Aging in the Natural World: Comparative Data Reveal Similar Mortality Patterns Across Primates

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Human senescence patterns—late onset of mortality increase, slow mortality acceleration, and exceptional longevity—are often described as unique in the animal world. Using an individual-based data set from longitudinal studies of wild populations of seven primate species, we show that contrary to assumptions of human uniqueness, human senescence falls within the primate continuum of aging; the tendency for males to have shorter life spans and higher age-specific mortality than females throughout much of adulthood is a common feature in many, but not all, primates; and the aging profiles of primate species do not reflect phylogenetic position. These findings suggest that mortality patterns in primates are shaped by local selective forces rather than phylogenetic history.

Humans are thought to age more slowly than other mammalian taxa ([1], but see (2]) on the basis of their low early-adult mortality, slow mortality acceleration, and long life span. However, it is not known if these human features are unique or are shared with other primates (3, 4). The rapid increase in human life expectancy in the 20th century (5) has increased the proportion of individuals in older age classes (6), raising questions about the flexibility of human aging patterns and the limits of the human life span (e.g., (7–9)). These questions necessitate a deeper understanding of natural aging patterns in other primates, which represent our closest living relatives (10).

Nonhuman primates, like humans, are cognitively and socially complex and behaviorally flexible. However, their long lives and the challenges of continuous, long-term observation make longitudinal demographic data on nonhuman primates uncommon, especially for wild populations ([11]; see also (12)]. We compiled rare data sets from seven species that span the Primate Order: one Indriid (a Madagascan prosimian), two New World monkeys, two Old World monkeys, and two great apes] and carried out a comparative demographic analysis of mortality. Our analyses used data from 226 observation-years of births and deaths on more than 2800 individually recognized male and female primates (13, 14).

We produced species-specific mortality tables for each sex and computed actuarial estimates of age-specific survival and mortality for each of the 2011. I. Virgín, J. R. Waldman, Mutat. Res. 55, 1897 (2004).
22. We acknowledge support of R01ES015447, P42ES007381, ES00260, the Hudson River Foundation, and the Northeast Fisheries Science Center. We thank M. Mattson, K. Sullivan, D. Danila, S. Courtenay, C. Burnett, L. Upchurch, P. Roy, S. Karchner, and A. Nadas for assistance. Accession nos. FJ215751 to FJ215756.

Supporting Online Material
www.sciencemag.org/cgi/content/full/science.1197296/DC1
Materials and Methods
Fig. S1
Tables S1 to S3
References and Notes

2 September 2010; accepted 26 January 2011
Published online 17 February 2011;
10.1126/science.1197296

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the primate populations (15). Analysis of mortality rates revealed the expected pattern for mammals: high infant mortality, followed by a period of low mortality during the juvenile stage, and an extended period of increasing age-specific mortality during mid to late life (Fig. 1). We focused on mid- to late-life demography and modeled initial mortality rate at the start of adulthood for each species, defined in Table 1, through the last age interval for which we had census data. For humans, we used published male and female age-at-death data, from age 15 through 100 years, from the U.S. Department of Health and Human Services life tables (16) and repeated the analyses with a second, independent life table for humans (17), which confirmed our findings.

Understanding flexibility and constraints in the expression and evolution of aging requires a careful analysis of key aging metrics (1, 18, 19). We used a maximum-likelihood framework for estimating two metrics that, together, describe the pattern of senescence for a population: the initial adult mortality rate (IMR, the risk of death on set of adulthood) and the rate of aging (RoA, the rate of increase in the age-specific mortalities with advancing adult age). These aging metrics are often best estimated by fitting the Gompertz model of increasing failure time. We thus tested among competing models for accelerating risk of death with advancing age on the basis of the Gompertz family of models in program WinModest (20) model fitting as described in (21). Our tests included a standard two-parameter Gompertz model and the Gompertz-Makeham and Logistic models. In all but 2 of the 13 species and sex comparisons we examined, the standard two-parameter Gompertz model yielded the best fit to the nonhuman primate data. In the other two cases (sifaka females and capuchin males), the Gompertz-Makeham model was recommended, but because of particular features of those two data sets (see table S1), we proceeded with the standard Gompertz model for males and females of all species. Our model was of the form \( \frac{dN}{dt} = IMR \times e^{RoA \times t} \), where \( u_x \) is the age-specific mortality, i.e., instantaneous mortality probability, at age \( x \) (results in Tables 2 and 3 for females and males, respectively).

