

1 **Coevolution of relative brain size and life expectancy in parrots**

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29

30 **Abstract**

31 Previous studies have demonstrated a correlation between longevity and brain size in a variety of  
32 taxa. Little research has been devoted to understanding this link in parrots; yet parrots are well-known  
33 for both their exceptionally long lives and cognitive complexity. We employed a large-scale  
34 comparative analysis that investigated the influence of brain size and life history variables on  
35 longevity in parrots. Specifically, we addressed two hypotheses for evolutionary drivers of longevity:  
36 the *Cognitive Buffer Hypothesis*, which proposes that increased cognitive abilities enable longer life  
37 spans, and the *Expensive Brain Hypothesis*, which holds that increases in life span are caused by  
38 prolonged developmental time of, and increased parental investment in, large brained offspring. We  
39 estimated life expectancy from detailed zoo records for 133,818 individuals across 244 parrot species.  
40 Using a principled Bayesian approach that addresses data uncertainty and imputation of missing  
41 values, we found a consistent correlation between relative brain size and life expectancy in parrots.  
42 This correlation was best explained by a direct effect of relative brain size. Notably, we found no  
43 effects of developmental time, clutch size, or age at first reproduction. Our results suggest that  
44 selection for enhanced cognitive abilities in parrots have in turn promoted longer lifespans.

45

46 **Keywords**

47 Psittaciformes; longevity; cognitive evolution; Bayesian structural equation model; cognitive buffer  
48 hypothesis; expensive brain hypothesis

49

## 50 Introduction

51 Evolutionary theories of ageing predict the inevitability of senescence in most iteroparous multicellular  
52 organisms (1–4). However, recent studies have highlighted the diversity of patterns and timing in  
53 which different taxa experience senescence, revealing species-specific patterns of longevity linked  
54 with allometry and life history variables (5,6). Generally, larger bodied species tend to live longer (7),  
55 but longevity is also associated with other variables such as diet, latitude and sociality (8,9). Perhaps  
56 of most recent interest, brain size has been correlated with longevity across diverse taxa ranging from  
57 amphibians (10) to primates (11). While some studies have proposed a negative relationship between  
58 brain size and longevity, suggesting a trade-off between the energetic costs into larger brains and  
59 investments in defences against ageing (e.g., (12)), the large majority of studies have suggested a  
60 positive effect of larger brain sizes on longevity (10,11,13–17). However, the causal pathways for this  
61 relationship between brain size and longevity are not yet well established.

62

63 Three non-mutually exclusive hypotheses have been proposed to explain the correlated evolution of  
64 larger brains and longer lifespans. First, the *Cognitive Buffer Hypothesis* posits that increased  
65 cognitive flexibility enabled by a relatively larger brain allows species to solve problems that would  
66 otherwise increase their extrinsic mortality, hence allowing for increased longevity (15). Second, the  
67 *Expensive Brain Hypothesis* argues that there is an indirect association between brains and longevity,  
68 with an investment in expensive brain tissue slowing down the pace of life through increased  
69 developmental time and increased parental investment per offspring (18). Third, the *Delayed Benefits*  
70 *Hypothesis* extends the *Expensive Brain Hypothesis* and reverses the directionality of its argument,  
71 positing that a shift to a higher quality, skill-based diet lowered adult mortality rates and supported a  
72 longer juvenile period that facilitated inter-generational skill transmission. This extended development  
73 in turn allows for investment in brain growth that further promotes skill-based foraging niches. In other  
74 words, long-lived, extractive foraging, species evolve larger brains because they can benefit most  
75 from learning (17). Previous work in mammals, amphibian and birds has found mixed support for all  
76 three hypotheses (13,16). For example, Isler et al. (18) showed that larger brained, monotokous  
77 (single offspring per reproduction), precocial mammals had longer developmental periods. This longer  
78 developmental period led to a prolonged life span; in other words, the effect of brain size on longevity

79 was indirect. In contrast, Jiménez-Ortega et al. (14) showed both a direct and an indirect effect of  
80 absolute brain size on lifespan in birds, with larger brained species also living longer independently  
81 from their developmental period.

