1. Introduction

Bats and birds live substantially longer on average than non-flying homeotherms of similar body size (Austad and Fischer, 1991; de Magalhaes et al., 2007; Prinzinger, 1993). Within mammals, the largest differences in longevity tend to occur between orders, whereas among birds the largest differences occur between genera (Fig. 1). On average, maximum bat lifespans are 3.5 times longer than non-flying eutherian mammals after correcting for body size (whereas among birds the largest differences occur between genera (Austad and Fischer, 1991; de Magalhaes et al., 2007; Prinzinger, 1993)). Similarly, many birds live three times longer than mammals of the same body size (Fig. 1, Holmes and South, 2002). Records now exist of tiny bat "Methuselahs," such as the 7 g Brandt's bat (Myotis brandti), surviving in the wild for over four decades (41 years, Gaisler et al., 2003; Podlutsky et al., 2005). Similarly, many birds live three times longer than mammals of the same body size (Fig. 1, Holmes and Austad, 1995a; Holmes and Austad, 1995b). Although reports of centenarian parrots are apocryphal, cockatoos and Amazon parrots do exhibit extreme lifespans after accounting for body mass (Munshi-South and Wilkinson, 2006). A salmon-crested cockatoo (Cacatua moluccensis) named "King Tut" lived at the San Diego Zoo for at least 65 years (Brouwer et al., 2000); much larger birds, such as the Andean condor (Vultur grifhicus), may live up to 75 years (Finch, 1990).

Evolutionary theories of longevity provide explanations for why bats and birds have evolved long lifespans. These theories predict that average lifespan should increase as the probability of death caused by extrinsic factors (e.g. accidents, infectious disease, and predation) decreases (Austad and Fischer, 1991). Deleterious mutations that act late in life will be exposed to relatively strong selection in populations that do not experience high extrinsic mortality at young ages (Austad, 1997), and thus will not accumulate over time. Antagonistic pleiotropy caused by late-acting deleterious mutations that have positive benefits early in life will also have a weaker impact on populations with low extrinsic mortality risk (Partridge, 2001). Experimental data comparing insular vs. mainland populations of both marsupials (Austad, 1993) and mice (Harper, 2008; Miller et al., 2000) indicate that insular populations experiencing lower predation risk have evolved greater longevity. Ageing rates are directly related to mortality risk in birds and mammals (Ricklefs, 1998; Ricklefs and Scheuerlein, 2001), and flight is believed to be the primary characteristic that helps birds and bats avoid extrinsic mortality early in life (Holmes and Austad, 1994). Bats and birds represent two independent evolutionary origins of flight, and thus comparative research may reveal common evolutionary pathways to long lifespan.

Life history tradeoffs may also explain why long lifespans have evolved in bat and bird species, because lifespan evolves as a consequence of joint selection for current reproduction along with survival and future reproduction. The "disposable soma" theory of ageing predicts that species experiencing low extrinsic mortality can make substantial investments in growth and somatic maintenance rather than early reproduction because they will have many opportunities to reproduce over a long lifespan.
that provide evidence of specific physiological mechanisms through which bats and birds either prevent or repair ROS damage.

Bats and birds are potentially excellent non-model systems to examine the evolution of longevity, especially in a comparative framework. Large longevity and life history datasets collected from wild populations now exist for both groups, primarily due to long-term banding studies (Ricklefs, 2008; Wilkinson and South, 2002) and increasingly sophisticated ageing methods (Brunet-Rossini and Wilkinson, 2009; Chaney et al., 2003; Vleck et al., 2003). Some long-lived birds, such as the parrots, have been kept in captivity for a long enough time to amass corroborated maximum lifespans for many species (Brouwer et al., 2000). Most of these records are freely available to researchers in a well-curated online database (AnAge, de Magalhaes et al., 2005). Comparative analyses have also benefited from the development of methods, such as independent contrasts analysis, that control for phylogenetic effects (Garland et al., 1992). Species data cannot be treated as statistically independent because species are related by descent from common ancestors (Felsenstein, 1985), but shared phylogenetic history has not always been accounted for in comparative ageing studies (Speakman, 2005). The availability of well-supported phylogenies was previously an impediment to these types of analyses, but the increasing acceptance of consensus ''supertrees'' (Hackett et al., 2008; Bininda-Emonds et al., 2007, bats, Jones et al., 2002, oscine passerine birds, Jonsson and Fjeldsa, 2006) and the production of robust molecular phylogenies (parrots, Wright et al., 2008) have largely removed these impediments.