We found significantly positive values for RoA in all study species, indicating that mortality rate increased with advancing age [Tables 2 and 3 and fig. S1; see also (22, 23)]. Notably, humans fell along a continuum with the other primate species for both IMR and RoA, which would be indicative of a trade-off between these two parameters (Fig. 2). Instead, our data suggest that they can evolve independently. Humans had low values for both parameters, which explains their exceptional longevity.

For females, we identified four distinct groups of IMR across the eight species (Fig. 2A). All species comparisons were computed on the basis of \( \chi^2 \) tests of pairwise comparisons of the log-likelihood ratio of models with unique versus identical Gompertz parameters (table S2). We identified three significant groups for RoA (Fig. 2A). The coefficient of variation among species for female IMR was 111%, much greater than that for RoA, which was 30%; females of these primate species exhibited a wide range of IMR values.

### Table 1. Summary of study populations. Details about and references for study sites are in (15).

<table>
<thead>
<tr>
<th>Common name</th>
<th>Species</th>
<th>Family</th>
<th>Country</th>
<th>Avg. annual rainfall (mm)*</th>
<th>Life-style</th>
<th>Start year</th>
<th>Sample size</th>
<th>Adult age‡</th>
<th>Predominant dispersing sex</th>
<th>Mean age of first dispersal (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sifaka</td>
<td>Propithecus verreauxi</td>
<td>Indriidae</td>
<td>Madagascar</td>
<td>578</td>
<td>Arboreal</td>
<td>1984</td>
<td>291</td>
<td>19</td>
<td>M</td>
<td>4–5</td>
</tr>
<tr>
<td>Northern Muriqui</td>
<td>Brachyteles hypoxanthus</td>
<td>Atelidae</td>
<td>Brazil</td>
<td>1180</td>
<td>Arboreal</td>
<td>1983</td>
<td>192</td>
<td>212</td>
<td>F</td>
<td>6–7</td>
</tr>
<tr>
<td>Capuchin</td>
<td>Cebus capucinus</td>
<td>Cebidae</td>
<td>Costa Rica</td>
<td>347</td>
<td>Arboreal</td>
<td>1983</td>
<td>98</td>
<td>58</td>
<td>M</td>
<td>4–5</td>
</tr>
<tr>
<td>Yellow baboon</td>
<td>Papio cynocephalus</td>
<td>Cercopithecidae</td>
<td>Kenya</td>
<td>489</td>
<td>Semi-terrestrial</td>
<td>1917</td>
<td>437</td>
<td></td>
<td>M</td>
<td>3–5</td>
</tr>
<tr>
<td>Blue monkey</td>
<td>Cercocepitis mitis</td>
<td>Cercopithecidae</td>
<td>Senegal</td>
<td>1962</td>
<td>Arboreal</td>
<td>1979</td>
<td>128</td>
<td>194</td>
<td>F</td>
<td>7–8</td>
</tr>
<tr>
<td>Chimpanzee</td>
<td>Pan troglodytes</td>
<td>Hominoidea</td>
<td>Tanzania</td>
<td>1330</td>
<td>Semi-terrestrial</td>
<td>1963</td>
<td>122</td>
<td>144</td>
<td>M</td>
<td>15–16</td>
</tr>
<tr>
<td>Gorilla</td>
<td>Gorilla beringei</td>
<td>Hominoidea</td>
<td>Rwanda</td>
<td>1358</td>
<td>Terrestrial</td>
<td>1967</td>
<td>128</td>
<td>120</td>
<td>M</td>
<td>15–16</td>
</tr>
</tbody>
</table>

