. [47,48]). Older males generally produce sperm with reduced fertilization ability [27,29,34] and lead to higher rates of developmental abnormalities among offspring [29].

2. Lake trout

(a) Desirable attributes

Lake trout present an ideal indeterminate growth model for studies of senescence in nature, with low adult mortality being a key attribute. They inhabit the hypolimnion of lakes [49], where there are functionally no predators on adults (unlike anadromous salmonids), and spawn on lake shoals at night [49,50] where they are not exposed to terrestrial predators (unlike stream spawning salmonids).

Reproductive quality and effort can be accurately estimated from gametes. Lake trout do not typically migrate to spawn, show few secondary sexual characteristics, no sexual dimorphism, have no energetically costly courtship and provide no parental care [49,50]. Fertility increases with size (age). Males do not compete for territories [49,50], but post-ejaculatory sexual selection [45] occurs due to sperm competition [50]. Larger (older) fish generally produce more sperm, and thus would gain paternity advantages (fertility) under a fair raffle system [51].

Variation in maximum body size across populations (variable realization of indeterminate growth) within the same species may be useful for testing predictions of negligible and negative senescence [20]. Lake trout are among the largest members of the Salmonidae family, but maximum body size varies greatly as a function of prey availability [52,53] and populations vary in life-history traits that influence their fitness [54]. Inter-population comparisons of senescence could exploit environmental variation (*sensu* [15,16,26,28,33]) in variables such as growing season, prey resources and juvenile predators.

(b) Support for theories of ageing

If senescence is optimized (figure 1) between fitness benefits early in life at a cost to either hyperfunctioning genes (DFT) or somatic maintenance (DSH), then selection against a decline in performance with age is predicted to be relatively high in lake trout, as fitness potential increases dramatically with size (age), given adult predation rates decline while fertility increases. We are unaware of any published data that can shed specific light on DFT in lake trout. However, low allocation in reproduction is predicted to plastically trade-off with high allocation in somatic maintenance under DSH [55]. Possibly supporting this, lake trout have relatively low secondary sexual characteristics/migration/courtship/fecundity (resulting in low annual reproductive effort) and a predictably high incidence of iteroparity [49]. Perhaps

consequently, they can live to ages of more than 60 years [56], making them among the longest lived fishes, vertebrates and animals on the planet. Using a variety of approaches, we sought to directly test the hypothesis that wild lake trout show little or no senescence [20].

(c) Case study of two populations

Our study populations have additional attributes making them valuable for testing hypotheses of senescence in the wild. Many potentially confounding variables can be ruled out, as the lakes are located at the IISD Experimental Lakes Area (Ontario, Canada), where recreational fishing is prohibited and there is no unquantifiable directed anthropogenic activity. Mark–recapture studies have been ongoing since 1976, enabling long-term monitoring of individuals. The lakes are very small (see Methods) and all adults within a population experience similar environmental conditions. There are no piscivorous predators (except lake trout), adult trout are too large to be taken by loons (*Gavia immer*), but might occasionally be prey to otters (*Lontra canadensis*). Environmentally driven adult mortality is thus very low, whereas mortality of small juveniles is likely relatively high (*sensu* [6]). The lakes are connected to their surrounding watershed by very small streams, effectively eliminating immigration/emigration for this hypolimnetic species. Owing to a simplified foodweb [52,57] adult trout in these two lakes do not switch diet as they age, and gain little body size after maturity (figure 2*a*, and published growth curves [57]). This is critically important, as diet is known to affect gamete quality in fishes (e.g. [58]) and would bias age (size) comparisons otherwise. Sampling over the course of 40+ years has shown that young and old adult male lake trout co-occur on the spawning shoals at the same time (M.D.R. 2021, unpublished data), thus our age comparisons are not confounded by differential spawn timing.