82

83 Parrots (Psittaciformes) are famous for both their long lives and complex cognition (19,20), with  
84 lifespans and relative brain size on par with primates (21). Indeed, recent studies on the genetics of  
85 longevity and cognition in parrots have revealed positive selection on lifespan-prolonging genes, as  
86 well as genes related to increased cognitive abilities and cell repair (22–24). Parrots are also  
87 morphologically and ecologically diverse, with an extensive global distribution of almost 400 species,  
88 ranging in size from adult yellow-capped pygmy parrots (*Micropsitta keiensis*, 12 g) to kakapo  
89 (*Strigops habroptilus*, 3000 g) (25). In the first comparative study to examine longevity in parrots,  
90 Munshi-South et al. (19) used maximum longevity records from 162 species, and found that both diet  
91 and communal roosting were correlated with longevity, with granivorous and communal roosting  
92 species living the longest on average. While not considering longevity, the potential drivers of the  
93 evolution of brain size in Neotropical parrots were explored in Schuck-Paim et al. (26), finding that  
94 brain size is associated with environmental and seasonal variability. Finally, highlighting the  
95 importance of life history variation, Young et al. (27) found that longer lived parrots were more likely to  
96 be threatened. To date, however, little research effort has been invested in understanding the link  
97 between longevity and brain size in parrots.

98

99 One of the greatest challenges for comparative life history studies is sourcing good quality data (28).  
100 For instance, the above studies all depended on maximum (or median) recorded lifespan, many used  
101 regressions on residuals (see e.g., González-Lagos et al. (29)) and some only included absolute brain  
102 size (see e.g., Jiménez-Ortega et al. (14)). Maximum recorded lifespan can be a problematic measure  
103 because it represents the longest-lived known individual and is therefore highly sensitive to sample  
104 size. Making matters worse, how much sample size influences results depends on the pattern of age-  
105 related mortality itself (30). For species where most individuals die around the same age, smaller  
106 samples are more likely to approximate maximum longevity than in species with many extreme ages  
107 of death. Therefore, a measure that accounts for all information available is preferable to a single-

108 point measure. Life expectancy is one such a measure and has been found to be the most  
109 appropriate measure of pace of life (31). It calculates the average age at death based on information  
110 across the full age range and therefore takes into account all available information. While life  
111 expectancy can be sensitive to both intrinsic and extrinsic sources of mortality, the use of captive  
112 records allows the removal of extrinsic sources of mortality as much as possible, thereby focusing on  
113 senescence. Yet even when using captive data, other variables and shared evolutionary history  
114 create confounds that need to be addressed within a multivariate framework. A principled way to  
115 decide which covariates to include is the use of Directed Acyclical Graphs (DAG) (32,33). Based on a  
116 specific hypothesis, a DAG represents all potential causal paths in the system by arrows. Conditional  
117 on the DAG being true, the back-door criterion informs which variables should be included and which  
118 should not be included (34). We additionally controlled for variables that only influence life expectancy  
119 to improve accuracy of the model estimates.

120

121 Here, we present a phylogenetic comparative analysis focused on brain size and its effects on  
122 longevity in parrots. First, we estimate life expectancy from Species360's Zoological Information  
123 Management System (ZIMS) with records of 133,818 individuals across 244 parrot species. We then  
124 test for a correlation between life expectancy and relative brain size after removing the effect of  
125 covariates. Third, we used a DAG to distinguish between two possible pathways for this correlation.  
126 The *Cognitive Buffer Hypothesis* predicts a direct effect of relative brain size on life expectancy, with  
127 larger brained species living longer (15), while the *Expensive Brain Hypothesis* predicts that the effect  
128 of brain size on life expectancy is indirect, emerging from increased developmental time and parental  
129 investment per offspring (18). In this case, we expect that any relationship between brain size and life  
130 expectancy will be reduced when also including parental investment (clutch size) and developmental  
131 time in the model. While the *Delayed Benefits Hypothesis* would also predict a direct relationship  
132 between relative brain size and longevity (17), it would argue for strong effect of diet, as well as  
133 reversed directionality (extended longevity leads to larger brain sizes). While we included diet in our  
134 models, our analysis focused explicitly on how brain size could affect longevity, and so we did not fully  
135 explore this hypothesis. Overall, our study demonstrates a robust methodology for comparative life  
136 history analysis using a comprehensive measure of life expectancy in a Bayesian statistical

137 framework. Moreover, it provides the most comprehensive analysis of longevity in Psittaciformes to  
138 date, and contributes to a broader understanding of this understudied group.

