



# Host Longevity and Parasite Species Richness in Mammals

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1 **Host longevity and parasite species richness in mammals**

2

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17 **Abstract**

18 Hosts and parasites co-evolve, with each lineage exerting selective pressures on the other. Thus,  
19 parasites may influence host life-history characteristics, such as longevity, and simultaneously  
20 host life-history may influence parasite diversity. If parasite burden causes increased mortality,  
21 we expect a negative association between host longevity and parasite species richness.

22 Alternatively, if long-lived species represent a more stable environment for parasite  
23 establishment, host longevity and parasite species richness may show a positive association. We  
24 tested these two opposing predictions in carnivores, primates and terrestrial ungulates using  
25 phylogenetic comparative methods and controlling for the potentially confounding effects of  
26 sampling effort and body mass. We also tested whether increased host longevity is associated  
27 with increased immunity, using white blood cell counts as a proxy for immune investment. Our  
28 analyses revealed weak relationships between parasite species richness and longevity. We found  
29 a significant negative relationship between longevity and parasite species richness for ungulates,  
30 but no significant associations in carnivores or primates. We also found no evidence for a  
31 relationship between immune investment and host longevity in any of our three groups. Our  
32 results suggest that greater parasite burden is linked to higher host mortality in ungulates. Thus,  
33 shorter-lived ungulates may be more vulnerable to disease outbreaks, which has implications for  
34 ungulate conservation, and may be applicable to other short-lived mammals.

35

36 **Keywords:** Artiodactyla, Carnivora, lifespan, Perissodactyla, phylogenetic generalized least  
37 squares.

38

## 39 **Introduction**

40           Understanding parasite infections in wild animals is of great importance. For example,  
41 infectious diseases are threatening various species (e.g., amphibians and Tasmanian devils;  
42 [1,2]), while biodiversity itself may influence the prevalence of parasites in ecological  
43 communities [3,4]. Additionally, we share approximately 60% of our infectious diseases with  
44 animals [5] and many recent human pandemics originated in wildlife, including HIV and SARS  
45 [6,7]. Identifying the host characteristics that support multiple parasites is therefore critically  
46 important for human health and the conservation of biodiversity.

47           Mammals are infected by a wide variety of parasites, ranging from microscopic viruses  
48 and bacteria to macroscopic tapeworms, flukes and biting arthropods [4,8]. These parasites are  
49 also diverse in terms of their transmission modes (e.g., sexual, vertical, vector-borne, airborne,  
50 and fecal-oral) and life cycles (e.g., direct or via one or more intermediate hosts). The diseases  
51 caused by these infectious agents can have profound fitness effects on individual hosts, resulting  
52 in selection for anti-parasite behaviors [9], immune defenses [10], and changes in life-history  
53 features such as birth weight [11]. Despite a great deal of study, however, it remains unclear how  
54 parasites influence many aspects of host biology, including basic life-history parameters.

55           Host longevity is a life-history parameter that is expected to covary with parasite  
56 infection [12]. A number of comparative studies have investigated the relationship between these  
57 variables in mammals but results have been mixed; some studies found limited evidence that  
58 longer-lived mammals had more parasites [13], some studies found that longer-lived mammals  
59 had fewer parasites [12,14], while other authors failed to find evidence for an association [15-  
60 18]. Additionally the relationship between parasites and host longevity is unclear because  
61 causality may be bidirectional, with parasites influencing measures of longevity, while longevity

62 simultaneously influences parasite success. We describe these two competing hypotheses below  
63 using parasite species richness as our measure of parasite burden.

64 Parasites often cause negative fitness effects on hosts and, while some behavioral  
65 defenses may help species avoid infection, a substantial level of unavoidable infection (and  
66 therefore mortality) is likely to exist in wild populations [12]. Thus, similar to the effects of  
67 unavoidable mortality through predation, higher parasite pressure may favor a shorter lifespan  
68 (and faster reproduction). This should result in a negative correlation between parasite burden  
69 and host longevity, with higher parasite species richness in shorter-lived species.