3. Methods

In polyandrous mating systems such as that of lake trout, male 'fertility' is influenced by the ability to achieve fertilizations under sperm competition [34,45], a key component [46] being sperm swimming performance. We thus quantified male 'fertility' by measuring sperm traits that predict paternity. We also measured adult mortality estimates, body condition as a surrogate for general health [59,60], and relative telomere length as a cellular-level marker of senescence [61–63]. Our study thus combines actuarial senescence, phenotypic measures of bodily function with age (including reproductive senescence), along with a potential biochemical senescence marker, providing a more holistic approach others have highlighted as being needed (e.g. [8]).

(a) Longitudinal study

At first capture, fish were tagged, measured (total length, mass) and sexed, with the leading fin ray of a pectoral fin removed for ageing [64]. Recaptures used tag identification to assign age. Fish over the entire duration of monitoring in Lake 224 (27.3 ha, 1976–2017) were used, while from Lake 223 (26.4 ha) we restricted data to 1990–2017, to exclude the potential influence of an historical acidification experiment [57].

(i) Actuarial senescence

We estimated annual individual recapture and mortality probabilities using all adult males with known ages (Lake 223 = 385, Lake 224 = 422). To test for changes in adult mortality with age, we fitted a capture–mark–recapture model with a Bayesian framework (see electronic supplementary material, methods). Recapture and mortality probabilities were modelled as logistic regression functions of age, which was treated as a continuous variable.

(ii) Phenotypic performance senescence: body condition

Length-based body condition was estimated as a percentage of standard weight [65]. Fish that were recaptured at least twice during the autumn in adult life were used to determine if condition declined with age, and were analysed with a mixed effects modelling framework (electronic supplementary material, methods). Condition was evaluated as a function of fish age (fixed effect), and repeated measures on the same individuals (random slope), and the year sampled (random intercept).

(b) Cross-sectional study

(i) Fish collection

We collected fish on spawning shoals at night from 11 to 16 October 2017 and sampled the next morning following previous procedures [66]. Ages of recaptured fish were determined in the field by cross-referencing a database of tag IDs. Younger adult trout were more abundant than older individuals. To avoid potential confounding variables associated with date of sampling (e.g. weather, transport time to laboratory), we grouped fish as either being young (ages 4–10) or old (18–37), and processed them in a 'group design' (i.e. the same number of young and old fish were sampled each day). We analysed 15 groups in each lake (60 total; electronic supplementary material, methods).

(ii) Sample collection

Eggs were extruded from one female each day and separated from ovarian fluid [67], which was used in sperm swimming performance trials [68], to avoid neutral media when post-ejaculatory sexual selection occurs [29]. From each male, blood was taken from the caudal peduncle and semen was expressed by gentle abdominal massage. All samples were immediately immersed in ice, and transported to the laboratory for further processing (completed within 8 h of collection).

Aliquots of blood and semen were removed from ice and centrifuged (5000*g* at approx. 15°C for 5 min). Prior to freezing in liquid nitrogen, plasma was separated from blood cells. A separate semen aliquot was centrifuged in hematocrit tubes, and spermatocrit was computed [69]. This correlates with semen sperm density and often varies within individuals through a spawning season (e.g. [70]).

(iii) Sperm swimming performance

Details (electronic supplementary material, methods) closely followed Purchase & Rooke [67]. Four technical replicates of sperm activation were obtained for each fish. We were able to get useful data within 6 s of sperm/media mixing. Videos of swimming sperm were analysed in 0.5 s increments using open source software [71]. We used sperm curvilinear swimming velocity (VCL; $\mu m\ s^{-1}$) as a metric of male fertility, as it has been repeatedly shown to be correlated to paternity under sperm competition [72].