Mechanistic research on longevity in bats and birds has lagged because few species have been kept in laboratory colonies (Holmes and Ottinger, 2003). However, the number and diversity of bird species in labs is slowly increasing, with long-lived budgerigars (Melopsittacus undulatus) showing particular promise as a model system (Ogburn et al., 2001; Pamplona et al., 2005). Captive bat colonies have been maintained for behavioral and physiological studies in the past (Brunet-Rossini and Austad, 2004), and now a few extremely long-lived Myotis species are emerging as ageing research models (Brunet-Rossini, 2004). These advances suggest that bats and birds are leading candidates for the "non-model" outgroup system sought by ageing researchers (Holmes and Kristan, 2008).

2. Longevity research in bats

2.1. Evolution of long lifespan and the risk of extrinsic mortality in bats

Hypothetical selective pressures responsible for the evolution of long lifespan in bats generally fall into two categories: (1) adaptations that lower the risk of extrinsic mortality (evolutionary theories of ageing), and (2) life history tradeoffs that favor long lifespan (disposable soma theory of ageing). Escape from extrinsic mortality due to the evolution of flight in bats is consistent with evolutionary theories for long lifespan, but convincing evidence for a general association between flight and longevity in mammals is scarce. Flying and gliding mammals exhibit longer lifespans (Austad and Fischer, 1991; Holmes and Austad, 1994), but flight or gliding behavior have evolved so few times in mammals that rigorous, phylogenetically controlled studies are not possible.

Roosting in caves should lower the risk of extrinsic mortality for bats, as caves provide protection from extreme weather events. Caves may also be inaccessible to predators, and communal roosting may provide increased vigilance against predators that do reach the cave. Among chiropterans, bats that occasionally roost in caves live longer than bats that never or always use caves, independently of reproductive rate, body mass, hibernation, or
phylegentic effects (Wilkinson and South, 2002). It is unclear why
obligate cave roosting is not associated with lifespan extension, but
increased transmission of disease or ectoparasites in permanent
cave roosts may influence extrinsic mortality rates. Species
richness of parasitic bat flies is higher in enclosed, permanent
roosts (Bordes et al., 2008), and bats within these roosts exhibit
greater prevalence and intensity of parasitism (especially females,
Christe et al., 2007; Patterson et al., 2007). Field experiments have
also confirmed that some bats switch roosts to avoid the costs of
ectoparasite load (Reckardt and Kerth, 2007).
171 Hibernation may also reduce extrinsic mortality risk by
protecting bats from extreme weather or starvation during periods
of resource shortage. Initial studies did not find that hibernating
bats live longer than non-hibernating species (Austad and Fischer,
1991; Herreid, 1964), but analysis of a larger dataset revealed a
positive association between hibernation and longevity indepen-
dent of body size, reproductive rate, and phylegentic effects
(Wilkinson and South, 2002). Latitude was not an effective
predictor of longevity after controlling for hibernation and
phylegentic effects in this analysis, despite a predicted associa-
tion of high latitude and long hibernation times. The current
longevity record holder among bats is a 41-year-old M. brandti in
Siberia (Podlutsky et al., 2005), and multiple individuals have lived
over 25 years in this population. An association between longer
duration of hibernation and increased lifespan should not be
discarded until more data from hibernating bats are available.
2.2. Physiological tradeoffs and longevity in bats

Hibernation may also extend lifespan in bats by reducing the
costs of reproduction relative to body size. Wilkinson and South
(2002) found that hibernating species have lower reproductive
rates, but that reproductive rate increases with body mass in
hibernating bats. The disposable soma theory predicts that ageing
results from progressive physiological deterioration when
resources are allocated to reproduction rather than somatic
maintenance and repair (Kirkwood, 2002). Hibernation in bats,
by reducing the need for somatic maintenance for weeks to months
per year, may conserve resources that can be used later for
reproduction.