*Average annual rainfall for each study, representative of the study years. Rainfall data for gorillas were collected by the Rwandan Government Meteorological Office at a location several kilometers from the field site and at a lower elevation. Rainfall data for other studies were collected at the study site. †Year study was established. Latest census date for all populations in these analyses was December 2008. ‡Mean age class at which adulthood is attained for each sex. Male onset of adult stage was defined as mean age of likely first reproduction (using physical criteria such as copulation with ejaculation, behavioral criteria such as the onset of mate guarding behavior, or genetically confirmed paternity). Female onset of adult stage is defined as the mean age of first live birth. ¶Twelve percent of female capuchins disperse. The average age interval of dispersing capuchin females is 6 to 7 years. *Both sexes disperse in gorillas.

### Table 2. Gompertz estimates of female mortality parameters and life-span summary statistics. Adult age interval is the age interval containing the mean age of first live birth; IMR (= Gompertz a) is the Gompertz estimate of instantaneous mortality rate at the first adult age interval [with its 95% confidence interval (CI)]; RoA (= Gompertz b) is the adult rate of aging estimated with Gompertz acceleration (with its 95% CI); MRDT is the mortality rate doubling time during adulthood; Oldest age reached is the age class of the oldest observed individual; Median age is the 50% survival age with its range.

<table>
<thead>
<tr>
<th>Species</th>
<th>Adult age interval</th>
<th>IMR (year)</th>
<th>95% CI</th>
<th>RoA</th>
<th>95% CI</th>
<th>MRDT (no. of years)</th>
<th>Oldest age reached (years)</th>
<th>Median age</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sifaka</td>
<td>6–7</td>
<td>0.0278</td>
<td>[0.019, 0.0410]</td>
<td>0.0991</td>
<td>[0.072, 0.136]</td>
<td>7.0</td>
<td>31–32</td>
<td>10</td>
<td>[9, 12]</td>
</tr>
<tr>
<td>Muriqui</td>
<td>8–9</td>
<td>0.00170</td>
<td>[0.00042, 0.00685]</td>
<td>0.129</td>
<td>[0.0722, 0.230]</td>
<td>5.4</td>
<td>40–41</td>
<td>25</td>
<td>[18, 33]</td>
</tr>
<tr>
<td>Capuchin</td>
<td>6–7</td>
<td>0.0415</td>
<td>[0.0150, 0.114]</td>
<td>0.165</td>
<td>[0.055, 0.494]</td>
<td>4.2</td>
<td>26–27</td>
<td>11</td>
<td>[10, 13]</td>
</tr>
<tr>
<td>Baboon</td>
<td>5–6</td>
<td>0.0285</td>
<td>[0.020, 0.040]</td>
<td>0.123</td>
<td>[0.0926, 0.165]</td>
<td>5.6</td>
<td>27–28</td>
<td>8</td>
<td>[7, 9]</td>
</tr>
<tr>
<td>Blue monkey</td>
<td>7–8</td>
<td>0.00723</td>
<td>[0.00367, 0.0143]</td>
<td>0.160</td>
<td>[0.123, 0.209]</td>
<td>4.3</td>
<td>33–34</td>
<td>18</td>
<td>[17, 22]</td>
</tr>
<tr>
<td>Chimpanzee</td>
<td>14–15</td>
<td>0.00744</td>
<td>[0.0038, 0.0156]</td>
<td>0.0992</td>
<td>[0.070, 0.140]</td>
<td>7.0</td>
<td>53–54</td>
<td>16</td>
<td>[10, 25]</td>
</tr>
<tr>
<td>Gorilla</td>
<td>9–10</td>
<td>0.000028</td>
<td>[0.00004, 0.0021]</td>
<td>0.211</td>
<td>[0.148, 0.303]</td>
<td>3.3</td>
<td>44–43</td>
<td>33</td>
<td>[31, 35]</td>
</tr>
<tr>
<td>Human‡</td>
<td>0.000009</td>
<td>[0.00008, 0.00009]</td>
<td>0.0961</td>
<td>[0.0956, 0.0967]</td>
<td>7.2</td>
<td>100+</td>
<td>83.5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Oldest individual with known date of birth. †Truncated at 18–19 for mortality analysis because of relatively smaller sample sizes of deaths and transitions in later age classes. ‡Data from (16), modeled beginning at age interval 15–16 years through 99–100 years.
whereas RoA was less variable (equality of variance test: $F_{1,7} = 6.44, P = 0.02$). Moreover, all combinations of high and low IMR with high and low RoA were found in the females of the seven nonhuman species. For example, female chimpanzees were characterized by both low IMR and low RoA, whereas female sifaka exhibited high IMR but relatively low RoA. In contrast, female gorillas had low IMR and high RoA, while female capuchins exhibited both high IMR and high RoA. The RoA for human females was statistically indistinguishable from that of the four other slowly aging female primates (Fig. 2A and table S2). Human females had one of the two lowest IMRs (statistically indistinguishable from gorilla; Fig. 2A and table S2), but this trait is arguably more reflective of environmental plasticity than is RoA (24). This similarity between humans and nonhuman primates indicates that aging in humans is not evolutionarily divergent from that in other primate species [see also (1)]. This similarity is particularly noteworthy given that our human-nonhuman comparison was a conservative one, in that it used data from modern human populations rather than hunter-gatherers or historical populations [which might resemble wild nonhuman primates more than modern humans do (23, 25)].