(iv) Relative telomere length

We measured relative telomere length from DNA recovered from red blood cells and sperm pellets using a qPCR-based approach that produces a telomere repeat (T) to single gene (S) copy number ratio (T/S). The assay was performed with two single copy genes, *orexin* (*Ox*) and *follicle stimulating hormone beta subunit* (*FSH*), to verify consistency of T/S ratios (electronic

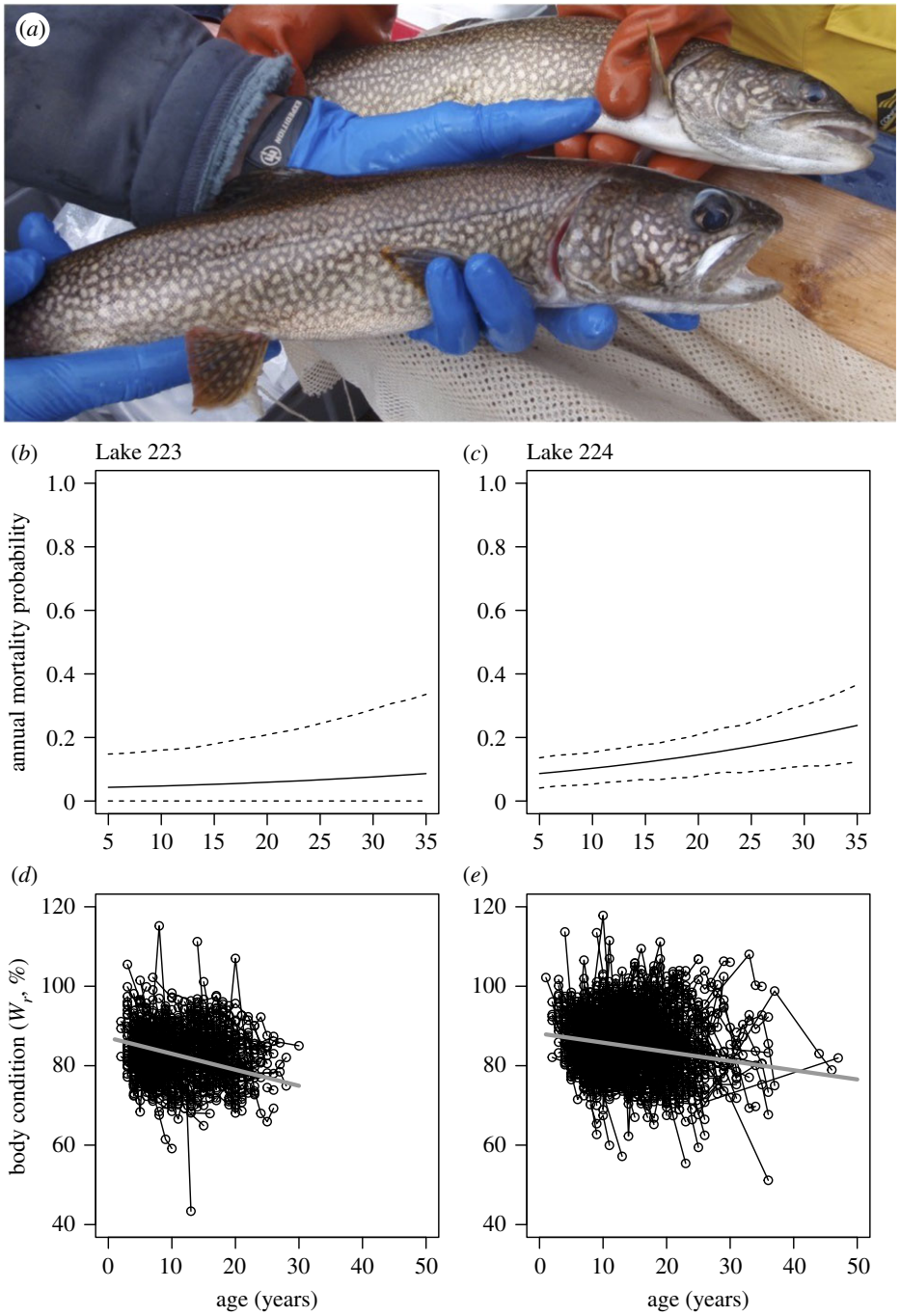

Figure 2. Longitudinal data from individually tagged adult male lake trout in Lake 223 (1990–2017) and Lake 224 (1976–2017): (*a*) adults of age 9 years (back) and 37 years (front) from Lake 224; (*b*) and (*c*) annual mortality probability as a function of continuous age, the solid line is the mean predicted probability, dashed lines are 95% credible intervals; (*d*) and (*e*) fall body condition relative to age for fish resampled at least twice, with predicted slope. (Online version in colour.)

supplementary material, methods). Both genes (*Ox* and *FSH*) garnered congruent relative T/S ratios (Pearson's correlation; blood: $r = 0.67$, $p < 0.0001$, sperm: $r = 0.72$, $p < 0.0001$), thus only the results of *Ox* are presented.