70 Conversely, host longevity may influence parasite burden through epidemiological  
71 processes, predicting a positive association between longevity and parasite richness. Increases in  
72 host background mortality should make it more difficult for parasites to establish in host  
73 populations because the death of a host also results in the death of its parasites. Given that a  
74 higher background mortality rate is equivalent to a shorter longevity, it is reasonable to expect  
75 that more parasites will meet the conditions for establishment (i.e.,  $R_0 > 1$ ; [19]) in hosts that live  
76 longer. Based on these basic epidemiological principles, we expect to find a positive correlation  
77 between parasite species richness and host longevity, with highest parasite species richness in  
78 long-lived species [20]. Increased longevity may also lead to greater parasite species richness  
79 because a longer-lived individual is likely to be exposed to more parasites throughout its lifetime  
80 [12,21]. Although not all of these infections will be retained throughout the life of an individual,  
81 sampling across individuals should reveal more species of parasite in longer-lived host species.

82 Host immune investment may provide crucial insights into the relationship between host  
83 longevity and parasite burden. Immune investment is costly, so one might expect a trade-off  
84 between immune investment and investment in other life-history traits such as growth and

85 reproduction [22]. A heavily parasitized host may achieve the same fitness by either (a) investing  
86 in immunity and reproducing over a longer lifespan, or (b) investing in rapid reproduction to the  
87 detriment of immune investment, leading to increased mortality and a shorter lifespan. Thus,  
88 immune investment may either decrease or increase with parasite burden. In addition, the  
89 optimal life-history strategy may depend on the kind of infections to which the host is exposed:  
90 chronic infections may select for increased immune investment and a longer lifespan, whereas  
91 acute infections with high mortality rates may select for a faster life-history, reduced immune  
92 investment and shorter longevities.

93         Here, we investigate the relationship between maximum longevity and parasite species  
94 richness in mammals using data from terrestrial Carnivora, Primates and terrestrial ungulates  
95 (Artiodactyla and Perissodactyla). Our study extends previous studies and aims to resolve  
96 previously conflicting findings by more than doubling the number of host species in the  
97 comparative dataset. Compared to previous research, we also use more advanced phylogenetic  
98 methods, including methods to estimate and take into account phylogenetic signal in the data,  
99 while rigorously controlling for the potentially confounding effects of body mass and sampling  
100 effort (for estimates of both parasite species richness and maximum longevity). We also  
101 investigate the relationships among immune system investment, maximum longevity and parasite  
102 species richness.

103

## 104 **Materials and methods**

### 105 **DATA**

106         We used parasite species richness (PSR) data from the *Global Mammal Parasite*  
107 *Database* (GMPD; [23]). This database contains host-parasite records taken from the literature

108 since 1929, and continues to be updated as new papers are published. All records come from  
109 wild host populations and represent natural infections. To date, the database contains over 20,000  
110 host-parasite records from over 500 host species and over 2100 parasite species, including both  
111 macro- (i.e., helminthes) and micro- (i.e., viruses, bacteria, protozoa and fungi) and ecto-  
112 parasites (i.e., arthropods). The GMPD contains information on parasites found in wild  
113 Carnivora, Primates and terrestrial ungulates (Artiodactyla and Perissodactyla); thus we  
114 restricted our analyses to these groups. We excluded the marine Carnivora (Phocidae, Otariidae,  
115 Odobenidae) because aquatic environments may result in differences among parasite  
116 transmission patterns, immune investment and life-history features (e.g., aquatic carnivores have  
117 higher white blood cell counts than terrestrial carnivores; [24]).

118 We estimated total parasite species richness (PSR) for each host species, using the  
119 taxonomy of Wilson and Reeder [25], and also estimated PSR for macro- (i.e., helminthes) and  
120 micro-parasites (i.e., viruses, bacteria, protozoa and fungi) separately ( $PSR_{\text{macro}}$  and  $PSR_{\text{micro}}$ ).  
121 For some host-parasite records, parasites were identified only to the genus-level. To use as much  
122 data as possible, we included these parasites in estimates of PSR provided that no other members  
123 of the genus were recorded for the host species. In total, our PSR values used 2174 species of  
124 parasite (994 macro-, 779 micro- and 401 ecto-parasites).

