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Review

Senescence in natural populations of animals: Widespread evidence and its implications for bio-gerontology

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ABSTRACT

That senescence is rarely, if ever, observed in natural populations is an oft-quoted fallacy within bio-gerontology. We identify the roots of this fallacy in the otherwise seminal works of Medawar and Comfort, and explain that under antagonistic pleiotropy or disposable soma explanations for the evolution of senescence there is no reason why senescence cannot evolve to be manifest within the life expectancies of wild organisms. The recent emergence of long-term field studies presents irrefutable evidence that senescence is commonly detected in nature. We found such evidence in 175 different animal species from 340 separate studies. Although the bulk of this evidence comes from birds and mammals, we also found evidence for senescence in other vertebrates and insects. We describe how high-quality longitudinal field data allow us to test evolutionary explanations for differences in senescence between the sexes and among traits and individuals. Recent studies indicate that genes, prior environment and investment in growth and reproduction influence aging rates in the wild. We argue that – with the fallacy that wild animals do not senesce finally dead and buried – collaborations between bio-gerontologists and field biologists can begin to test the ecological generality of purportedly 'public' mechanisms regulating aging in laboratory models.

1. Introduction: Wild animals fail to senesce – a brief history of the fallacy

1.1. Medawar and the origins of the fallacy

The notion that animals in nature do not senesce – that environmental challenges whether they be predators, floods, famine or something else kill all wild animals before aging can take a measurable toll – can be traced at least as far back as Peter Medawar's first full theoretical treatment of the evolution of senescence (Medawar, 1952). This idea is clearly fallacious as we will show, undercut by both subsequent theoretical work and copious empirical data from a wide range of animals, yet it was crucial to the development of Medawar's central hypothesis about the genetic mechanism by which senescence could evolve.

Medawar's paradigm-shifting contribution to the evolutionary understanding of aging was his insight that due to the inevitability of death from environmentally driven causes, the ability of natural selection to favour or disfavour genetically-based traits depended on the age at which those traits appeared. As he phrased it, "the force of natural selection weakens with increasing age". This specific insight forms the basis of all subsequent analyses of the evolution of senescence. Based on this idea, Medawar proposed a particular genetic mechanism – that senescence evolves by the accumulation in the genome of harmful alleles, such as those predisposing to cancer, dementia, or heart disease, whose effects appear sufficiently late in life that "the force of natural selection will be too attenuated to oppose their establishment and spread." In other words, such deleterious alleles could spread only when the probability that they could have a measurable effect on reproductive success was very low. Consequently observable senescence should only occur at ages "which the great majority of the population do not reach" (all quotes from Medawar, 1952). Only under conditions in which animals are protected from natural hazards, he theorized, such that they commonly survive to ages they would very seldom or never achieve in the wild, would senescence be manifest. He was quite specific about this latter point, repeating it four times in the same monograph. To cite one of these,

> "Whether animals *can*, or cannot, reveal an innate deterioration is almost literally a domestic problem; the *fact* is that under the

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exactions of natural life they do not do so. They simply do not live that long."
– Medawar, 1952 (italics in original)

As evidence for this claim, Medawar cites a personal communication from field mammalogist Dennis Chitty, in which Chitty states that "wild mammals of any perceptible degree of senility" seldom turned up in his traps, and if they did he assumed that some cause other than senescence (e.g. infection, injury) was responsible for their condition (Medawar, 1952). For wild birds, Medawar's source was David Lack, the pioneering field ornithologist. Lack had noted in several papers in the 1940's, that the probability of death seemed to be independent of age in birds in nature, implying that senescence, at least in its actuarial sense, did not occur (Lack, 1943a,b). Medawar's claim was repeated in the first comprehensive consideration of senescence as a general biological phenomenon – Alex Comfort's classic tome, *The Biology of Senescence* (Comfort, 1956). Since then, the idea that senescence fails to occur in nature has been often repeated within bio-gerontology (e.g. Hayflick, 2000; Kirkwood and Austad, 2000; Rose, 1991).

In Medawar and Comfort's time there was in fact scant evidence for senescence in the wild because few detailed long-term studies of natural populations existed. Those that were available tended to be short-term, cross-sectional and often did not know the exact age of adults in the population. It has since become apparent that long-term studies that monitor marked individuals from birth to death are required to reliably detect and investigate senescence in the wild. In an earlier monograph on aging and death published in 1946, Medawar seems to acknowledge this, highlighting the importance of studies of exactly this kind:

"No-one has yet made a systematic study of whether even mammals *in their natural habitat* do indeed live long enough to reach a moderate though certifiable degree of senility. . . The difficulties of constructing life tables in the wild are technically formidable, but they must be solved."
– Medawar, 1946 (italics in original)

As we will demonstrate, despite Medawar and Comfort's subsequent assertions to the contrary, there was never any theoretical reason to adduce the absence of senescence in the wild (Williams, 1957), and there has been an avalanche of recent data from wild populations clearly demonstrating the senescence does indeed occur commonly in natural populations (Bennett and Owens, 2002; Brunet-Rossinni and Austad, 2006; Nussey et al., 2008a).

1.2. Unravelling the fallacy

Quite probably, Medawar's belief that animals in nature failed to senesce allowed him to dismiss one clear implication of his insight. Whilst he focused nearly exclusively on the accumulation of late-acting deleterious alleles, he mentioned in passing – even giving an example – that because of the gradual weakening strength of natural selection with age, "a relative small advantage conferred early [in life]. . .may outweigh a catastrophic disadvantage withheld until later" (Medawar, 1952). This, of course, is a succinct description of an idea George Williams later developed: antagonistic pleiotropy, in which alleles with beneficial effects on survival or reproduction early in life can be actively favoured by natural selection despite negative effects on health and fitness later in life (Williams, 1957). So although Medawar described antagonistic pleiotropy, he failed to appreciate its significance. In summarizing the implications of his theory at the end of his 1952 paper, he never mentioned it, focusing instead on the accumulation of late-acting alleles and on hypothetical modifier alleles that might postpone the effects of deleterious alleles to ages at which they would be effectively neutral (Medawar, 1952). However, if antagonistic pleiotropy is a common mechanism of senescence, then there is no necessary expectation that wild animals will fail to display progressive deterioration of health in later life or that such deterioration need be subtle. Williams' clearly appreciated this, referring to Comfort's argument that senescence was 'outside the developmental program that concerns natural selection', as follows:

"I believe that this theory is incorrect. Its fallacy lies in the confusion of the process of senescence with the state of senility, and in an inaccurate conception of the relationship of age to selection processes."
– Williams, 1957

By way of example, Williams noted that an examination of athletic records reveals 'rampant' senescence in humans as early as their 30's, a period which no-one could disagree humans commonly reached even in a state of nature (Williams, 1957). Williams highlighted two crucial points that alter the way we think about senescence in natural populations. First, in an evolutionary sense, senescence is the progressive physiological process of deterioration leading to a decline in fitness with age, which is not synonymous with infirmity and frailty associated with extreme old age in humans and captive animals. Classical evolutionary theory does not refer to or consider a state of senility in very late adulthood, rather it predicts that senescence should begin at the age of sexual maturity and progress from that point as the force of natural selection weakens (Hamilton, 1966; Williams, 1957). Second, under antagonistic pleiotropy, natural selection is expected to favour the evolution of life histories in which senescence has a detectable fitness cost within the natural life expectancies of organisms, as long as the genes associated with this deterioration confer a sufficient fitness benefit in earlier life.