Physiological tradeoffs between reproductive rate, investment
in offspring, and lifespan in bats also support the disposable soma
toery. Bats generally exhibit lower reproductive rates than
shorter lived mammals (Barclay et al., 2004), and within Chiroptera
lifespan is shortened among species with high reproductive rates
regardless of whether the longevity record comes from captive or
wild individuals (Wilkinson and South, 2002). Within some
species, such as Rhinolophus ferrumequinum, individuals that breed
earlier also exhibit reduced lifespan compared to individuals that
breed later (Ransenome, 1995). Rates of embryo development and
postnatal growth also explain a significant proportion of variation
in ageing-related mortality among mammals (Rickles, 2006; Rickles and Scheurerlein, 2001). A recent analysis of 606 mammal
species that accounted for phylogeny further indicated that species
that live a long time for their body size (i.e. bats and primates) take
a long time to reach maturity (de Magalhaes et al., 2007). Energetic
investment in rapid development and early reproduction is
predicted to impose a cost on somatic maintenance later in life,
and these results from mammals provide support for this idea.
However, the evolution of these same life history traits may be
influenced by extrinsic mortality rates, and thus disposable soma
and evolutionary theories of ageing may not provide simple,
mutually exclusive explanations for lifespan evolution. In other
words, it is difficult to distinguish between evolutionary changes in
lifespan that are due to life history changes driven by extrinsic
mortality vs. life history tradeoffs driven by other factors.
2.3. Potential biomarkers of longevity in bats: fibroblast replication and calpain activity

The exceptional longevity of bats has been noted for a few
decades now, but few mechanistic ageing studies have been
conducted on bats in the laboratory. Rohme (1981) included one
bat (Vespertilio murinus) in an analysis of fibroblast lifespan and
longevity of eight mammalian species that sought to examine the
hypothesis that fibroblast activity is regulated by a process related
to organisal longevity. Fibroblast life span was positively correlated with species maximum life span in this study, and
the bat species had the third longest period of fibroblast activity
despite its relatively small body size. This study has been criticized
for mixing adult- and embryo-derived fibroblasts with different
replicative potential (Cristofalo et al., 1998), and an earlier analysis
found no association between fibroblast replication and longevity
in mammals (Stanley et al., 1975). A recent analysis of cell lines from 1 bat and 10 other mammalian species found that body size is
a much better predictor of fibroblast replication than maximum
longevity (Lorenzini et al., 2005). Some long-lived species in this
study still exhibited very high fibroblast proliferation after controlling for body size, but the authors are silent on whether
the bat species (Eptesicus fuscus) was among them.
Calpain activity in the brain has been implicated as a biomarker of longevity in bats, but only one study has been completed to date
(Baudry et al., 1986). Calpains perform important proteolysis
functions in many cell types, and elevated calpain activity has been
hypothesized to result in cellular ageing due to oxidative
destruction of structural proteins and coupled generation of
cell-damaging protein fragments (Nixon, 2003). Calpain-related
tissue degeneration manifests in several human ageing disorders,
including cataract formation, arthritis, and Alzheimer’s disease.
Baudry et al. (1986) hypothesized that calpain activity in brain
tissue from two long-lived bat species (Antrozous pallidus and
Tadarida brasiliensis) would be lower than calpain activity in the
brain of the short-lived laboratory mouse. While this study did
defy the new levels of calpain activity in bat vs. mouse brains,
larger comparative datasets are needed to confirm whether this
mechanism is a prominent explanation for extended bat lifespan.
2.4. Mitochondrial DNA mutation rates, oxidative damage, and
longevity in bats