125 For each host species, we then collated data on maximum longevity (months) from the  
126 PanTHERIA and AnAge databases [26,27], Walker's Mammal Species of the World [28], and a  
127 few additional sources (Supporting Information S1). We used a mammal supertree for all  
128 phylogenetic analyses [29,30].

129 Both PSR and longevity show correlations with body mass in some mammals [e.g.,  
130 13,14,31-33]. Thus, any correlation between longevity and PSR could be the result of

131 covariation with body mass. To address this possibility, we included body mass in our models  
132 (see below). We collated data on adult body mass (g) from PanTHERIA and AnAge [26,27],  
133 Walker's Mammal Species of the World [28], and a few additional sources (Supporting  
134 Information S1). We note that other variables also covary with taxonomic subsets of PSR in  
135 some mammals, including social group size and geographic range size (e.g., [13]). However,  
136 when we performed phylogenetic generalized least squares models (see below) controlling for  
137 body mass and sampling effort, these variables were not correlated with PSR for carnivores or  
138 ungulates (Table S1). We found weak significant positive correlations between PSR and both  
139 social group size and geographic range size for primates (Table S1). However, these significant  
140 associations disappear in full models (Table S2). To simplify our results, we therefore do not  
141 include social group size or geographic range size in the statistical models investigated here.

142 PSR and life-history data are also sensitive to sampling effort: host species which have  
143 been thoroughly sampled for parasites may appear to have higher PSR values than those which  
144 have been less well-sampled [13,34]. Similarly, a well-studied host species may appear to have  
145 higher maximum longevity than its less-well studied counterparts [33,35]. To control for these  
146 sampling biases we included a measure of sampling effort (citation count) for each host species.  
147 We defined this as the number of ISI Web of Knowledge (<http://wokinfo.com/>) references where  
148 the Latin binomial of the species appeared in either the title or topic fields. Where the species  
149 binomial had changed between the 1993 and 2005 taxonomies [25,36] we summed the number  
150 of citations for the species names from both taxonomies.

151 For analyses of host immune investment, we extracted mean white blood cell counts  
152 (WBC; expressed as the number of cells in  $10^{-9}$  liters of blood) from the International Species  
153 Information System (ISIS) database [37]. We used WBC as a proxy for host immune investment



154 because white blood cells represent the first line of defense against pathogens, they are probably  
155 costly to produce, and WBC is used by both physicians and wildlife ecologists to gauge the  
156 health of individuals [e.g., 38]. Within primates, for example, significantly higher WBC are  
157 observed in diseased individuals [39]. Other components of the vertebrate immune system, such  
158 as spleen size and the diversity of major histocompatibility complex (MHC) genes are also likely  
159 to be important indicators of immune investment; however, these data are not available for most  
160 of our species.

161         The ISIS database contains physiological data from putatively healthy captive individuals  
162 only. This helps to remove the confounding effects of differences in health or stress levels on  
163 physiology. Ideally, we would use data from wild individuals with information on their health  
164 and stress levels. However, these data are rare for wild populations making the ISIS database the  
165 best alternative available. Although WBC may vary between sexes and among age classes [40],  
166 most ISIS records do not separate WBC records into separate sexes or age classes for all species  
167 in our dataset. Thus, we used WBC from all ages and sexes combined to get the largest sample  
168 size possible.

169         In total we have data on PSR, longevity, body mass and citation counts for 361 species  
170 (132 carnivores, 128 primates and 101 ungulates). We also have white blood cell counts for 219  
171 of these species (64 carnivores, 81 primates and 74 ungulates). The data are available in  
172 Supporting Information S2.

173

## 174 **ANALYSES**

175 We found that natural-log transformed data improved model diagnostics, resulting in a better  
176 distribution of residuals from the regression model. Thus, all variables were ln-transformed prior

177 to analysis. Before fitting multivariate models, we also checked the predictors for collinearity  
178 (following the method of [41]) because it can lead to unreliable model parameter estimates.  
179 Variance inflation factors (VIF) were less than three, indicating acceptable levels of collinearity  
180 [41].