(v) Cross-sectional statistical analyses (2017)

Body condition, spermatocrit and relative telomere length were evaluated as a function of fish age (young versus old) crossed with lake. Sperm swimming declines rapidly after activation, with most fertilizations occurring within a few seconds. As such, we quantified sperm swimming velocity using two approaches. First we measured sperm at 6 s post-activation as a function of fish age (continuous variable: 4–37 years) crossed with lake, including tag ID (random intercept) to account for the four technical replicates per male. We also compared changes in sperm swimming velocity over time post-activation (continuous: 6–30 s) against age (young versus old). Lake, tag ID (random slope and intercept) and technical replicate (random slope and intercept) were included. In all cross-sectional analyses, the interaction between age and lake was not statistically significant ($p > 0.23$), indicating that the effect of age was similar in both populations. We removed these non-significant interactions prior to reporting final results.

4. Results

(a) Actuarial senescence

Annual mortality probability estimates of adult male lake trout were low (less than 0.20) across all ages in both lakes, and suggest a modest increase with age (figure 2*b*,*c*). This

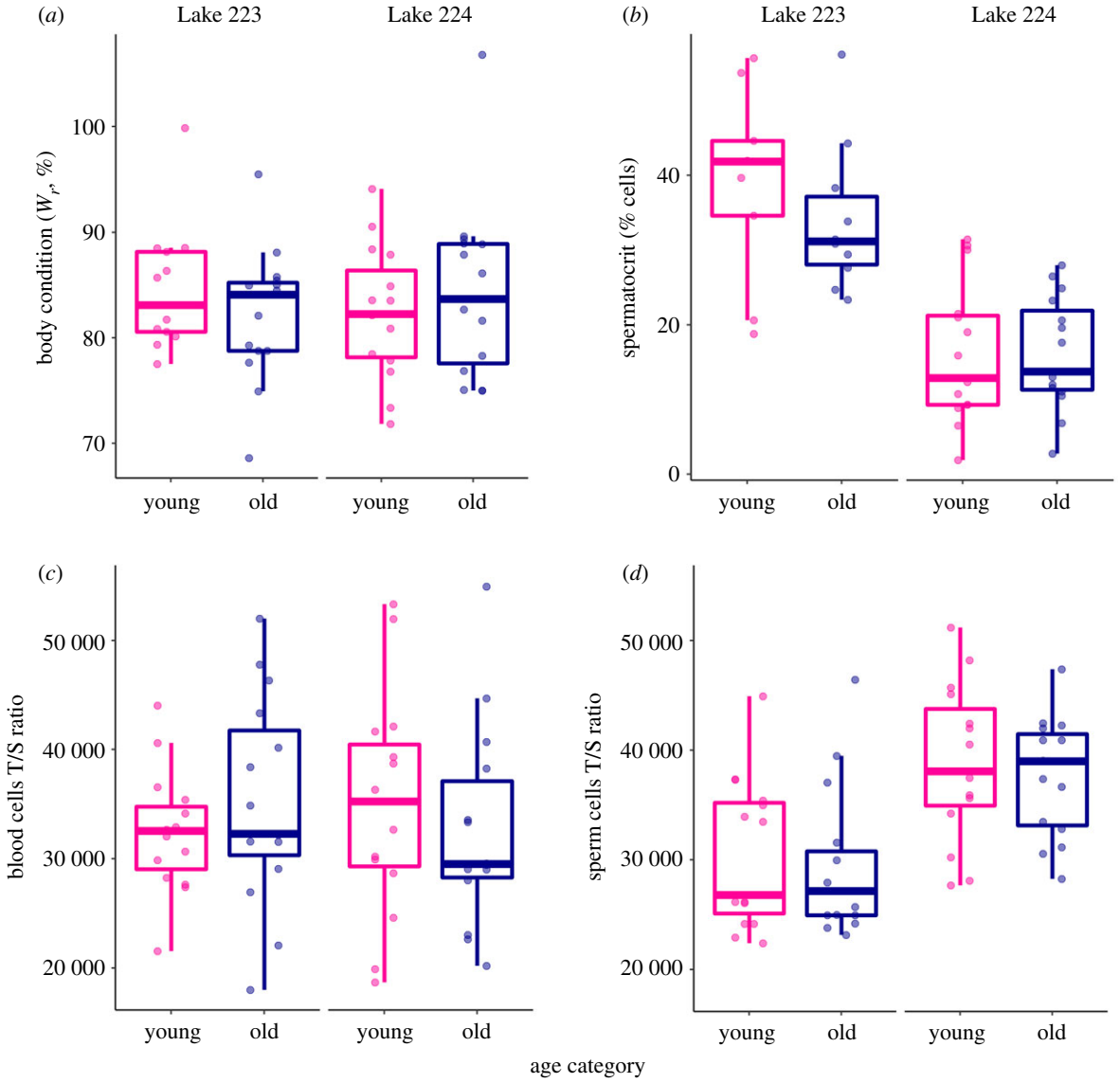

