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Review

Bats and birds: Exceptional longevity despite high metabolic rates

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ABSTRACT

Bats and birds live substantially longer on average than non-flying mammals of similar body size. The combination of small body size, high metabolic rates, and long lifespan in bats and birds would not seem to support oxidative theories of ageing that view senescence as the gradual accumulation of damage from metabolic byproducts. However, large-scale comparative analyses and laboratory studies on a few emerging model species have identified multiple mechanisms for resisting oxidative damage to mitochondrial DNA and cellular structures in both bats and birds. Here we review these recent findings, and suggest areas in which additional progress on ageing mechanisms can be made using bats and birds as novel systems. New techniques for determining the age of free-living, wild individuals, and robustly supported molecular phylogenies, are under development and will improve the efforts of comparative biologists to identify ecological and evolutionary factors promoting long lifespan. In the laboratory, greater development of emerging laboratory models and comparative functional genomic approaches will be needed to identify the molecular pathways of longevity extension in birds and bats.

1. Introduction

Bats and birds live substantially longer 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 (Fig. 1, Wilkinson 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 gryphus*), 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 supporting evolutionary theories are scarce, but natural "experiments" 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

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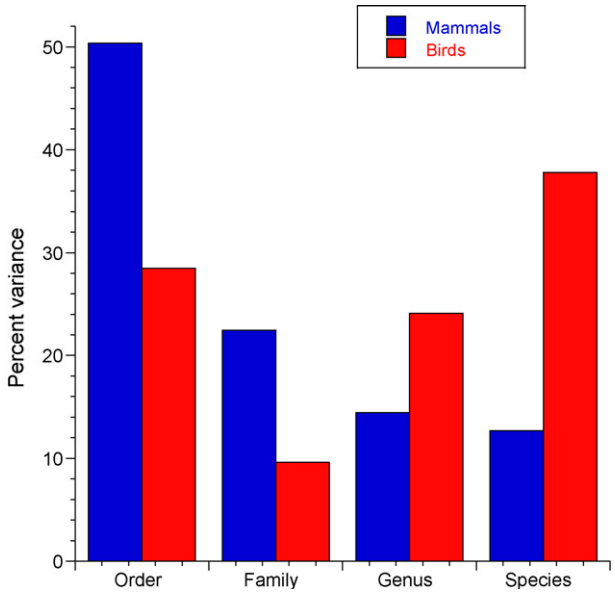

Fig. 1. Percent variance explained in log maximum longevity for 993 species of birds and 977 species of mammals. These values were obtained from a mixed model with log body weight as a covariate and taxonomic level as nested random effects using JMP 5.0.1.2. Body weight and longevity data were taken from the AnAge database (de Magalhaes et al., 2005). The "Species" category refers to the longevity for each species corrected for body weight (i.e. the residuals from the mixed model).

(Kirkwood, 2002). However, investments in brain size (Isler and Van Schaik, 2009), developmental times (Barclay et al., 2004), and most commonly, reproductive rates (Bennett and Owens, 2002; Lack, 1968; Speakman, 2008), are believed to induce tradeoffs with longevity in birds and bats. The tradeoffs operating in these two taxa are not always the same, but the evidence discussed below suggests that these tradeoffs exert significant selective pressure on longevity.

The question of *how* bats and birds live a long time has attracted considerable attention, because of the combination of small body size, long lifespan and high metabolic rate in these groups. These characteristics seemingly contradict "rate of living" theories of ageing that propose a positive correlation between body size and longevity due to lower metabolic rates in larger species (Pearl, 1928). Bats have higher metabolic rates and ultimately use twice as much energy over their lifetimes compared to other mammals (Austad and Fischer, 1991). Hibernation slows down the rate of energy use, and hibernating bats do live 6 years longer on average than non-hibernating bats (Wilkinson and South, 2002). However, non-hibernating bats still live longer than other mammals of the same body size (Brunet-Rossinni and Austad, 2004). Similarly, birds have higher metabolic rates than mammals (Holmes and Austad, 1995b), and long-lived bird species use more energy over their lifetimes (Furness and Speakman, 2008) and have higher field metabolic rates than shorter lived bird species (Moller, 2008).

The patterns above, combined with the failure of recent studies to find evidence of a clear relationship between basal metabolic rate and longevity (de Magalhaes et al., 2007), have prompted researchers to investigate mechanistic explanations for how the flying vertebrates avoid negative physiological effects of their high metabolism. Oxidative theories of ageing predict that reactive oxygen species (ROS) generated by mitochondrial metabolism result in cumulative, irreversible damage leading to senescent decline (Sanz et al., 2006). Bats and birds would seemingly provide little support for this hypothesis given that their high metabolic rates should result in substantial oxidative stress and ageing (Buffenstein et al., 2008). However, below we review recent studies 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-Rossinni 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" (all extant mammals, 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-Rossinni and Austad, 2004), and now a few extremely long-lived *Myotis* species are emerging as ageing research models (Brunet-Rossinni, 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

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phylogenetic 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).