139

## 140 **Materials and Methods**

### 141 *Estimating life expectancy*

142 We obtained data on birth and death dates from Species360's ZIMS. After cleaning (see  
143 Supplementary Methods) we included records for 133,818 individuals across 244 species. To  
144 estimate life expectancy, we implemented Bayesian Survival Trajectory Analysis (BaSTA, (35)), which  
145 allowed us to make inferences on age-specific survival based on census data when ages of some  
146 individuals are unknown. The model, implemented in R (36), uses a Markov Chain Monte Carlo  
147 (MCMC) algorithm with Metropolis-Hastings sampling of mortality parameters and latent times of birth.  
148 Here, we used a Siler hazard model (37) for each species, given by

$$149 \quad \mu(x) = \exp[a_0 - a_1x] + c + \exp[b_0 + b_1x],$$

150 where  $a_1, c, b_1 > 0$  and  $a_0, b_0 \in (-\infty, \infty)$ . These five parameters can fit infant and juvenile mortality  
151 (controlled by  $a_0$  and  $a_1$ ), age independent (adult) mortality ( $c$ ) as well as senescent mortality  
152 (controlled by  $b_0$  for initial mortality and  $b_1$  for the rate of aging). Cumulative survival can be calculated  
153 as

$$154 \quad S(x) = \exp\left[-\int_0^x \mu(t)dt\right].$$

155 Life expectancy at birth is calculated as

$$156 \quad e_0 = \int_0^{\infty} S(x)dx.$$

157 We used the Gelman-Rubin statistic (Rhat, (38)) to determine if models converged and visually  
158 assessed the traces and model goodness of fit. When models did not converge, they were rerun with  
159 longer burn-in and more iterations. If models clearly did not fit the data, the results were excluded.  
160 This was the case for 27 out of 244 species. In most cases this was due to issues with data quality  
161 (e.g., when the number of individuals without a recorded date of death was too high).

162

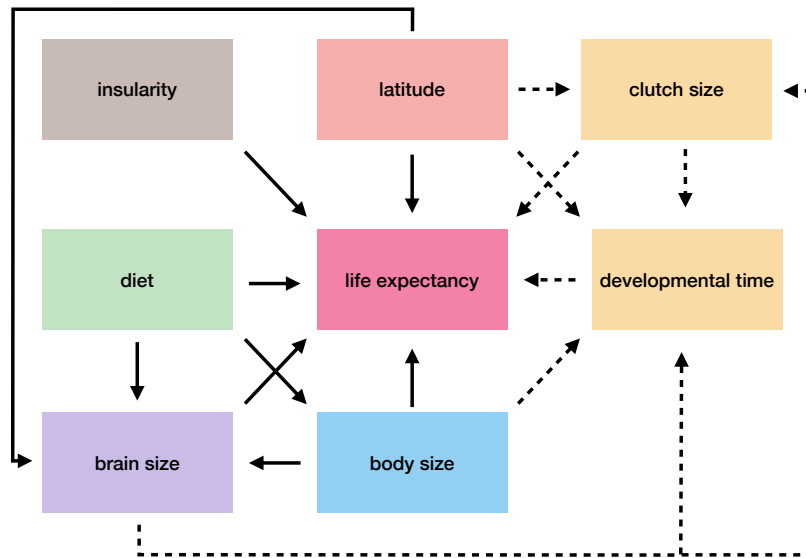
163 *Life-history covariates*

164 We collected body mass data from ZIMS. Additional body mass measurements were included from  
165 the literature if no captive records were available for a species (28). We then used a Bayesian multi-  
166 level model to extract species-level averages and standard errors (see Supplemental Methods more  
167 details). Brain mass was collected by AI, from Iwaniuk et al. (39), from Schuck-Paim et al. (26) and  
168 from Ksepka et al. (40), and similarly to body size, we fitted a Bayesian multi-level model to extract  
169 species-level averages and standard errors. We also collected data for six additional potential  
170 explanatory variables, based on previously proposed causal relationships with life expectancy: diet  
171 (estimated protein content of main food items) (19), insularity (whether a species includes a  
172 continental range or not) (19), maximum latitudinal range (as a proxy for environmental variability)  
173 (41), clutch size (42), developmental time (from the start of incubation until fledging) and age of first  
174 possible reproduction (AFR) (18). Diet, insularity, maximum latitude range, clutch size and  
175 developmental time were collected from the literature. When data were not freely available, we  
176 collected estimates directly from experts (see Supplemental Methods for the details). Finally, AFR is  
177 unknown for the large majority of parrot species. We therefore estimated it directly from the  
178 distribution of first breeding records in ZIMS, using the 5% percentile. To control for possible issues  
179 arising from low sample sizes, we restricted this analysis to species with at least 30 breeding  
180 individuals.