Kirkwood's (1977) disposable soma theory of senescence is also compatible with the manifestation of senescence in the wild. Developed from a different theoretical framework – optimization theory as opposed to population genetics theory – the disposable soma theory makes largely similar predictions to antagonistic pleiotropy. Briefly, it hypothesizes that because critical resources such as energy are limited, natural selection will adjust the allocation of cellular and physiological resources between the fundamental processes of somatic maintenance and reproduction appropriately for an organism's ecological context (Kirkwood, 1977; Kirkwood and Rose, 1991). Since natural selection is expected to weaken with age, selection will tend to favour investment in early reproduction over long-term somatic maintenance, and senescence will result (Kirkwood and Rose, 1991). Antagonistic pleiotropy and disposable soma theories, often referred to together as 'life history' theories of aging (Partridge and Barton, 1996), now form the basis of the majority of theoretical work on the evolution of aging. This emerging body of evolutionary theory now clearly demonstrates that selection can favour senescence occurring within a species' natural life expectancy, and that the pattern of senescence can vary depending on the specifics of organism's life history, ecology and the interplay between "intrinsic" and "extrinsic" factors influencing mortality (e.g. Abrams and Ludwig, 1995; Baudisch, 2008; Cichon, 2001; Mangel, 2008; McNamara et al., 2009; Williams and Day, 2003).

Current empirical support from model laboratory organisms for disposable soma theory and antagonistic pleiotropy as mechanisms of senescence is dramatically stronger than for mutation accumulation. For instance, many single gene mutations known to extend life in model laboratory organisms have detrimental effects on early components of Darwinian fitness (Table 1). To pick one illuminating example, a partial loss of function mutation in *daf-2*, the *Caenorhabditis elegans* ortholog of the vertebrate insulin/IGF receptor, doubles the longevity of worms in the laboratory, yet its depressive effects on early reproduction (Chen et al., 2007) cause it to be quickly

Table 1
A sampling of antagonistic pleiotropy in long-lived genetically modified model organisms. Note that all genetic manipulations which lengthen life also decrease one or more measures of early life fitness. "*ox*-" prefix indicates over-expression of the gene.

Species/modification	Longevity increase	Pleiotropy effect	Reference
Caenorhabditis elegans			
daf-2	112%	Reduced reproductive rate	Jenkins et al. (2004)
clk-1	27%	Reduced reproductive rate	Chen et al. (2007)
age-1	50%	Reduced reproductive rate[a]	Walker et al. (2000)
Mit mutants (*atp-3*, *nuo-2*, *issp-1*, *cco-1*, *frh-1*)	60–70%	Reduced fecundity, *reduced* egg viability, delayed development	Rea et al. (2007)
Fruitfly (*Drosophila melanogaster*)			
chico	40–50%	Reduced fecundity/sterility	Clancy et al. (2001)
inR	48%	Reduced fertility/sterility	Tatar et al. (2001)
indy	90%	Reduced reproductive rate[b]	Marden et al. (2003)
ox-FOXO (fat body)	20–40%	Reduced reproductive rate	Giannakou et al. (2004)
Mouse (*Mus musculus*)			
Ames (prop1^{df})	35–70%	Reduced reproductive rate, delayed maturation	Bartke (2005)
Snell (pit1^{dw})	42%	Reduced reproductive rate, delayed maturation	
Little (GHRHR^{lit})	24%[c]	Reduced fertility, enhanced infectious disease susceptibility	Chubb (1987) Alt et al. (2003)
GHRKO	40–55%	Reduced fertility, delayed maturation	
ox-αMUPA	20%	Reduced reproductive rate, cognitive impairment	Miskin and Masos (1997)
ox-klotho	19–31%	Reduced fertility	Kurosu et al. (2005)

[a] Only under cyclical starvation (as experienced in the wild).
[b] Only under reduced food intake.
[c] Low fat diet only.

eliminated when directly competed again worms carrying a wild-type allele (Jenkins et al., 2004). If antagonistic pleiotropy and disposable soma are the main mechanisms responsible for the evolution of senescence and the maintenance of genetic variation in aging and lifespan, we should expect to observe senescence in the wild.

2. Senescence in wild animals – an evidentiary review

During the last two decades an avalanche of empirical evidence for senescence in wild animals has accumulated (Table S1, Fig. 1). However, at least one piece of published evidence for senescence in the form of increasing mortality with age already existed for wild mammals at the time of Medawar's 1952 monograph. More than 600 Dall sheep in Alaska had their ages at death estimated by annual growth rings on their horns, revealing a clear increase in mortality with age (Deevey, 1947). In birds, Lack's contention – cited by Medawar – that birds did not show actuarial senescence continued to be the conventional wisdom among ornithologists at least until 1974, when Botkin and Miller pointed out that if mortality were indeed independent of age in birds then a number of long-lived seabirds such as gulls or albatrosses should reach ages of 200–400 years, many times longer than appears realistic given what we know about the age distribution of such populations (Botkin and Miller, 1974). Furthermore, a visionary 18-year study of yellow-eyed penguins in New Zealand published in 1957 provided clear evidence for declines in some reproductive traits with age in a wild bird (Richdale, 1957). Over a decade later, compelling evidence emerged for both actuarial and reproductive senescence in a wild population of a much shorter lived species, the great tit (*Parus major*), based on long-term field data from a study that Lack himself started in the 1940s (Perrins, 1979). A decade or so later, several studies used life table information from wild birds and mammals to suggest that mortality rates increased with age (Finch et al., 1990; Nesse, 1988; Promislow, 1991), although the inferences that can be made about senescence from such vertical (or static) life tables are known to be limited (Gaillard et al., 1994; Jones et al., 2008). More recently, the maturation of numerous long-term, individual-based studies of wild vertebrates has provided unequivocal, longitudinal evidence for age-related declines in survival, reproduction and physiological function in birds and mammals (Brunet-Rossinni and Austad, 2006; Clutton-Brock and Sheldon, 2011; Jones et al., 2008) and, to a lesser degree, reptiles and insects (Bonduriansky and Brassil, 2002; Massot et al., 2011; Sherratt et al., 2010).