Figure 3. Phenotypic measures of young (pink: 4–10 years, $n = 30$) and old (blue: 18–37 years, $n = 30$) adult male lake trout from Lake 223 ($n = 30$) and Lake 224 ($n = 30$) in October 2017. Each point represents an individual trout. (*a*) Body condition, (*b*) spermatocrit, relative telomere length of (*c*) red blood cells, and (*d*) sperm cells. Telomere data presented using 0x reference gene, points represent average of three technical replicates per individual.

effect of age was clearer in Lake 224 compared to Lake 223 (99.8% and 80.5% of the posterior distributions of the slope parameter were positive, respectively). Doubling time of annual adult mortality is predicted to be 21 (Lake 224) to 30 (Lake 223) years.

(b) Phenotypic performance senescence

(i) Longitudinal condition

We observed a change in adult body condition with age in Lake 223 ($t_{52.7} = 125.2$, $p < 0.0001$; figure 2*d*; 446 fish, 1449 observations) and Lake 224 ($t_{216.2} = -2.6$, $p = 0.009$; figure 2*e*; 615 fish, 2888 observations). The rate of decline was negligible in Lake 224 at 2.3 units per decade, well within the variation among fish and years (most observations between 70–105 units), but slightly higher in Lake 223 (4 units per decade). Variation among random effects for both lakes is reported in electronic supplementary material.

(ii) Cross-sectional condition (2017)

Overall mean body condition was 83.4 ± 0.9%, similar to historical records (figure 2*d*,*e*). Body condition was similar in both lakes (Lake 224–Lake 223 means ± s.e.: 0.053 ± 1.79%, $t_{55} = 0.03$, $p = 0.976$) and there was no difference between young and old trout (old–young means ± s.e.: −0.146 ± 1.79%, $t_{55} = -0.082$, $p = 0.935$; figure 3*a*).