181

182 We used a DAG (see Figure 1) to decide how to incorporate variables in the statistical models,  
183 accounting for their influences on each other in proposed causal pathways. It is important to note that  
184 evolutionary time is not included explicitly in the DAG, thus arrows can potentially go in both  
185 directions, representing evolutionary feedbacks. However, in our view, it represents the most  
186 principled representation of the potential causal relationships for evolution of longevity in parrots,  
187 based on available data and current knowledge. Although not depicted in the DAG, phylogenetic co-  
188 variance was assumed to influence all variables and was included in all analyses using the L2-norm

189 (which calculates the covariance between two species based on a maximum possible covariance and  
190 the squared distance between the two species) and the phylogenetic tree from Burgio et al. (43).



191

192 **Figure 1** Directed Acyclic Graph of the potential causal pathways that could drive parrot life expectancy. Colours  
193 represent different covariate groups and are kept consistent throughout the manuscript. Solid lines represent  
194 assumed causal effects in all models (see Statistical analysis for model definitions). Dashed lines represent  
195 additional causal relationships in model 2 and 3.

196

### 197 Statistical analysis

198 To test for a correlation between life expectancy and relative brain size, we first constructed a  
199 Bayesian structural equation model (model 1) with life expectancy as the main variable to be  
200 explained by relative brain size and four other potential covariates. We included a total of 360 species  
201 for which at least one variable was known. The structure of this first model was as follows:  $LE \sim I +$   
202  $BO + RB + LA + D$ , where  $LE$  = standardised log life expectancy,  $I$  = insularity (binary),  $BO$  =  
203 standardised log body mass,  $RB$  = relative brain size,  $LA$  = standardised maximum latitude range and  
204  $D$  = protein content diet (ordinal). Relative brain size was calculated as:  $BR - pBR$ , where  $BR$  =  
205 standardised log brain mass and  $pBR$  = predicted brain mass from a second model that ran  
206 simultaneously:  $pBR \sim BO$ . Relative brain size has been shown to correlate with innovation rates in  
207 birds (44) and we therefore use it as a proxy for cognitive flexibility. Our implementation is similar to  
208 residual brain size in multiple regressions, but since both models are evaluated at each step of the



209 sampling, information flows in both directions and measurement error is modelled correctly (45). We  
210 included standard error around the mean for life expectancy, body mass and brain mass. We also  
211 included a phylogenetic variance-covariance matrix based on the phylogenetic distances calculated  
212 from Burgio et al. (43), using the L2-norm. For each variable with missing data, missing values were  
213 imputed using a multinormal distribution with mean and standard deviation based on the observed  
214 data, variance-covariance based on the phylogenetic signal and means further informed by the causal  
215 relationships outlined in Figure 1. For life expectancy we had data for 244 species, but the models  
216 only converged for 217 species. Life expectancy for the remaining 143 parrot species was therefore  
217 imputed (see Supplemental Methods for details).

218

219 To test whether any correlation between relative brain size and longevity could be indirectly caused  
220 by developmental time, delayed juvenile periods, and/or parental investment, we ran a second model  
221 (model 2) where developmental time (incubation period plus fledging period in model 2) and clutch  
222 size were included as additional covariates. Both variables were log transformed and standardised.  
223 Since data on AFR (a third measure of developmental time) was only available for 89 species and the  
224 available data was biased towards later AFR (see Supplemental Methods for more detail), we did not  
225 attempt to impute this variable, but tested its effect in a third model (model 3) limited to cases where  
226 AFR was known.

227

228 To assess which hypothesis was best supported by the data, we compared the effect of relative brain  
229 size in the three models. If an increase in relative brain size directly causes an increase in life  
230 expectancy (*Cognitive Buffer Hypothesis*), we would expect the coefficient of the brain size effect to  
231 be positive and similar in all three models. If an increase in relative brain size only causes an increase  
232 in developmental time (*Expensive Brain Hypothesis*), we would expect the coefficient of the brain size  
233 effect to be positive in model 1 and much reduced or zero in model 2 and 3.