Among males, the coefficient of variation for IMR was 107%, much greater than the coefficient of variation in RoA, which was 40% (equality of variance $F_{6,6} = 26.0, P = 0.001$). Males and females showed similar variation in IMR, but males showed greater variation than females in RoA. Males exhibited fewer combinations of IMR and RoA than females: Baboon, sifaka, and capuchin males were characterized by high IMR and high RoA, whereas gorilla, muriqui, and chimpanzee males had intermediate IMR and intermediate RoA. Like females, males exhibited four significant groupings of IMR and three significant groupings of RoA (Fig. 2B and table S2). RoA in human males, unlike in human females, was significantly lower than the next closest value, that of chimpanzees, and the IMR for human males was relatively even lower (Fig. 2B).

Males of monogamous animal species tend to age at rates similar to those of females, whereas males of polygynous species exhibit increased aging rates relative to females (26, 27). All of the nonhuman primate species studied here are polygynous (or more accurately polygynandrous, as multiple mating is exhibited by females as well as males). Further, six of the seven experience relatively intense male-male competition for access to mates [see (28) for genus-level data on Cebus, Cercopithecus, Gorilla, Papio, and Pan; (29) for data on Propithecus]. The exception is the muriqui, a sexually monomorphic species in which male-male competition for access to females appears to be absent (30). In the species with relatively

Table 3. Gompertz estimates of male mortality and life-span summary statistics. Adult age interval is the mean age class of likely first reproduction. IMR ($\approx$ Gompertz $a$) is the instantaneous mortality at adulthood (with its 95%CI); RoA ($\approx$ Gompertz $b$) is the rate of aging estimated with Gompertz acceleration (with its 95% CI); MRDT is the mortality rate doubling time during adulthood; Oldest age reached is the age class of the oldest observed individual; Median age is the 50% survival age with its range.