(iii) Cross-sectional semen quality (2017)

Although spermatocrit was higher in Lake 223 (Lake 224–Lake 223: −0.203 ± 0.028%, $t_{46.0} = -7.21$, $p < 0.0001$), there was no difference between young and old fish (old–young: −0.019 ± 0.028%, $t_{46.0} = -0.70$, $p = 0.49$; figure 3*b*). Swimming speed at 6 s post-activation was not different between lakes (Lake 224–Lake 223: −15.0 ± 8.1 µm s^{-1}, $t_{56.9} = -1.84$, $p = 0.071$), and was not related to fish age (0.71 ± 0.45 µm s^{-1} per year, $t_{56.9} = 1.58$, $p = 0.12$; figure 4*a*). The rate of decline in swimming velocity over time post-activation was faster in Lake 223 (rate difference, Lake 224–Lake 223: 0.97 ± 0.37 µm s^{-1} per second post-activation, $t_{57.0} = 2.65$, $p = 0.01$); however, there was no difference between young and old trout (rate difference, old–young: −0.43 ± 0.37 µm s^{-1} per second post-activation, $t_{57.0} = -1.18$, $p = 0.24$; figure 4*b*).

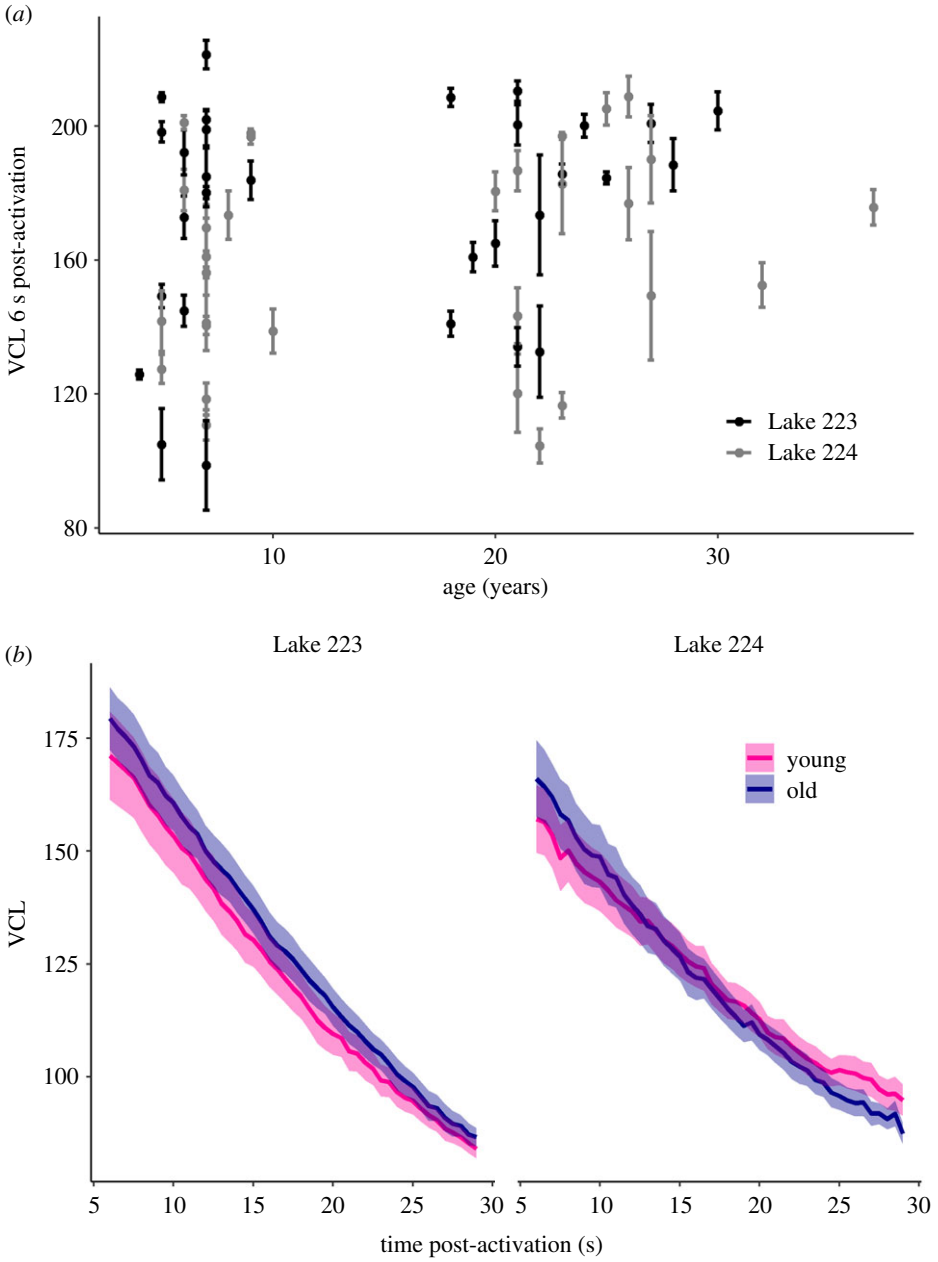

Figure 4. Sperm swimming velocity (μm s^{-1}) of lake trout in October 2017. (*a*) Velocity (VCL) at 6 s post-activation across age in years (black: Lake 223, $n = 30$; grey: Lake 224, $n = 30$), and (*b*) decline in sperm swimming velocity with time post-activation in young (pink: 4–10 years, $n = 30$) and old (blue: 18–37 years, $n = 30$) fish. Points represent average of technical replicates for each fish; lines represent average among individuals from the same lake and age category. Error bars/bands represent ± 1 s.e. (Online version in colour.)

(c) Biochemical proxy (2017)

Relative telomere length in red blood cells was similar in both lakes (Lake 224–Lake 223: 28 ± 2308, $t_{56} = 0.012$, $p = 0.99$), and in young and old individuals (old–young: 251 ± 2308, $t_{56.0} = 0.11$, $p = 0.914$; figure 3*c*). Relative telomere length in sperm cells was higher in Lake 224 (Lake 224–Lake 223: 8406 ± 1673, $t_{57} = 5.03$, $p < 0.0001$); however, there was no difference between young and old trout (old–young: −1196 ± 1673, $t_{57} = -0.72$, $p = 0.48$; figure 3*d*).

5. Discussion