The majority of studies on mechanisms of longevity in bats have
tested predictions of free radical or oxidative stress theories of
ageing. These theories describe the ageing process as the result of
accumulating cellular damage from reactive oxygen species (ROS)
that are produced continuously throughout life by aerobic
metabolism (Sanz et al., 2006). Long-lived species should experience less oxidative damage from ROS and/or have better
defenses against such damage, but some controversy remains over
whether long-lived, non-model organisms such as bats generally
exhibit these characteristics (Buffenstein et al., 2008). Several
recent studies have reported characteristics of the bat mitochon-
drial genome that may protect against oxidative damage to mitochondrial DNA (mtDNA). The mitochondrial genome should
be particularly susceptible to deleterious mutagenesis due to the
proximity of mtDNA to the site of ROS generation; mtDNA also
contains many direct repeats that are inherently more susceptible
to deletions that degrade mitochondrial function over time. Khaidakov et al. (2006) reported that bats have significantly fewer
direct mtDNA repeats (8–10 bp) than other mammals, and
predict that a lower mtDNA deletion rate partially explains
exceptional longevity in bats. However, all vespertilionid bats
possess direct, tandem repeats of a 78–85 bp portion of the mtDNA
control region (Wilkinson et al., 1997), and this family contains

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species with the greatest range of size-adjusted longevity of any family of mammals (Fig. 2). Given that duplication and deletion events may be common in the mtDNA of vespertilionid bats (Brunet-Rossinni and Wilkinson, 2009; Wilkinson and Chapman, 1991), a relationship between direct repeats and longevity is not a simple explanation for bat lifespans.

Mitochondrial theories of ageing predict that long-lived species will exhibit lower mtDNA mutation rates as an adaptation to reduce cumulative damage from ROS (Kujoth et al., 2007). In a comparative study of cytochrome b neutral substitution rate in 1696 mammalian species, Nabholz et al. (2008) found that bats (n = 222 spp.) exhibit substitution rates that are two times lower on average than substitution rates in rodents (n = 734 spp.), despite 6.6 times lower body size of the bats. They propose that genes involved in mtDNA replication or oxidative stress reduction should be under stronger selective pressure in long-lived bats than in short-lived rodents, resulting in a lower mitochondrial mutation rate among bats. Further support for this hypothesis comes from the finding that synonymous substitution rates for nine mitochondrial genes, but not rates from six nuclear genes, are negatively correlated with maximum lifespan in mammals (including several bat species) after accounting for body mass and phylogeny (Welch et al., 2008). Additionally, GC content in mtDNA genes is positively correlated with longevity in bats and other long-lived mammals, possibly due to a lower substitution rate resulting from protection against ROS-driven mutagenesis (Lehmann et al., 2008; Min and Hickey, 2008).

While low rates of synonymous substitutions provide indirect evidence of protection from ROS damage to mtDNA in bats, high rates of change in mitochondrial amino acid sequences may indicate direct genetic adaptations associated with long lifespan. Rottenberg (2006) reported a positive correlation between maximum longevity and substitution rate in peptides coded for by ATP6, cytochrome b, and ND3 mitochondrial genes among 72 mammalian genera (including three chiropteran genera). A subsequent study that included 11 bat and 80 mammalian genera found that the relative rate of cytochrome b evolution was positively correlated with the residuals of maximum longevity after factoring out body mass and basal metabolic rate (Rottenberg, 2007a). Given that long-lived bats have relatively high metabolic rates for their body size, Rottenberg (2007a) suggests that accelerated evolutionary rates in mtDNA proteins could facilitate the evolution of long lifespan by producing mutations that reduce ROS generation. Although few bats were included in the analysis, Mossmann and Behl (2008) found a strong negative correlation between cysteine percentage in mtDNA-encoded proteins and maximum longevity in a wide diversity of animal species. Cysteine is particularly susceptible to damage from ROS, and thus a high mutation rate in mitochondrial proteins may facilitate strong purifying selection that removes cysteine in long-lived bat species. Taken together with lower synonymous substitution rates, these results suggest that mtDNA is an active target of ageing-related natural selection in bats.