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To give some idea of the scope of current knowledge, we surveyed the literature for studies providing evidence of senescence in free-living animals, the results of which are presented in Fig. 1 (and listed in detail in Table S1). Using previous non-exhaustive reviews of birds (Bennett and Owens, 2002) and both birds and mammals (Brunet-Rossinni and Austad, 2006) as a basis, we included studies of free-living populations that documented age-related changes in adulthood consistent with senescent deterioration, noting whether the changes were in survival probability, reproductive performance traits (e.g. fecundity, offspring size, offspring survival), or other physiological or behavioural traits (e.g. foraging efficiency, immunity, body mass). Although no such survey will ever be exhaustive, we have made our best efforts to locate and include studies documenting age-related changes consistent with senescence in the wild. We have included only studies that analysed primary field data in our survey; comparative studies analysing previously published life tables were excluded. Whilst the vast majority of studies were on genuinely wild populations, we also included a handful of 'semi-natural' studies in which animals were kept in some form of protective enclosure but were still exposed to some forms of natural environmental stressors and mortality risk (e.g. Hruska et al., 2010; Zajitschek et al., 2009a, and other studies denoted with a "$" in Table S1).

We found 340 studies of 175 different animal species that provided evidence of senescence in the wild (Fig. 1A and B; Table S1). Of these, the vast majority were birds and mammals – 149 studies of 75 bird species and 165 studies of 79 mammal species. Table 2 spreads the evidence for senescence in the wild (Table S1) across the orders of birds and placental mammals. This serves to illustrate that the evidence is reasonably well taxonomically spread. Indeed, our survey of the literature suggests to us that where senescence has been looked for with detailed longitudinal data in wild birds and mammals it is usually found. However, Table 2 also clearly shows that there are many, many orders and genera for which data are

Table 2
Phylogenetic distribution of studies documenting evidence of senescence in wild populations of birds and placental mammals. Note that an absence of evidence for senescence within orders or genera most likely reflects ecological ignorance regarding the taxa in question, rather than evidence that senescence does not occur.

Birds				Placental mammals			
Order	Number of genera	Number of genera with evidence of senescence	Percentage of genera with evidence of senescence	Order	Number of genera	Number of genera with evidence of senescence	Percentage of genera with evidence of senescence
Struthioniformes	15	0	0	Edentata	15	0	0
Tinamiformes	9	0	0	Pholidota	1	0	0
Galliformes	84	3	4	Lagomorpha	12	0	0
Anseriformes	52	7	13	Rodentia	491	4	1
Podicipediformes	7	0	0	Macroscelidea	4	0	0
Phoenicopteriformes	3	1	33	Primates	74	11	15
Phaethontiformes	1	0	0	Scandentia	5	0	0
Pteroclidiformes	2	0	0	Chiroptera	209	1	0
Columbiformes	46	0	0	Dermoptera	2	0	0
Caprimulgiformes	22	0	0	Insectivora	73	2	3
Apodiformes	128	1	1	Carnivora	126	15	12
Opisthocomiformes	1	0	0	Artiodactyla	90	17	19
Gruiformes	42	0	0	Cetacea	40	4	10
Cuculiformes	32	0	0	Tubulidentata	1	0	0
Gaviiformes	1	0	0	Perissodactyla	6	1	17
Sphenisciformes	6	3	50	Hyracoidea	3	1	33
Procellariiformes	26	6	23	Sirenia	2	0	0
Ciconiiformes	6	0	0	Proboscidea	2	2	100
Pelecaniformes	35	2	6				
Charadriiformes	95	9	9				
Passeriformes	1290	20	2				
Psittaciformes	88	0	0				
Accipitriformes	74	4	5				
Coliiformes	2	0	0				
Strigiformes	29	2	7				
Trogoniformes	7	0	0				
Piciformes	71	0	0				
Coraciiformes	35	0	0				

lacking. It is very important to be aware that in Table 2 an absence of evidence for senescence reflects ecological ignorance regarding the taxa in question, rather than evidence that senescence does not occur. It is notable that two particularly speciose mammalian orders, the bats (Chiroptera) and rodents (Rodentia), which contain both the main mammalian laboratory model organisms for the study of aging (mice) and long-lived species of growing interest in bio-gerontology (bats and naked mole rats, Austad, 2010), are poorly represented in our survey of the literature. Gaps of this kind are, at least in some part, due to challenges studying particular kinds of animals in the field (e.g. subterranean, aquatic, highly motile and nocturnal species), and the fact research effort has been geographically focused in Europe and North America. However, this serves to highlight that we are very much swimming in the shallows when it comes to understanding the natural diversity of aging patterns across avian and mammalian taxa.

Although comparative studies testing evolutionary theories of senescence have tended to focus on age-related declines in survival probability (Promislow, 1991; Ricklefs, 1998, 2010, although see Jones et al., 2008), survival is only one component of an organism's lifetime reproductive success (Partridge and Barton, 1996). As Fig. 1C illustrates, the number of studies documenting age-related mortality rate increases in the wild is well-matched by evidence of declines in traits associated with reproductive fitness, such as fecundity, offspring size, offspring survival, sperm function and secondary sexual characteristics (Table S1). Studies documenting age-related changes in other physiological, behavioural and morphological traits consistent with senescence in the wild have begun to emerge since the late 1990s (Fig. 1C). This reflects a growing interest among evolutionary ecologists in the physiological processes underpinning age-related declines in survival and reproductive performance (Monaghan et al., 2008). There is mounting evidence for body mass declines in late life in wild ungulates (e.g. Mysterud et al., 2001; Nussey et al., 2011; Weladji et al., 2010), as well as evidence of muscular senescence in samples from adult wild-caught seals and shrews (Hindle et al., 2009a,b). Evidence for important changes in foraging behaviour with age – potentially due to loss of muscle function or wider physiological condition – is also beginning to emerge (e.g. Catry et al., 2006; Lecomte et al., 2010; MacNulty et al., 2009). Changes in markers of the immune system, broadly consistent with immunosenescence, have also been documented in some studies of wild birds and reptiles (reviewed in Palacios et al., 2011), and in one wild mammal population (Nussey et al., 2012), although it is notable that several studies in birds and reptiles have failed to find patterns consistent with age-related declines in immune function (e.g. Lecomte et al., 2010; Massot et al., 2011; Palacios et al., 2011; Schwanz et al., 2011).