As a species, lake trout have evolved under indeterminate growth, and all individuals have this genetic potential. However, realized growth in lake trout varies depending on diet availability, with fish achieving enormous sizes in some lakes, but being stunted in others. We exploited this scenario to make age comparisons among male trout that were not confounded by diet differences. Adult trout in our study lakes have realized growth that is functionally determinate, due to a simplified foodweb. Despite this, and as predicted by both DSH and DFT, negative effects of ageing were either undetectable or modest in our measured traits, and while we conclude that, as a whole, there is little senescence in these populations, we argue that there may be negative (reverse) senescence in other populations that are not growth constrained.

Although we have no molecular data to underpin endorsement of DFT, the DSH is clearly supported in our lake trout model. That low adult mortality (relative to juveniles) [6] should be associated with few negative effects of ageing [9,11] in indeterminate growers [20] paints an incomplete picture of how selection across generations interacts with life-history trade-offs in individuals. Life-history theory predicts that consistently low adult mortality *across generations* leads

to low annual reproductive effort as a means of bet hedging reproductive success across many episodes/years [73]. Under the DSH, through phenotypically plastic allocation of resources, this low reproductive effort would result in high somatic maintenance and thus low senescence *within a generation* (individual). Connecting these concepts for the case of lake trout suggests that due to (i) the lack of adult predation in the growing and spawning environments they evolved under, adult mortality is consistently very low across generations (it is the lowest of any salmonid), resulting in (ii) low reproductive effort in a given year (it is the lowest of any salmonid), and through plasticity within-individuals, (iii) high somatic maintenance results in limited senescence, enabling full potential of long life (it is the highest of any salmonid) to hedge reproductive success against environmental stochasticity.