181 Species in comparative analyses are related to one another and thus may share similarities  
182 because they inherited them from a common ancestor, rather than through independent evolution  
183 [42,43]. To deal with the potential statistical non-independence of the interspecific data, we used  
184 phylogenetic generalized least squares models (PGLS). PGLS is based on the usual GLS model  
185 except that the phylogenetic dependence of the data is incorporated into structure of the error  
186 term [44-46]. This error term can be constructed in a number of ways. Here it consists of a  
187 matrix of expected trait covariances calculated using the phylogeny and the maximum likelihood  
188 (ML) estimate of  $\lambda$ . The parameter  $\lambda$  is a multiplier of the off-diagonal elements of a  
189 phylogenetic variance-covariance matrix that best fits the data, and varies between  $\lambda = 1$ , where  
190 the data are structured according to a Brownian motion model of trait evolution, and  $\lambda = 0$ , where  
191 the data show no phylogenetic structure and the analysis reduces down to a non-phylogenetic  
192 OLS analysis [45,47]. For each regression,  $\lambda$  is estimated for the residual error term [48], along  
193 with the other regression parameters so regressions are carried out whilst controlling for the  
194 actual degree of phylogenetic non-independence present. For interest, we report the phylogenetic  
195 signal ( $\lambda$ ) in individual variables in Table S3, however we note that this does not provide any  
196 justification for using PGLS or non-phylogenetic methods [48].

197 We used R v.2.13.0 [49] to run all of the analyses. Specifically, we used the function  
198 `pgls` in the package `caper` [50] to fit the following model for carnivores, primates and ungulates  
199 separately:

200 
$$\ln(\text{PSR}) = f(\ln(\text{longevity}) + \ln(\text{body mass}) + \ln(\text{citation count})) \quad (1)$$

201 We focus on these three clades separately because each offers sufficient sample sizes to test the  
202 hypotheses, and when the data are combined, we found that patterns were driven by a strong  
203 positive relationship in only one of the clades (ungulates). To test relationships among longevity,  
204 immune system investment and parasite species richness, we also used PGLS to fit the following  
205 model for carnivores, primates and ungulates separately:

206 
$$\ln(\text{WBC}) = f(\ln(\text{PSR}) + \ln(\text{longevity}) + \ln(\text{body mass}) + \ln(\text{citation count})) \quad (2)$$

207 We predict that different types of parasites will affect host longevity in different ways;  
208 specifically we expect chronic infections to select for longer lifespans and increased immune  
209 investment, and acute infections to select for shorter lifespans and decreased immune investment.  
210 Therefore we also fitted each model using PSR for macro- and micro-parasites separately,  
211 because macroparasites are generally thought to cause chronic infections and microparasites to  
212 cause acute infections [51]. Obviously there are exceptions to this generalization; however, data  
213 on the type of infection was unavailable for most of our parasite species so this was the best  
214 approximation available.

215 The statistical performance of PGLS can be strongly influenced by outliers, especially  
216 where large evolutionary changes have occurred on short branches. This can result in points with  
217 very high leverage that could affect parameter estimates and increase the error rates of the  
218 regressions. To avoid this, we repeated our regressions after removing any points with a  
219 studentized residual exceeding  $\pm 3$  [52]. However, results were qualitatively similar, and so we  
220 only report results from analyses in which all the data were used.

221 We also used phylogenetic analysis of variance (ANOVA) to investigate differences among  
222 our three host groups in their PSR, longevity, body mass, and WBC values. Phylogenetic

223 ANOVAs perform a standard ANOVA but determine the significance value of the F statistic by  
224 comparing the observed value to a null distribution obtained by simulating new sets of data  
225 under a Brownian motion model along the phylogeny [53]. We fit these using the function  
226 `phy.anova` in the package `geiger` [54].