Evidence for senescence in vertebrates other than birds and mammals is much harder to come by (Fig. 1A and B, Table S1). We were able to find only one study of amphibians, which suggested retinal structure and function might be compromised in larger – and therefore older – wild tiger salamanders (Townes-Anderson et al., 1998). Similarly, evidence in wild fish appeared limited to studies of Pacific salmon (*Oncorhynchus nerka*), which are semelparous and show rapid senescence on returning to their natal freshwater streams to breed (e.g. Morbey et al., 2005), and guppies (*Poecilia reticulata*; Bryant and Reznick, 2004). The evidence from wild reptiles is a little healthier, with particularly compelling evidence of age-related declines in survival from a long-term study of common lizards (*Lacerta vivipara*, Massot et al., 2011). However, long-term studies of two species of turtles found no evidence for survival senescence (Congdon et al., 2001, 2003). Monitoring fecundity and reproductive performance in the wild is exceptionally challenging in reptiles (not to mention fish or amphibians), and many studies have circumvented this by bringing wild-caught females into the laboratory to breed and subsequently releasing them and their young back at their original capture site (e.g. Massot et al., 2011; Sparkman et al., 2007). Evidence from studies of this nature for

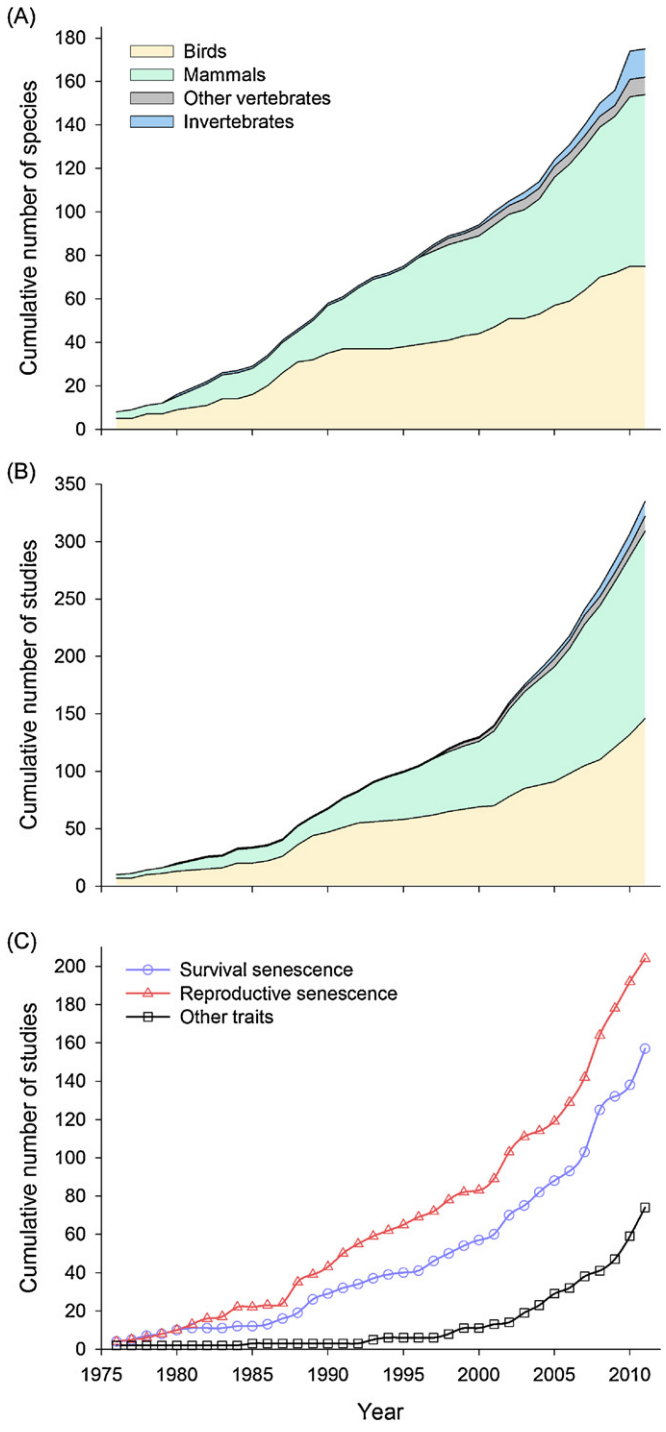

Fig. 1. The evidence in support of senescence occurring in wild animals is now overwhelming. The plots use the studies listed in Table S1 to illustrate the rapidly accumulating number of: (A) new species in which age-related changes consistent with senescence have been documented in the wild and, (B) individual studies documenting or investigating senescence patterns in wild populations. The data are split to separately illustrate the evidence for birds, mammals, other vertebrates (fish, reptiles and amphibians), and invertebrates. Part (C) shows the cumulative number of studies documenting age-related change in survival probability, reproductive performance traits and change in other, typically physiological, traits with age indicative of senescence. Note that many studies document senescence in more than one type of trait, and that in all plots the first year (1976) on the graphs included the handful of studies up to and including that year.

reproductive senescence in reptiles remains very limited: common lizards show age-related declines in fecundity and offspring survival when kept in semi-natural enclosures (Richard et al., 2005) but studies of wild turtles and garter snakes show very little evidence of reproductive aging (Congdon et al., 2001, 2003; Miller, 2001; Sparkman et al., 2007). Age-related increases in reproductive performance, associated with indeterminate patterns of growth observed in many fish, reptiles and amphibians have been shown theoretically to offset the weakening of natural selection with age due to age-independent environmentally-driven mortality, making these taxa potentially very interesting as models of delayed, negligible or even negative senescence (Baudisch, 2008; Congdon et al., 2003; Sparkman et al., 2007).

Evidence for senescence in wild insects is also emerging (Fig. 1A and B), although we were unable to find evidence for senescence in the wild from other invertebrate taxa (e.g. molluscs, crustaceans or cephalopods). The challenges involved in monitoring the demography and age structure of such small and dispersive creatures in the wild are extreme. Studies to date have focused on species that form aggregations of some kind, so that individuals can be marked and subsequently recaptured. In a ground-breaking study, Bonduriansky and Brassil (2002) individually marked and followed male antler flies (*Protopiophila litigata*) across their reproductive lives, which are spent on the cast antlers of ungulates. Their data on age-specific mortality and mating rates provides incontrovertible evidence for senescence in the wild in a species that lives only a few weeks (Bonduriansky and Brassil, 2002). A recent re-analysis of mark-recapture data from wild dragonflies and damselflies suggests that survival senescence is common in these groups (Sherratt et al., 2011). In addition, age-related increases in wing damage and mortality risk, as well as declining foraging performance with age indicate senescence in worker honey bees (*Apis mellifera*) foraging in the wild (Dukas, 2008a,b; Higginson and Barnard, 2004). Recently, methods have been developed to estimate the age structure and aging rates in taxa like insects that are notoriously difficult to monitor longitudinally in the wild (Carey et al., 2008; Muller et al., 2004; Zajitschek et al., 2009b). Although they rest on quite restrictive assumptions, these methods have already provided evidence for survival senescence in wild black field crickets (*Teleogryllus commodus*, Zajitschek et al., 2009b). Research into senescence and lifespan in wild insects is an exciting area with unique potential to link our understanding of aging in laboratory conditions to aging in a realistic evolutionary context (Carey, 2011; Zajitschek et al., 2009b).