<table>
<thead>
<tr>
<th>Species</th>
<th>Adult age interval (years)</th>
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<th>95% CI</th>
<th>RoA</th>
<th>95% CI</th>
<th>MRDT (no. of years)</th>
<th>Oldest age reached (years)</th>
<th>Median age</th>
<th>[Range]</th>
</tr>
</thead>
<tbody>
<tr>
<td>†Sifaka</td>
<td>5–6</td>
<td>0.0201</td>
<td>[0.0140, 0.0290]</td>
<td>0.186</td>
<td>[0.155, 0.222]</td>
<td>3.73</td>
<td>26–27</td>
<td>19–20</td>
<td>[12, 13]</td>
</tr>
<tr>
<td>Muriqui</td>
<td>6–7</td>
<td>0.00187</td>
<td>[0.00044, 0.00784]</td>
<td>0.148</td>
<td>[0.0820, 0.266]</td>
<td>4.70</td>
<td>33–34</td>
<td>26–27</td>
<td>[22, 27]</td>
</tr>
<tr>
<td>Capuchin</td>
<td>6–7</td>
<td>0.010</td>
<td>[0.0027, 0.036]</td>
<td>0.294</td>
<td>[0.159, 0.542]</td>
<td>2.36</td>
<td>24–25†</td>
<td>12–13</td>
<td>4 [3, 5]</td>
</tr>
<tr>
<td>Baboon†‡</td>
<td>7–8</td>
<td>0.0371</td>
<td>[0.0266, 0.0517]</td>
<td>0.213</td>
<td>[0.177, 0.256]</td>
<td>3.26</td>
<td>24–25</td>
<td>22–23</td>
<td>10 [9, 11]</td>
</tr>
<tr>
<td>Blue monkey</td>
<td>8–9</td>
<td>No est.</td>
<td>No est.</td>
<td></td>
<td></td>
<td></td>
<td>19–20</td>
<td>19–20</td>
<td>No est.</td>
</tr>
<tr>
<td>Chimpanzee</td>
<td>14–15</td>
<td>0.00787</td>
<td>[0.00346, 0.0117]</td>
<td>0.137</td>
<td>[0.0980, 0.190]</td>
<td>5.07</td>
<td>43–44</td>
<td>40–41</td>
<td>11 [9, 14]</td>
</tr>
<tr>
<td>Gorilla</td>
<td>15–16</td>
<td>0.00594</td>
<td>[0.00139, 0.0254]</td>
<td>0.182</td>
<td>[0.106, 0.313]</td>
<td>3.81</td>
<td>38–39</td>
<td>34–35</td>
<td>23 [22, 29]</td>
</tr>
<tr>
<td>Human†</td>
<td>0.00024</td>
<td>[0.00023, 0.00025]</td>
<td>0.086</td>
<td>[0.0854, 0.0863]</td>
<td>8.07</td>
<td>100+</td>
<td>100+</td>
<td>79.5</td>
<td></td>
</tr>
</tbody>
</table>

*Oldest individual with known date of birth. †Distribution of deaths imputed from onset of adulthood with age structure. See methods in the Supporting Online Material (SOM). ‡Age structure corrected for population growth. See methods in the SOM. ¶Truncated at 18–19 for mortality analysis because of relatively smaller samples sizes of deaths and transitions in later age classes. ††Data from (26), modeled beginning at age interval 15–16 years through 99–100 years.

Fig. 2. **IMR versus RoA** for (A) females and (B) males. Phylogenetic relationships among species are shown in (C). Letters over bars denote statistically significant groupings. [Female IMR: human, gorilla (A) ≤ gorilla, muriqui (B) < blue monkey, chimpanzee (C) < sifaka, baboon, capuchin (D);] female RoA: human, chimpanzee, sifaka, baboon, muriqui (A) ≤ muriqui, blue monkey, capuchin (B) ≤ blue monkey, capuchin, gorilla (C); male IMR: human (A) < muriqui, gorilla, chimpanzee, capuchin (B) ≤ capuchin, sifaka (C) < baboon (D); male RoA: human (A) < chimpanzee, muriqui, gorilla, sifaka (B) ≤ muriqui, gorilla, sifaka, baboon, capuchin (C).] See table S2 for tests of pairwise comparisons of IMR and RoA.
intense male-male competition for mates, males and females showed significant differences in either IMR or RoA, and male life span was shorter than female life span (baboons, sihaka, gorillas, chimpanzees, and capuchins; we lacked mortality data for male blue monkeys; see Fig. 1 and table S3). In contrast, male and female muriquis mortality data for male blue monkeys; see Fig. 1 and chimpanzees, and capuchins; we lacked mor-

