



Host Longevity and Parasite Species Richness in Mammals

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

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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_{macro} and PSR_{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 λ . The parameter λ 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, λ 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 (λ) 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 ± 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_{micro};
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 ± 0.400 , $t_{129} = 2.741$, $p = 0.007$, AIC = 460.5 [AIC with citation count =
244 367.2]; primates: longevity slope = 1.048 ± 0.354 , $t_{124} = 2.958$, $p = 0.004$, AIC = 384.3 [AIC
245 with citation count = 325.7]; ungulates: longevity slope = -0.138 ± 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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