Observed variation in senescent patterns among species [5,18,19] suggests contrasting selection pressures as an ultimate cause. Indeterminate growth is predicted [20] to increase selection against senescence when adult individuals experience reduced mortality and increased fertility with age (increasing size). In support, doubling time of adult male mortality in our lake trout (21 and 30 years in Lakes 224 and 223, respectively) was slow compared to long-lived mammals (e.g. 11 years for Asian elephants [74], 4 years for non-human primates and 8 years for humans [75]). Furthermore, a recent cross-sectional study on another long-lived freshwater fish [31] found improvements, not declines in functionality with age. Though confounding variables have made testing such predictions in wild populations challenging elsewhere, lake trout from our populations provide unique opportunities to control such issues, including diet. However, a stable diet probably results in old fish having inferior performance than their inherent potential. Negative senescence is predicted when size at maturity is much smaller than maximum size [20]. In lake trout populations where adults can switch to larger or more energy dense prey as they grow, old fish achieve much larger sizes than young adults [52]. Very large adults would have high sperm quantity (predicted to win under sperm competition = fertility), and high sperm quality (predicted to win under sperm competition = fertility) under suitable diets (and females would have much greater egg production). Such data would not only support negligible senescence, but also show the aptitude for negative (reverse) senescence in this species.

Our study found little evidence of senescence in growth-limited lake trout (cross-sectional data including fertility and condition showed no decline with age, longitudinal data showed modest declines in condition and increases in adult mortality). These data provide support of evolutionary theories of ageing, from rarely studied long-lived indeterminate growing animals in the wild. Our data are unique in that they coalesce information on (i) actuarial senescence using mortality rates from mark–recapture, with (ii) measures of phenotypic performance including reproductive senescence. Furthermore, blood and sperm cell telomere lengths did not decline with age, suggesting that (iii) telomere maintenance through adulthood may in part underpin maintenance. Our age comparisons combine longitudinal (same individuals across decades) with cross-sectional data (difference aged individuals at the same time), which is an infrequent approach. These assessments are strengthened by the unique characteristics of the study populations that control for confounding variables that are profuse in most natural situations.

If our conclusions are accurate, one can make predictions that should be supported by other data. We predict lake trout have (i) potential to exhibit negative (reverse) senescence in populations where adults can attain maximum sizes that are much larger than those at maturity, due to prey availability. In our populations, (ii) old and young males should show equal paternity if tested under sperm competition in the laboratory, (iii) pedigrees of wild populations should show equal average contributions of individual old and young males as fathers in a given year, and (iv) laboratory studies should indicate no increase in abnormalities in offspring development from old versus young fathers. (v) If DFT is involved, relative adult to juvenile proxies for cellular hyperfunction, should be correlated with senescence, within and among genera of salmonids in relation to the degree of iteroparity and semelparity, with lake trout at one extreme. (vi) Given the unusual insensitivity of relative telomere length to ageing in this species, further laboratory and field studies are needed to test if levels of other common molecular markers of senescence in this, and other long-lived ectotherms, may also fail to recapitulate the patterns expected from studies on endotherms or more typical laboratory model organisms. We encourage such studies to be undertaken where possible, along with comparisons across lake trout populations that vary in adult mortality and growth potential.

Ethics. All fish handling was conducted under an animal use protocol approved by the Lakehead University Animal Care Committee (file no. 1464656), and under the authority of annual scientific collection permits from the Ontario Ministry of Natural Resources and Forestry.

Data accessibility. The data are provided in electronic supplementary material [76].

Authors' contributions. C.F.P.: conceptualization, data curation, formal analysis, funding acquisition, investigation, methodology, project administration, resources, software, supervision, validation, visualization, writing—original draft, writing—review & editing; A.C.R.: data curation, formal analysis, methodology, visualization, writing—review & editing; M.J.G.: methodology, writing—review & editing; J.R.T.: conceptualization, data curation, funding acquisition, investigation, methodology, resources, supervision, writing—review & editing; E.A.M.: data curation, formal analysis, methodology, writing—review & editing; M.B.M.: data curation, formal analysis, funding acquisition, methodology, supervision, validation, writing—review & editing; M.D.R.: conceptualization, data curation, formal analysis, funding acquisition, investigation, methodology, project administration, resources, software, supervision, validation, visualization, writing—review & editing.

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

Competing interests. We declare we have no competing interests.

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