227

## 228 **Results**

229 We found a significant negative relationship between maximum longevity and total  
230 parasite species richness (PSR) for ungulates but not for carnivores or primates (Table 1). When  
231 we investigated macro- and micro-parasites separately, only ungulates showed a significant  
232 negative relationship between maximum longevity and microparasite species richness (PSR<sub>micro</sub>;  
233 Table 1). The PSR values of the three mammalian groups were not statistically different,  
234 suggesting that variation in the results was not due simply to clade specific differences in total  
235 parasite burden, or differences in the numbers of macro- or micro-parasites infecting each clade  
236 (Table 2). However, we did find significant differences in longevity among groups; primates  
237 have the longest lifespans, followed by ungulates and carnivores (Table 2).

238 In each of the models in Table 1, citation count (our measure of sampling effort) was  
239 highly significantly positively correlated with PSR. This confirms our suggestion that better  
240 studied mammals may appear to have more parasites than less well-studied species. If citation  
241 count is not included in the models, all groups except the ungulates show a significant positive  
242 association between longevity and PSR, although AIC values increase substantially (carnivores:  
243 longevity slope =  $1.010 \pm 0.400$ ,  $t_{129} = 2.741$ ,  $p = 0.007$ , AIC = 460.5 [AIC with citation count =  
244 367.2]; primates: longevity slope =  $1.048 \pm 0.354$ ,  $t_{124} = 2.958$ ,  $p = 0.004$ , AIC = 384.3 [AIC  
245 with citation count = 325.7]; ungulates: longevity slope =  $-0.138 \pm 0.442$ ,  $t_{98} = -0.313$ ,  $p = 0.755$ ,

246 AIC = 369.2 [AIC with citation count = 345.4]). This would completely change our conclusions  
247 and thus highlights the importance of controlling for sampling effort in our models. Although  
248 citation count explains a great deal of the variation in PSR, models including citation count alone  
249 have much higher AIC values than our full models including body size and longevity (Table S4).  
250 Thus, our results are not completely driven by differences in sampling effort.

251 We did not find a significant positive relationship between host longevity and white  
252 blood cell counts (WBC; Table 3). However, we did find a significant negative association  
253 between WBC and PSR in ungulates using total PSR or microparasite PSR (Table 3). WBC was  
254 also significantly positively correlated with body mass across all three groups (Table 3).

255

## 256 **Discussion**

257 Overall, our results show that there is, at best, a weak relationship between parasite  
258 species richness and longevity, at least in this dataset. Longer-lived ungulates have fewer  
259 parasites than short-lived species. However, analyses of primates and carnivores failed to  
260 produce significant associations between longevity and parasite burden, despite generally similar  
261 sample sizes. Several factors may account for the absence of a significant relationship between  
262 parasite species richness and longevity in these groups. Perhaps variables other than longevity  
263 are important in primates and carnivores; for example, Nunn et al. [13] found more compelling  
264 evidence for variables such as geographic range size predicting parasite species richness in  
265 primates. Other studies in carnivores also found no significant relationship between longevity  
266 and parasite burden [16-18]. Lindenfors *et al.* [18] suggested that this resulted from a limit to the  
267 number of parasites a host could acquire in its lifetime; if carnivores more quickly reach this  
268 saturation point, variation in longevity may have less influence on the number of parasites.

269 Perhaps this is true in carnivores; however, it seems unlikely given that we found a significant  
270 relationship in ungulates, which, on average, live longer than carnivores. Alternatively, a  
271 negative relationship between longevity and parasite species richness in carnivores and primates  
272 may be counterbalanced by the loss of parasites as host longevity declines, as predicted by  
273 epidemiological theory. Indeed, the hypotheses are not mutually exclusive, and our tests will  
274 only detect a significant effect when one of the two hypotheses operates particularly strongly.