2.5. Generation of reactive oxygen species and antioxidant activity in bats

Physiological studies in the laboratory have also supported oxidative stress theories of ageing in bats. Little brown bat (Myotis lucifugus, maximum longevity = 34 years) mitochondria generate less than half the amount of hydrogen peroxide per unit of oxygen consumed compared to mitochondria from short-tailed shrews (Blarina brevicauda, maximum longevity = 22 years) or white-footed mouse (Peromyscus leucopus, maximum longevity = 7.9 years); hydrogen peroxide is a highly reactive substance known to cause damage to cells and mitochondria, resulting in progressively degraded metabolic activity (Brunet-Rossinni, 2004). Endothelial cells from the arteries of M. lucifugus also generate fewer ROS, and are more resistant to induced cell death from ROS, than P. leucopus cells (Ungvari et al., 2008). Fibroblast cell lines from M. lucifugus, mentioned above for exhibiting long replicative lifespans in other bat species, also exhibit heightened resistance to hydrogen peroxide- or cadmium-induced apoptosis compared to mouse fibroblasts, but not to UV light, the free radical generator paraquat, or a DNA alkylating agent (Harper et al., 2007). These results provide robust evidence that at least one species of long-lived bat experiences less cellular damage from an important ROS (hydrogen peroxide) than shorter lived rodents, but it is not yet clear whether this finding results from greater mitochondrial efficiency or reduced constitutive activity of oxidoreductases (Ungvari et al., 2008) and whether it occurs among other long-lived bat species.

Molecular adaptations for detoxifying or repairing damage from ROS are predicted to evolve in long-lived species (Zimniak, 2008), but few studies have convincingly documented such phenomena in bats. Wilhelm et al. (2007) examined several potential antioxidant defenses in five South American bat species,
but either did not find significant differences between activity in
bar vs. rodent tissue, or did not perform comparisons between the
same tissue types from bats and rodents. Greater superoxide
dismutase activity in bat vs. rodent liver was one exception,
indicating that bats may exhibit enhanced enzymatic protection
from one ROS (i.e. superoxide). Torpid individuals of the little
yellow-shouldered bat (Sturnira lilium) exhibited greater super-
oxide dismutase and catalase blood levels compared to active
individuals (Wilhelm et al., 2007), which provides intraspecific
support for the positive evolutionary association between hiber-
nation and maximum lifespan among bats (Wilkinson and South,
2002). However, these results should be interpreted with caution
given the small number of individuals (n = 5 active and 3 torpid
individuals) examined by Wilhelm et al. (2007) and Brunet-
Rossiní’s (2004) finding of no difference in superoxide dismutase
activity between little brown bats, mice, and shrews.

3. Longevity research in birds

3.1. Flight, social behavior, and the evolution of lifespan in birds

Birds generally live longer than non-flying mammals of similar
body size (Lindstedt and Calder, 1976; Prinzinger, 1993),
presumably due to lower extrinsic mortality rates that expose
late-acting deleterious mutations to purifying selection (Holmes
and Austad, 1995a; Ricklefs, 1998). As for bats, there are too few
known, independent origins of flight in birds for a phylogenetically
controlled analysis of associations between the evolution of flight
and long lifespan. Several hypotheses have been examined to
explain variation in maximum lifespan with Aves. However,
general explanations for the evolution of long lifespan in birds have
proved elusive, potentially due to flight acting as an energetically
costly constraint on variation in bird lifespan (Ricklefs and Cadena,
2008).