The difficulties associated with detecting and measuring senescence in the wild are manifold, and have been widely discussed (Brunet-Rossinni and Austad, 2006; Gaillard et al., 1994; Nussey et al., 2008a). For instance, imperfect recapture probabilities of marked individuals can bias estimates of age-dependent survival and reproduction (Bouwhuis et al., 2012; Gimenez et al., 2008), phenotypic heterogeneity among age classes associated with selection can generate cross-sectional age patterns that are unrelated to underlying within-individual aging patterns (Cam et al., 2002; Reid et al., 2003), and survival can decline in young or old age as a result of costs associated with increased investment in growth or reproduction instead of senescence (Bonduriansky and Brassil, 2002; Promislow, 1991). However, recent studies analysing high-quality longitudinal data with statistical tools capable of accounting for selective effects and recapture rates, alongside simultaneous assessment of survival and reproductive traits, have overcome these issues. Although some meticulous analyses of high quality data from wild bird and mammal populations have failed to find evidence of senescence (e.g. Altwegg et al., 2007; Nichols et al., 1997; Slade, 1995), such cases are now in the clear minority. Any debate about whether wild animals senesce or not is over, quashed beyond argument by the empirical data highlighted in Fig. 1 and

Table S1. The major outstanding challenge is to understand the physiological, evolutionary and ecological causes of variation in senescence and its fitness costs in the natural world. Why do some species not appear to show senescence in the wild (Austad, 2010)? Why do the fitness costs of senescence vary among species and populations (Bouwhuis et al., 2012; Ricklefs, 2010)? And why do individuals vary so much in the onset and pattern of senescence within wild populations (Brunet-Rossinni and Austad, 2006; Nussey et al., 2008a)?

3. Understanding the causes and consequences of variation in senescence in wild animal populations

The identification of environmental and genetic manipulations that extend life- and health-span in a handful of short-lived organisms under laboratory conditions has revolutionized our understanding of the aging process and raised real hopes of developing medical interventions that extend healthy life in humans (Fontana et al., 2010; Partridge, 2010). However, variation in lifespan and patterns of senescence evident among species, populations and individuals in nature dwarfs the variation observed in model laboratory organisms. The causes of this natural variation remain poorly understood. Long-term individual-based field studies provide longitudinal data on age-related fitness and phenotypic changes that represent high-quality – but largely neglected – fuel for comparative studies to test evolutionary predictions regarding the causes of variation in aging among species. Most comparative studies to date used either maximum recorded lifespan or life table-derived survival data (de Magalhaes and Costa, 2009; Ricklefs, 2010; Turbill and Ruf, 2010), which come mostly from captive populations and ignore the wealth of available higher quality longitudinal survival and reproduction data from capture-mark-recapture studies in natural settings. A recent study which collated such field data and used them to estimate the onset of reproductive and survival senescence among bird and mammal species, illustrates the possibilities of such an approach (Jones et al., 2008). Similarly, in-depth ecological studies across populations of a species experiencing different environmental conditions and mortality pressures have facilitated "common garden" experiments, which compare lifespan and senescence in individuals from different natural populations under controlled laboratory conditions (e.g. Bronikowski and Arnold, 1999; Bryant and Reznick, 2004). Such work has shed important new light on how natural selection might shape the ageing process (e.g. Reznick et al., 2004; Robert and Bronikowski, 2010; Robert et al., 2007).

Longitudinal studies in natural populations can also provide crucial insight into the drivers of individual variation in senescence, and in the last decade, there has been growing interest and activity among researchers studying wild animals towards this aim. Fig. 2 shows that the increase through time in the number of studies investigating senescence in the wild has been considerably more rapid than the increase in number of new species in which senescence has been documented in nature, over the last 15 years or so. This pattern most likely reflects the fact that ecologists are increasingly shifting focus from simply describing patterns of age-related variation in a given species or population, and towards using data from long-term, individual-based field studies to test evolutionary hypotheses of aging and understand the causes and consequences of among individual variation in senescence. Whilst it is undoubtedly important to broaden the phylogenetic spectrum of aging studies in nature, this shift towards more detailed research into single or a few very well-studied wild systems is important as it has lead to a rapid advance in our understanding of senescence in natural systems. Below we review progress in addressing several questions of considerable interest to bio-gerontologists in wild animal populations.

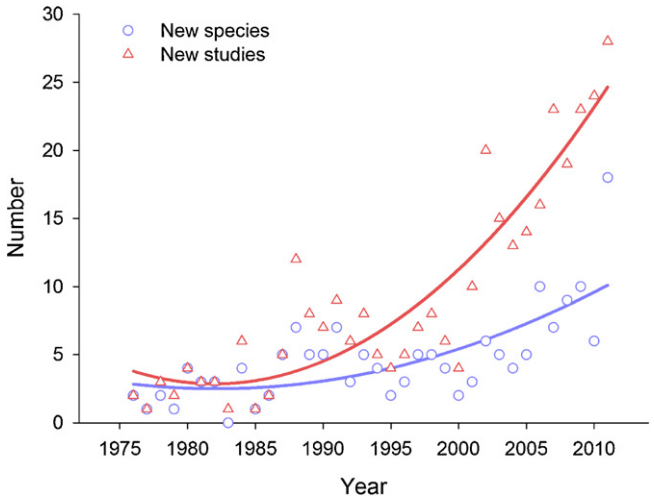

Fig. 2. The total number of new species per year (blue circles) and total number of studies per year (red triangles) documenting senescence in wild animal populations (from Table S1), with quadratic regression lines plotted through the points. There has been an accelerating increase in the number of new studies per year over the last decade, reflecting a shift towards in-depth research programs into aging patterns on single high-quality long-term study systems in the wild.

3.1. Sex differences in senescence

In humans, sex differences in lifespan are well documented, with evidence suggesting women have lower mortality rates throughout life but a similar increase in mortality risk with age than males (Austad, 2006, 2011). However, reproductive senescence – in the form of menopause – happens much earlier in females than in males, and the degree to which other patterns of physiological deterioration with age are sex-biased remains a source of debate (Bonduriansky et al., 2008; Graves et al., 2006). Mammalian and invertebrate laboratory model systems also show sex differences in lifespan and sexes often differ in their response to genetic or dietary manipulations associated with lifespan extension (e.g. Bokov et al., 2011; Clancy et al., 2001; Selman et al., 2009; Strong et al., 2008). Evolutionary theory offers explanations for how and why differences in lifespan and aging might have arisen under natural selection (Bonduriansky et al., 2008; Williams, 1957). Based on the idea that the strength of natural selection against senescence hinges on the rate of 'extrinsic mortality' experienced in nature, Williams (1957) predicted "where there is a sex difference [in "extrinsic" mortality], the sex with the higher mortality rate and lesser rate of increase with fecundity should undergo the more rapid senescence." Male-biased natural mortality rates are very widely observed in polygynous species, including many mammals and insects, where males tend to be larger and show mating behaviours and secondary sexual characteristics associated with substantial energy expenditure and mortality risk, but evidence for sex differences in senescence rates from the wild is much more limited (Bonduriansky et al., 2008; Clutton-Brock and Isvaran, 2007). Some support for the prediction has been obtained from a comparative study using data from long-term field studies of birds and mammals which found that male-bias in survival senescence rates increased with the degree of polygyny, and was largely absent in monogamous birds (Clutton-Brock and Isvaran, 2007). However, most studies of senescence in the wild have tended to focus on a single sex – typically females – and direct and detailed comparison of aging patterns between the sexes remain rare.