than female life span (baboons, sifaka, gorillas, also (26) competitive environment, not just multiple mat-

in muriqui aging patterns, combined with the ob-

Figure 2C and fig. S1). This related species of primates to exhibit similar aging evolutions to phylogeny (Fig. 2C and fig. S1). This related species of primates to exhibit similar aging related species of primates to exhibit similar aging patterns. Instead, the species rankings of IMR and RoA in males and females showed no relation-

ship to phylogeny (Fig. 2C and fig. S1). This implies that the study species have not been constrained phylogenetically to high or low aging rates, and have the flexibility to respond to evo-

lutionary forces at the species level or potentially even the local population level. Furthermore, within-species comparisons of baboons (31), chimpanzees (23, 32), and humans (23, 25) all support the view that both IMR and RoA can vary substantially among populations within a species. Notably, in all three species, populations existing in more demanding habitats, without benefit of modern medical intervention (e.g., hunter-gatherer humans and wild as opposed to captive primates), exhibit higher IMR and, for both chimpanzees and humans, higher RoA. That is, aging appears to be both evolutionarily labile and phenotypically plastic. The slowing of aging-related disease un-

der dietary restriction (33) is further evidence of the flexibility of aging rates in primates.

We examined our data for the existence of mortality plateaus (34), a subject of much recent interest in the aging literature, but none of the age-specific mortality relationships in our non-human primate analyses demonstrated the type of leveling off that has been shown in human and fly data sets [e.g., (35)]. Whether additional long-term data from natural primate populations will demonstrate a generalized mortality deceleration in old age remains an open question that should motivate future comparative analyses of aging in other natural populations.

References and Notes

Positive Supercoiling of Mitotic DNA Drives Decatenation by Topoisomerase II in Eukaryotes

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DNA topoisomerase II completely removes DNA intertwining, or catenation, between sister chromatids before they are segregated during cell division. How this occurs throughout the genome is poorly understood. We demonstrate that in yeast, centromeric plasmids undergo a dramatic change in their topology as the cells pass through mitosis. This change is characterized by positive supercoiling of the DNA and requires mitotic spindles and the condensin factor Smc2. When mitotic positive supercoiling occurs on decatenated DNA, it is rapidly relaxed by topoisomerase II. However, when positive supercoiling takes place in catenated plasmid, topoisomerase II activity is directed toward decatenation of the molecules before relaxation. Thus, a topological change on DNA drives topoisomerase II to decatenate molecules during mitosis, potentially driving the full decatenation of the genome.

In eukaryotes, most topological links between the DNA strands are removed during DNA replication by topoisomerases I and II (fig. S1A) (1). However, many links are converted into double-stranded DNA intertwines or catenanes during the completion of replication (2, 3). These can only be resolved by topoisomerase II (fig. S1) (4).

Passage through mitosis is required for complete decatenation, because topoisomerase II activity is essential during mitosis as late as anaphase (fig. S1B) (4–6). Because mitotic spindles are required to complete decatenation, it is assumed to occur only after chromosome segregation during anaphase (4). However, sister chromatids appear to be fully decatenated before their physical separa-

tion by spindles (7, 8). Potential alternate mech-

anisms, in which decatenation is promoted by supercoiling, have been proposed in prokaryotes (9–11). These mechanisms prompted us to study whether mitotic changes to DNA topology help drive decatenation in eukaryotes.

References