Associations between the evolution of sociality from breeding
apair ancestors and the evolution of long lifespan have recently been
predicted by multiple authors. Ridley et al. (2005) provide
theoretical justifications for this pattern based on (1) increased
likelihood that long-lived subordinates in social species will inherit
ecologically valuable territories, or (2) increased likelihood of
reciprocal altruism among neighboring individuals that protects
the interests of long-lived, local residents. The reciprocal altruism
hypothesis may operate most effectively in environments with
unpredictable resources, and is predicted to create a positive
feedback loop favoring longer lifespan and greater rates of
altruistic behavior (Ridley et al., 2005). Long lifespan has also
been identified as crucial to the evolution of family living in birds
because longevity favors delayed reproduction and large invest-
ments in offspring (Covas and Griesser, 2007). Parrots exhibit a
significant positive association between communal roosting and
longevity after factoring out body size and phylogeny, but this
pattern is statistically dependent on an association between
longevity and diet type (Munshi-South and Wilkinson, 2006).
Blumstein and Moller (2008) found that cooperative parental care
(a proxy of sociality) is not associated with longevity in 257 North
American bird species after factoring out body size, survival rate,
and age at first reproduction, regardless of whether species data or
phylogenetically independent contrasts were analyzed.

3.2. Physiological tradeoffs and longevity in birds

Tradeoffs between energy expenditure and longevity, key
predictions of the disposable soma theory of aging, have not
typically been found in birds. One clear exception is the rate of
embryo growth, which is positively associated with the rate of
ageing-related mortality in birds (Ricklefs, 2006). Age at first
reproduction did not affect subsequent longevity in captive zoo
populations of 12 bird species, although tradeoffs could still
operate on wild populations experiencing resource shortages
(Ricklefs and Cadena, 2007). Longevity of southern African
passerines with insectivorous or nectarivorous diets is twice that of
granivorous species, but Peach et al. (2001) argued that shorter
granivore lifespan is due to their larger clutch size. However,
Munshi-South and Wilkinson (2006) found that granivorous
parrots live longer and have more progeny per year than
frugivorous/nectarivorous or omnivorous parrots.

Maximum lifespan is also positively associated with brain size
in birds, even though brain tissue requires a greater physiological
investment than other somatic cell types. Large-brained species
experience a tradeoff between brain tissue and maximum rates of
population increase, but cooperatively breeding birds with altricial
young overcome this tradeoff through supplemental feeding of
young by non-parent helpers (Iser and Van Schaik, 2009). In
general, long-lived bird species exhibit faster resting metabolic
rates and higher daily and lifetime energy expenditure than
shorter lived bird species (Furness and Speakman, 2008). These
associations are no longer significant after factoring out phylogeny
and body mass covariance, and taken together these results
provide little support for the disposable soma theory in birds.

3.3. Genome size and longevity in birds

Compared to bats, greater research effort has been expended on
interspecific comparative analyses of longevity-extending
mechanisms in birds. One such analysis that currently lacks a
convincing biological mechanism is a positive correlation between
longevity and genome size in birds (independent of family-level
phylogenetic effects, Monaghan and Metcalfe, 2000). This study
has been criticized because a subsequent longevity analysis did not
find any association with genome size (Ricklefs and Scheuerlein,
2001), and a reanalysis of Monaghan and Metcalfe’s (2000) dataset
did not find an effect of avian genome size when factoring out
species-level phylogenetic effects (Morand and Ricklefs, 2001).
However, the original study (Monaghan and Metcalfe, 2000) used
longevity estimates from banded wild birds whereas Ricklefs and
Scheuerlein (2001) did not account for phylogeny and used records
from zoo animals that may not experience substantial extrinsic
mortality (Monaghan and Metcalfe, 2001). A subsequent analysis
that used a much larger bird database did not find an association
between avian longevity and genome size (Gregory, 2002).
However, parrots do exhibit a positive correlation between
genome size and longevity despite high metabolic rates, poten-
tially because adaptations to avoid damage from ROS do not
constrain genome size evolution as greatly as in other species
(Costantini et al., 2008). Parrots are among the longest-lived avian
families (Fig. 2, Munshi-South and Wilkinson, 2006); shorter lived
avian families may exhibit consistently smaller genome sizes than
parrots due to constraints imposed by oxidative DNA damages.