There is evidence that males show more rapid actuarial senescence than females from several wild ungulate populations (Catchpole et al., 2004; Gaillard et al., 1993; Jorgenson et al., 1997; Loison et al., 1999; Toigo et al., 2007), and from a single study of

common lizards (Massot et al., 2011). Perhaps unsurprising, given their typically monogamous mating system and generally lack of sex bias in mortality; we have not been able to find evidence in the literature for significantly sex-biased survival senescence in wild birds. The few relevant studies on insects are puzzling: black field crickets studied in the wild showed no sex differences in the Gompertz function describing the rate of age-related increase in mortality risk, but when kept and monitored in "semi-natural" enclosures females showed a more rapid mortality rate increase but with a later onset than males (Zajitschek et al., 2009a,b). In stalk-legged flies, males were found to show rapid actuarial senescence in the wild but females were not (Kawasaki et al., 2008).

Studies directly comparing rates of reproductive senescence between the sexes in the wild remain very rare, not least because of the considerable challenges in determining reproductive success of males in polygynous species and in separating the contributions of the sexes to reproductive success in socially monogamous species. Field studies have provided evidence for age-related declines in a variety of male reproductive traits including competitive behaviour (Bonduriansky and Brassil, 2005; Jennings et al., 2010; Nussey et al., 2009), secondary sexual characteristics (Moller and De Lope, 1999; Vanpé et al., 2007; Velando et al., 2010), sperm performance and damage levels (Moller et al., 2009; Velando et al., 2011), and ultimately the number of offspring sired (Coltman et al., 1999; Nussey et al., 2009; Vanpé et al., 2009). Likewise, there is evidence across a large suite of females traits associated with reproductive performance – from timing of egg laying or parturition to number of offspring and egg or offspring size, right through to maternal investment after hatching or birth and offspring survival (e.g. Beamonte-Barrientos et al., 2010; Bouwhuis et al., 2009; Hewison and Gaillard, 2001; Martin and Festa-Bianchet, 2011; Nussey et al., 2009) – and several recent studies suggest important longer-term effects of parental age on offspring fitness (Bouwhuis et al., 2010b; Torres et al., 2011).

The challenge remains to quantitatively integrate and compare these changes in reproductive performance with age, and ultimately the differences in fitness costs of senescence, between the sexes (Bonduriansky et al., 2008; Promislow, 2003). In birds, social monogamy makes separating the contributions of female and male age from reproductive performance traits challenging, although a few studies have found evidence of sex-differences in aging of reproductive performance traits (Lecomte et al., 2010; Reed et al., 2008; Reid et al., 2003), or interactions between male and female age influencing such traits (Auld and Charmantier, 2011). In mammals, age-dependent fecundity trajectories have been compared between sexes in wild red deer and Soay sheep; both studies suggested that age-related declines begin slightly later but progresses more rapidly in males than in females (Nussey et al., 2009; Robinson et al., 2006), although the generality of this pattern remains to be confirmed.

From an evolutionary perspective, a central question is whether differences in natural selection on life history between sexes (sexually antagonistic selection) has resulted in the evolution of genetically programmed differences in allocation to somatic maintenance, and therefore in lifespan and aging patterns. Some support for this hypothesis comes from a cross-sectional study of culled red deer, in which – like many other ungulates – adult teeth are not replaced and tooth wear has been suggested as an important determinant of mortality in older animals (Carranza et al., 2004). The study took starting tooth size as an indicator of resource allocation to somatic maintenance and wear rate as a proxy of the need for repair and showed that, despite males being considerably larger and heavier than females and showing faster molar wear, sexual dimorphism in molar height in early life was minimal (Carranza et al., 2004). This supports life history theories, suggesting males allocate less in maintenance in accordance with their shorter life expectancy (Carranza et al., 2004). An alternative possibility is that earlier or more rapid senescence in males may be the direct physiological result of their heavier energetic allocation to growth and reproduction in earlier life, compared to females. Supporting evidence from wild populations remains scarce, although there is evidence of increased costs of reproduction in older individuals in single sex studies (e.g. Descamps et al., 2009; Torres and Velando, 2007). Studies comparing age-dependent costs of reproduction across sexes are lacking, although a very recent analysis of four European populations of barn swallows suggests males, but not females, pay a longevity cost of allocating resources to a secondary sexual characteristic, namely having long tail feathers (Balbontin et al., 2011).

3.2. Variation in senescence among traits

Williams (1957) predicted that senescence in different physiological systems associated with fitness should progress in synchrony, and reiterated the "expected evolution of synchrony" in a much later monograph on aging (Williams, 1999). This was odd as in both papers he discussed at length the discrepancies between the onset of athletic decline, menopause and life expectancy in humans. He appeared to think this obvious heterochrony was a quirk of our own species, stating: "modern human populations. . . have grossly abnormal environments that may greatly disrupt the coupling of senescence" (Williams, 1999). Evidence from the laboratory has revealed similarly complex and divergent patterns of senescence across physiological systems and functions (Burger and Promislow, 2006; Grotewiel et al., 2005; Herndon et al., 2002), leading to calls for more careful consideration of the system of heterochronous interactions associated with organismal aging (Promislow et al., 2006; Walker and Herndon, 2010). Of course, these examples might also simply reflect vagaries of the 'grossly abnormal environments' experienced in the laboratory compared to ecological reality. However, emerging evidence from natural populations refutes Williams' prediction and suggests heterochrony is also a reality in evolutionarily relevant natural contexts.