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 independent of body size, reproductive rate, and phylogenetic effects (Wilkinson and South, 2002). Latitude was not an effective predictor of longevity after controlling for hibernation and phylogenetic effects in this analysis, despite a predicted association 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 theory. 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 (Ransome, 1995). Rates of embryo development and postnatal growth also explain a significant proportion of variation in ageing-related mortality among mammals (Ricklefs, 2006; Ricklefs and Scheuerlein, 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 organismal 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 overactive 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 confirm lower 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 mitochondrial 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 (of 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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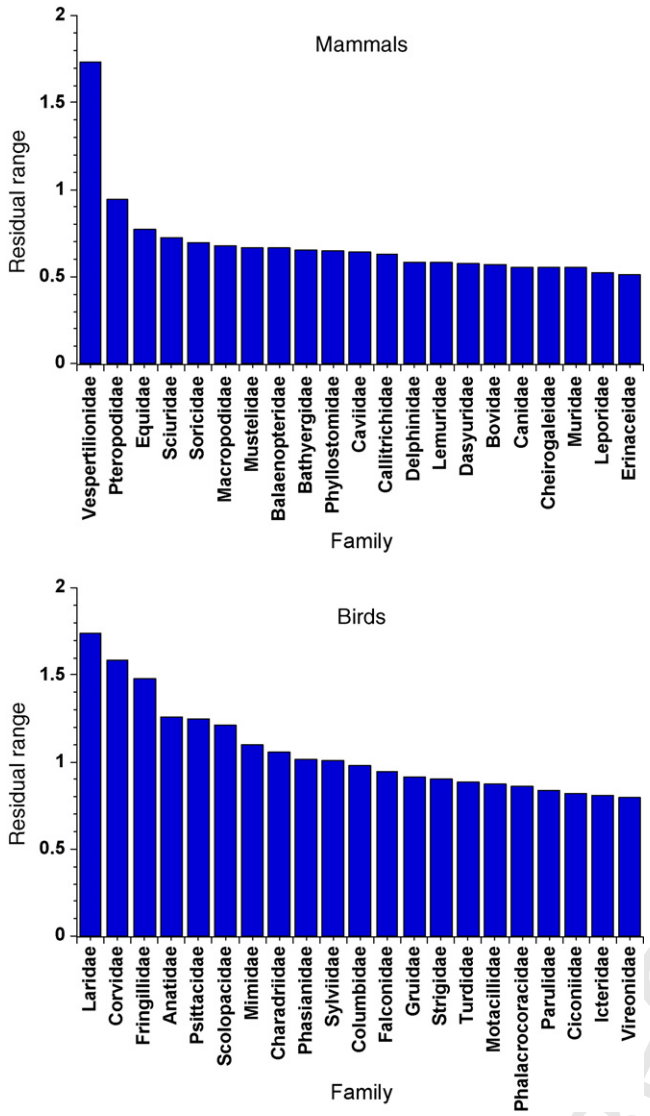

Fig. 2. The top 20 mammal (top) and bird (bottom) families with regard to the range of residual values from the mixed model illustrated in Fig. 1. Thus, those families with the largest ranges have the greatest variation in maximum longevities, relative to their body size.

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, Moosmann 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 mice (*Peromsycus 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,

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but either did not find significant differences between activity in bat 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 superoxide dismutase and catalase blood levels compared to active individuals (Wilhelm et al., 2007), which provides intraspecific support for the positive evolutionary association between hibernation 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-Rossinni'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 pair 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 investments 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 ageing, 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 (Isler 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, potentially 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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garden experiment on nestling stonechats (*Saxicola torquata*) found that resting metabolic rates were lower in individuals from sedentary tropical populations compared to individuals from northern migratory populations (Wikelski et al., 2003). These types of experimental approaches will need to be carried out over the full lifespan of long-lived birds to elucidate relationships between longevity, latitude, migration, and anti-ageing mechanisms.

3.5. 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 removes short-telomere individuals 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 tradeoffs. Recent findings from mammals indicate that telomere shortening occurs due to repression 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.

3.6. Resistance to oxidative damage in birds

Several bird species, many with a long history of captive breeding, have recently been adopted as laboratory models of ageing. Many of these species exhibit less damage from ROS than short-lived laboratory rodents, particularly in reactions involving harmful byproducts from glycoxidation reactions (reviewed in Holmes and Ottinger, 2003). Two cage species that exhibit long maximum lifespans for their body size, budgerigars (*M. undulatus*, 21 years) and canaries (*Serinus canarius*; 24 years), have been particularly useful for comparative laboratory studies of ROS generation and oxidation. Budgerigar cell cultures display enhanced survival compared to Japanese quail (*Coturnix coturnix*, maximum longevity = 11 years) cells when exposed to oxygen or hydrogen peroxide challenges (Ogburn et al., 2001). Budgerigars and canaries also exhibit significantly lower levels of oxidative damage to both proteins and lipids in brain and heart tissue compared to laboratory mice (Herrero and Barja, 1999; Pamplona et al., 2005). Furthermore, heart cells from budgerigars and canaries are less sensitive to lipid peroxidation than cells from pigeons or rodents because cell membranes from the former species have lower fatty acid unsaturation (Pamplona et al., 1999). Saturated membrane fatty acids are potentially a general cellular mechanism for protection against oxidative damage, as many long-lived mammals exhibit a high degree of membrane saturation as well (although bats have not yet been examined, Hulbert, 2008; Hulbert et al., 2006, 2007).

Cellular resistance to oxidative damage in long-lived birds is now a well-documented phenomenon, but research on the biochemical and genetic mechanisms have lagged behind. Rottenberg (2007b) has documented very high rates of cytochrome *b* evolution in long-lived finches (Family Fringillidae, including the laboratory canary) that correlates with mass-corrected longevity. Base-pair substitutions are particularly common in a ubiquinone binding site that may be selected for increased reduction of ubiquinone damage (Rottenberg, 2007b). Future genomics work may generate many more hypothetical targets of selection in the mtDNA genome for enhanced lifespan through suppression of oxidative damage or ROS generation.

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-Rossinni and Wilkinson, 2009). Telomere length in birds (Vleck et al., 2003) and non-flying mammals (Nakagawa et al., 2004) has recently been shown to undergo 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; Nakagawa 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 correct for the bias of

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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 needs in captivity, relatively long life span (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, vespertilionid 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-ageing 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).

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