3.4. Latitude, migration and longevity in birds

Other comparative studies of avian longevity have tested
hypotheses derived from rate of living and oxidative stress theories
of ageing. Moller (2007) reported that rates of senescence
decreased with increasing migration distance among 169 avian
species, and increased with latitude as predicted by the slower life
histories of tropical birds. Migration and/or tropical residence may
result in lower exposure to extrinsic mortality if species migrate or
remain in relatively benign environments. Additionally, migratory
species may boost antioxidant levels to combat damage from ROS
generated by high metabolic rates during migration, although such
adaptations have not been described (Moller, 2007). A common-

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3. Telomere length and longevity in birds

Telomeres, repetitive sequences that cap the ends of eukaryotic chromosomes (Pauliny et al., 2006), have recently been identified as sites of interest to avian ageing research due to their role in chromosome stability and cellular replication. Longer telomeres are more likely to prevent chromosomes from fusing together at their ends over time than shorter telomeres (Blackburn, 2000), and thus accumulated oxidative damage to telomeres may act as a constraint on cellular replicative lifespan. Short-lived bird species lose telomeric repeats at a faster rate than long-lived species, but absolute telomere length does not correlate with longevity (Haussmann et al., 2003; Vleck et al., 2003). Residual telomere length predicts longevity in sand martins (Riparia riparia), suggesting that both individuals and species with longer telomeres may exhibit longer lifespans (Pauliny et al., 2006). One exceptionally long-lived species, the storm petrel (Oceanodroma leucorhoa), does not exhibit telomere shortening across its life span, and may be released from the telomere limit to cellular replication (Haussmann et al., 2003). There is considerable variation in telomere length among storm petrel chicks but not adults, suggesting that selection shortens or prolongs telomeres individually from the population (Haussmann and Mauck, 2008). Telomere studies from other long-lived birds have found that telomere shortening preferentially occurs at earlier life stages, and correlates with life history variables such as hatching date and rate of body mass change (Hall et al., 2004). Further experimental work will be needed to determine if telomere shortening is a primary cause of ageing or a consequence of related life history trade-offs. Recent findings from mammals indicate that telomere shortening occurs due to repress of telomerase, potentially as an anti-cancer mechanism that prevents uncontrolled cell proliferation (Gorbunova and Seluanov, 2009). Replicative senescence resulting from low telomerase activity is associated with large body mass, but not shorter lifespan, in mammals (Seluanov et al., 2007). Future research on telomere length–longevity associations in birds should examine whether high telomerase activity explains long lifespan, especially in large-bodied species such as the storm petrel.

4. Improvements and future directions

The recent, substantial progress in understanding the exceptional longevity of the flying vertebrates has been derived from two types of research: (1) comparative, phylogenetically controlled studies that examine associations between maximum lifespan and other biological (primarily life history) variables among dozens or even hundreds of species, and (2) laboratory analysis of genetic and physiological mechanisms (primarily those implicated in oxidative damage theories) that extend longevity in a few non-model or emerging model species. The former studies are currently limited by the quality of the lifespan and life history estimates for birds and bats. Banding studies have provided increasingly long lifespan records for many species (Martino et al., 2006; Podlutsky et al., 2005), but new lifespan estimates for currently unstudied species may require time frames longer than the careers of individual scientists. Development of new methods to age bats and birds could provide data much faster and in larger quantities, although to date research into potential age biomarkers in bats (such as measures of accumulated oxidative damage) is scarce (Brunet-Rossini and Wilkinson, 2009). Telomere length in birds (Vleck et al., 2003) and non-flying mammals (Nagakawa et al., 2004) has recently been shown to underlie predictable decline with age in several species. Although well supported by many studies, this measure still suffers from highly variable, non-linear, or no decline in telomere length with age in some taxa (Juola et al., 2006; Nagakawa et al., 2004), potentially due to telomerase levels that vary with biological characteristics other than age (such as body mass in mammals, Gorbunova and Seluanov, 2009). When validated for individual species, however, telomere shortening may be an important tool for age estimation going forward. Other methods, such as the predictable accumulation of pentosidine or other metabolic byproducts over time in bird tissue (Chaney et al., 2003; Fallon et al., 2006), are promising but have only been validated for a relatively small number of species. Development of these methods will require substantial effort, but the ability to accurately estimate age classes in wild populations will provide information on the ageing process rather than simple correlates of maximum lifespan. Understanding senescent decline in reproduction and other fitness correlates may ultimately lead to a robust integration of ageing research with evolutionary and ecological concepts (Monaghan et al., 2008; Ricklefs, 2008).