275 Epidemiological theory suggests that there should be a positive relationship between host  
276 longevity and parasite species richness [20]. Empirical evidence for such a positive correlation  
277 is, however, weak at best. We found no positive significant correlations between longevity and  
278 parasite species richness in our analyses, and although a few previous studies have found  
279 significant positive correlations in primates, Iberian carnivores and freshwater fish [13,17,21],  
280 two of these results only held when outliers were included [13] or body mass was excluded [21].  
281 Thus, empirical evidence for the positive association between longevity and parasite species  
282 richness is generally lacking, suggesting that epidemiological processes involving mortality may  
283 have limited influence on the accumulation of parasite species in hosts [12].

284 If greater parasite burden generally favors low longevity in mammals, then when other  
285 ecological and social conditions favor high longevity, we might expect to find that animals invest  
286 in immune system defenses [55]. Thus, we should see a general association between longevity  
287 and investment in immune defenses, such as immune system cells circulating in the blood.  
288 However, we find no evidence for this hypothesis, with white blood cell counts showing no  
289 significant associations with longevity. This is in contrast to the results of Nunn *et al.* [40] who  
290 found positive correlations between longevity and monocyte and eosinophil counts in mammals  
291 (but only in females). One possible explanation for our results is that longer-lived mammals also

292 invest more in behavioral anti-parasite defenses, for example, avoiding contaminated areas or  
293 individuals, allogrooming, or ingesting medicinal plants [9,56-58]. Such defenses have been  
294 particularly well documented in social mammals like primates [58]. Equally, some long-lived  
295 species may simply not face high parasite risk due to their geographic location or ecology; thus,  
296 parasite infection may have little effect on their longevity. In addition, different parasites may  
297 select for different life histories. For example, chronic infections may select for increased  
298 immune investment and high longevity, whereas acute infections may select for decreased  
299 immune investment and faster reproduction. Our results did not detect any differences in  
300 response to macro- versus micro-parasites; however, these subdivisions may have imprecisely  
301 estimated the degree to which the parasites exhibit chronic versus acute effects.

302         Several methodological issues deserve mention. We used parasite species richness as a  
303 measure of parasite burden, but this ignores the intensity of infection: a host with one individual  
304 of each of 100 species of parasite may not be as negatively affected by parasites as a host with  
305 1000 individuals of just one parasite species. The type of parasite involved may also matter;  
306 some parasites are more virulent than others and thus fitness costs will vary. Hosts should only  
307 invest in immune defenses if the cost of losses due to parasite infections exceeds the often high  
308 costs of immunity. Ideally intensity and virulence should be entered into our models. Finally, we  
309 only have data on three groups of mammals, all of which are fairly large-bodied and long-lived  
310 relative to the majority of mammals. It would be interesting to extend these analyses to include  
311 more species, particularly rodents and bats.

312         Our results indicate that longer-lived ungulates have fewer parasites than those that are  
313 short-lived, which supports previous studies in ungulates and other mammals [12,14]. This effect  
314 may be caused by parasite-induced mortality, which would select for faster life-histories, rather

315 than increased investment in anti-parasite defenses [12,59]. These results may have implications  
316 for ungulate conservation. Generally long-lived mammals are at greater risk of extinction than  
317 short-lived species [60]. However, if the extinction driver involved is an emerging disease, short-  
318 lived ungulates may be hardest hit because they already harbor a greater parasite burden  
319 compared to long-lived ungulates, and also tend to exist at higher densities, which favors the  
320 establishment of infections. Short-lived ungulates are generally smaller and more abundant, and  
321 are therefore common prey items for large carnivores. Thus, if populations of short-lived  
322 ungulates experience a disease outbreak, it could have knock-on effects at higher trophic levels.  
323 This may also be the case in other taxonomic groups that were not part of our analysis. Further  
324 study of the links between parasite burden and host life-history are needed to allow us to protect  
325 biodiversity from infectious disease threats.

326

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329

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455 **Supporting information**

456 The following Supporting Information is available for this article online.

457 Supporting Information S1: Additional sources for life-history data.

458 Supporting Information S2: Dataset.

459 Table S1: Models of parasite species richness including geographic range size and/or group size.

460 Table S2: Full model predicting parasite species richness in Primates.

461 Table S3: Phylogenetic signal in variables.

462 Table S4: AIC values for full models vs. models containing only citation counts.

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