Long-term individual-based field studies often collect longitudinal data on a host of phenotypic traits, including behavioural, reproductive and physiological parameters, as well as information on survival, as already discussed (see Section 3). There has been a very recent surge in studies directly comparing senescence rates across such traits, following calls for more integrated studies of aging in the wild (Massot et al., 2011; Nussey et al., 2008a). Evidence for uncoupling of patterns of survival and fecundity senescence remain quite scarce. In females, post-reproductive survival has been documented in wild mammals, but with the possible exception of some toothed whales, this seems more likely the product of chance than some adaptive reproductive cessation (Cohen, 2004; Packer et al., 1998; Thompson et al., 2007; Foote, 2008; Ward et al., 2009). There is also some evidence from wild female ungulates that fecundity senescence may begin later and progress more rapidly than age-related declines in survival probability (Bérubé et al., 1999; Catchpole et al., 2004; Jorgenson et al., 1997; Nussey et al., 2009). There is much clearer evidence for heterochrony of senescence among traits associated with reproduction in both males and females (e.g. Beamonte-Barrientos et al., 2010; Berman et al., 2009; Descamps et al., 2007; Ericsson et al., 2001; Evans et al., 2011; Hewison and Gaillard, 2001; Massot et al., 2011; Moller and De Lope, 1999; Nussey et al., 2009). For instance, emerging data suggest that male secondary sexual traits – despite theoretical expectations that they should be physiologically costly (Andersson, 1994) – do not actually senesce, although reproductive performance clearly does (Evans et al., 2011; Nussey et al., 2009). Evidence remains mixed on this front, with several studies documenting age-related declines in such traits (Moller and De Lope, 1999; Mysterud

et al., 2005; Vanpé et al., 2007; Velando et al., 2010). Further careful integrated cross-trait comparisons are now required to understand why and how such heterochrony might evolve under natural conditions (Massot et al., 2011; Nussey et al., 2008a; Promislow et al., 2006).

3.3. Individual, genetic and environmental variation in senescence rates

Just as patterns of senescence appear to vary across systems and traits in humans and in model organisms, so they are known to vary dramatically among individuals. Field studies testify that age-related declines in fitness-associated traits vary among individuals in natural populations. A number of studies have directly quantified individual variation in aging rates (Brommer et al., 2007, 2010; Wilson et al., 2007), whilst others have provided indirect evidence by documenting associations between early-life environment or life history and aging rates later on (Bouwhuis et al., 2010a; Hayward et al., 2009; Massot et al., 2011; Nussey et al., 2007; Reed et al., 2008). Several studies have also compared whether declines in such traits are better predicted by chronological age or years until death. The latter would provide support for the idea that declines were to some degree age-independent. Evidence is mixed, although there is growing support for some degree of age-independent deterioration, presumably reflecting differences in past experience or genetic make-up among individuals (Hayward et al., 2009; Martin and Festa-Bianchet, 2011; Nussey et al., 2011).

Classical evolutionary theory predicts that variation in the aging process should have a genetic basis (Rose, 1991), and work in model laboratory systems supports this prediction both through artificial selection experiments and through genetic manipulations that influence aging and lifespan (Kirkwood and Austad, 2000; Partridge, 2010). This research suggests conserved or 'public' genetic and physiological pathways, which are modulated by diet, across distantly related taxa to modulate aging and lifespan (Fontana et al., 2010; Partridge, 2010). However, the generality of these findings to longer-lived organisms or even to the same species experiencing the more complex and challenging environments of the natural world has yet to be established, as has the evolutionary significance of laboratory mutations, knock-outs and knock-ins associated with lifespan extension. Recent work on nematodes, flies and mice shows that changing the environmental context profoundly alters how long these mutants live and how well they perform, and suggests that the responses of wild-caught animals to typical treatments can differ markedly from those subjected to years of selection for laboratory life (e.g. Chen et al., 2007; Giorgio et al., 2012; Harper et al., 2006; Linnen et al., 2001; Sgro and Partridge, 2000; van Voorhies et al., 2005). In many respects, understanding these complex genotype-by-environment interactions and the lifelong fitness and health consequences of dietary, pharmaceutical and genetic manipulations that extend lifespan for lab models in long-lived organisms under more complex environmental conditions is pivotal to hopes of meaningful intervention to alleviate senescence in humans and domestic animals.

Several studies of wild birds and mammals have used reconstructed population pedigrees and quantitative genetic methods to test whether aging rates in fitness-related traits are heritable (reviewed by Wilson et al., 2008). Specifically, in red deer, Soay sheep, and mute swans increased genetic variation with age and, by implication, heritable variation in aging patterns, has been documented (Charmantier et al., 2006a; Wilson et al., 2007). Importantly in the swan study, the association between early first reproduction and early last reproduction mentioned earlier was shown to have a genetic basis, providing perhaps the most compelling evidence yet for antagonistic pleiotropy from a wild animal (Charmantier et al., 2006b). Also, in red deer, data suggest that the association between increased maternal resource allocation towards reproduction in early life and rapid reproductive senescence in red deer had a genetic basis, although the effect was marginally statistically non-significant ($P = 0.06$; Nussey et al., 2008b). Additionally, studies in common gulls and collared flycatchers documented variation in aging rates but did not find evidence that this variation had a genetic basis (Brommer et al., 2007, 2010). It remains unclear whether these latter studies failed to find a genetic basis for variation in aging rate due to a lack of statistical power or to real biological differences among species. Researchers studying aging in fish and reptiles have tended to adopt a different approach, bringing individuals from populations experiencing different selective pressures into the laboratory and comparing their survival and physiology under controlled laboratory conditions. This approach clearly identifies genetic differences among populations that influence aging rates or maintenance and repair mechanisms, providing some support as well as important challenges to classical evolutionary theories of aging (Reznick et al., 2004; Robert and Bronikowski, 2010; Robert et al., 2007).

Studies in wild animals provide spectacular examples of the effect that the environment can have on the aging process. For instance, a comparison of mortality curves in wild and laboratory stalked-legged flies revealed that males senesce at least twice as rapidly under natural conditions as in the laboratory (Kawasaki et al., 2008). Also, wild medflies brought into the laboratory as adults often survived considerably longer than laboratory-reared flies did from hatching, suggesting a profound effect of early environment on aging rates (Carey et al., 2008). In longitudinal field studies of birds and mammals poor conditions in early life are seen to exacerbate the rate and onset of decline in survival and reproductive performance (Beamonte-Barrientos et al., 2010; Bouwhuis et al., 2010a; Hayward et al., 2009; Nussey et al., 2007; Reed et al., 2008), and commonly show that the survival of older adults is more sensitive to environmental conditions (e.g. Gaillard et al., 2000; Reichert et al., 2010; Toigo et al., 2007). An interesting pattern emerged from a recent study of tawny owls, which experience profound variation in food availability associated with population cycles of their main prey. Food availability early in life affected survival and reproductive performance in later life, but it did not appear to influence aging rates per se (Millon et al., 2011). The relationship between environmental conditions and senescence is clearly very complex in natural populations, and identifying the precise environmental or physiological causes of relationships is often likely to be impossible. However, field researchers, particularly those working on birds, have a long history of integrating longitudinal field data collection with experimental manipulation. The integration of field observation and field experimentation could allow us to determine the effects of factors such as nutrition, infection, thermal stress and competition for lifespan and aging rates in nature.