Comparative studies are also limited by the quality of phylogenetic estimates that are available to researchers. Methods such as independent contrasts analysis are not suitable for the bias of...
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References

Cutler, D.J., 2008. Phylogenetic inertia, but require highly resolved trees to generate statistical power that resembles simple species comparisons. Phylogenetic supertrees, consensus estimates of previously published trees from multiple datasets, are now available for bats (Jones et al., 2002) and some groups of birds (Jonsson and Fjeldsa, 2006; Thomas et al., 2004). These supertrees will be improved over time as highly resolved molecular phylogenies are generated, and may help ageing researchers to uncover new evolutionary correlates of longevity and strengthen currently known relationships. Comparative approaches, while previously used primarily to examine life history and ecological correlates of longevity (Munshi-South and Wilkinson, 2006; Wilkinson and South, 2002), are now also being used to examine the generality of physiological mechanisms of long lifespan (Holmes and Kristan, 2008). Non-model approaches may lead to blind alleys in the search for general mechanisms if focal species have relatively unique ageing mechanisms. Comparative analyses identify common mechanisms in long-lived species that are not confined to only a few branches of the vertebrate tree.
Finally, greater effort should be devoted to developing new bat and avian laboratory models and utilizing current avian models for ageing research. As has been noted previously (Brunet-Rossinni and Austad, 2004; Brunet-Rossinni and Wilkinson, 2009), many captive bat colonies have been maintained by researchers for long periods of time and could easily be utilized for ageing research. The little brown bat (M. lucifugus) is perhaps the most promising candidate given its ease of attainability in North America, moderate costs in captivity, relatively long lifespan (30 years, Ungvari et al., 2008), availability of genomic sequence and previous research that has identified physiological targets for mechanistic research (Brunet-Rossinni, 2004). Comparisons between domestic chicken, Japanese quail, zebra finch, canary, and budgerigar have already led to useful insights. Additional candidates can be identified in families that exhibit the greatest variation in maximum lifespan relative to body size (i.e. families that contain both short and long-lived species). Among mammals, vespertilio-nid bats exhibit much greater lifespan variation than species in other families (Fig. 2). The Laridae (gulls), Corvidae (crows and jays), and Fringillidae (true finches) provide the best possibilities for comparisons of anti-aging mechanisms in bird species pairs with contrasting lifespans (Fig. 2). Genomic approaches that examine genes under selection in long-lived vs. short-lived related species or long-lived vs. short-lived strains of canaries or zebra finches will lead to discovery of biochemical mechanisms for resistance to oxidative stress. The chicken and zebra finch genomes, plus the little brown bat and several other mammalian genomes, have been or are currently being sequenced. Given the availability of complete mitochondrial and nuclear genomes for many species, and the increasing ease of sequencing entire nuclear transcriptomes of non-model organisms (Ellegren, 2008), comparative functional genomics should play a leading role in future ageing research on birds and bats (Austad, 2005).


Ricklefs, R.E., Cadena, C.D., 2008. Why long-lived species are more likely to be social: the role of local dominance. Behav. Ecol. 16, 358–363.