3.4. Tests of life history theories of aging

Both antagonistic pleiotropy and disposable soma theories of aging rest on the central idea of trade-offs between early life reproduction and later life maintenance of physiological function and reproductive output (Kirkwood and Rose, 1991). As already discussed, work on laboratory model systems generally supports these 'life history' theories of aging (Table 1, Kirkwood and Austad, 2000; Partridge and Barton, 1996). However, environmental conditions are likely to alter both the nature and magnitude of the costs involved and the way that natural selection acts on life history allocation, and laboratory conditions are profoundly different to those experienced by wild animals. To understand how the environment and natural selection interact to shape variation in senescence among species, populations and individuals it is

crucial to test these hypotheses in natural settings. The emergence of long-term, individual-based field studies and statistical tools to dissect within- and between-individual variation has now made it possible to robustly test the consequences of an individual's early life history decisions for life expectancy and aging patterns in later life (Cam et al., 2002; Rebke et al., 2010; van de Pol and Verhulst, 2006). Importantly, evidence is now emerging from natural populations supporting the central prediction that an increased allocation of resources to early life reproduction is associated with earlier or more rapid aging in later life.

For instance, a long-term study of collared flycatchers in Sweden, where researchers experimentally increased the brood size of females in early adulthood, has shown that these females produced consistently smaller subsequent broods, with a suggestion that brood size also declined more rapidly with age, compared to control females (Gustafsson and Part, 1990). Subsequently, studies of female great tits, guillemots and red deer have all demonstrated that increases in fecundity or reproductive performance in early adulthood are associated with more rapid declines in reproductive performance in later life (Bouwhuis et al., 2010a; Nussey et al., 2006; Reed et al., 2008). In meerkats, a social mammal in which a dominant pair almost completely monopolize breeding attempts within any group, females experiencing more stressful ascensions to breeding dominance – as a result of competition with same-age siblings in the group – show more rapid reproductive senescence in later life (Sharp and Clutton-Brock, 2011). Also, mute swans that start their breeding careers earlier in life end their reproductive lifespan earlier as well (Charmantier et al., 2006b), and red squirrels that start breeding early have shorter subsequent life expectancies (Descamps et al., 2006). An intriguing variation on this pattern is seen in female common lizards. Those that allocate relatively more in offspring at two years of age actually show increased subsequent reproductive success but at the cost of more rapid declines in survival probability, in essence sacrificing lifespan for high reproductive output (Massot et al., 2011). Similar evidence of a trade-off between parental lifespan and offspring survival has been recorded in a bird species, the red-billed chough (Reid et al., 2010). Whilst these studies support the expectation of maintenance or survival costs of early reproduction, some also show that natural selection favours individuals allocating more resources towards early-life reproduction, as theory would predict (Bouwhuis et al., 2010a; Descamps et al., 2006; Gustafsson and Part, 1990). Where field studies have tested the predictions of life history theories of aging with detailed longitudinal data and robust statistics, they have tended to find support (Peron et al., 2010).

4. Conclusions: why bio-gerontologists should care about aging in natural populations

We have discussed why, under the life history theories of aging, there is no theoretical reason to adduce that wild populations should not senesce. We have also presented the now very considerable evidence for senescence in wild birds and mammals, and the emerging evidence from wild populations of other vertebrates and insects. The emergence of long-term, individual-based field studies has provided the incontrovertible backbone of this evidence. It also represents high quality, but thus far largely unused, data for future comparative studies of aging among species and populations. Such longitudinal research in the wild has also provided important new insights and tests of evolutionary predictions regarding sex differences in senescence, the genetic and environmental drivers of among individual variation in aging, and the costs of early-life reproduction for somatic maintenance and senescence under natural conditions.

Few would dispute that aging and longevity differ drastically depending on the environment in which they are measured, and that we cannot characterize the patterns and processes of aging without reference to the specific environmental conditions they are measured in (Carey, 2011; Kawasaki et al., 2008). The laboratory-based model organism approach employed by biomedically-oriented researchers suggests potentially conserved pathways underlying variation in aging rate, but the importance of such pathways may depend on life history, genetic background and environmental context. Studies linking genes, environment and aging rates in wild animals could help test the generality of laboratory findings in species experiencing challenging environments, or suggest genes or environmental effects linked to aging in natural conditions that could be explored in model organisms. Free-living populations provide an alternative extreme to controlled and protected laboratory conditions, and if mechanisms of aging can be found to generalize from laboratory to wild then this could identify environmentally robust targets for aging intervention. Bio-gerontologists have focused on conserved or 'public' mechanisms of aging across species that have been maintained, bred for many generations, and measured under laboratory conditions, but is it not time to consider whether such mechanisms are public across a broader environmental and genetic spectrum? At the same time, population, evolutionary and physiological ecologists have a great deal to learn from bio-gerontologists and bio-demographers. Investigation of the cellular pathways identified as regulating lifespan and ageing in model organisms could shed important light on the physiological processes that link genes, nutritional availability and quality, life history and, ultimately, demographic rates and fitness in natural populations. Likewise, the more systematic incorporation of age-related demographic changes in later life within ecological models could help capture population dynamics in a more biologically relevant and complete manner.

To date, discourse between bio-gerontologists interested in identifying conserved mechanisms underpinning the aging process and evolutionary ecologists interested in explaining variation in the natural world using evolutionary theory has been limited. We argue this can and should change, and this would be to the mutual benefit of both sides. For instance, an integrative approach combining laboratory experiments, field experiments and analysis of longitudinal field data could provide more general insights, and several species may be amenable to comparison across environmental conditions, from laboratory or domestic conditions to natural state (e.g. Carey, 2011; Kawasaki et al., 2008; Mysterud et al., 2002). Furthermore, the increasing availability and affordability of next generation genomic tools in non-model systems means that field ecologists and bio-gerontologists could collaborate to test whether genes associated with aging and lifespan in humans and model organisms show any variation in wild populations, and determine how natural selection acts to maintain any evident genetic variation. Finally, the trade-offs between somatic maintenance and other fitness functions predicted under the disposable soma hypothesis of ageing represent a serious challenge for bio-gerontologists. Whilst evidence for such costs associated with dietary or physiological interventions that extend lifespan in model organisms are mounting (Table 1), these may only poorly reflect the actual costs of increasing life- or healthspan in more challenging environments. Field studies often fail to detect meaningful fitness costs of growth or reproduction except under harsh conditions, and the magnitude and nature of the costs are expected to vary with a species life history (Hamel et al., 2010). More generally, the detailed longitudinal data collected by field ecologists will allow researchers to link growth and development, parental care and infection in early life with health and survival in later adulthood in a manner rarely possible in either laboratory models or in extremely long-lived species like humans.8

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