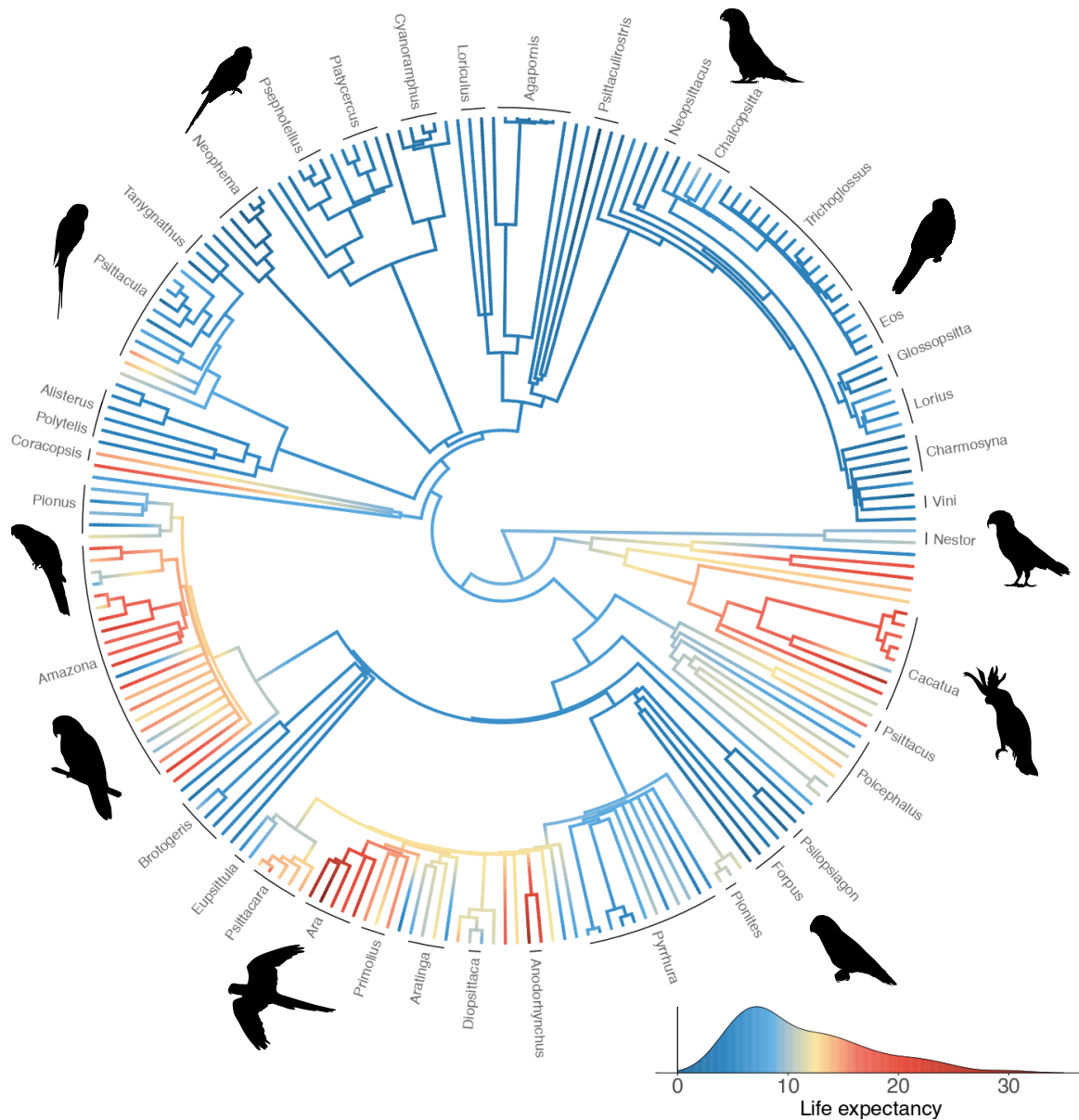
234

235 **Results**

236

237 Overall, we were able to estimate life expectancy for 217 species out of 244 species for which we had  
238 data. This included representatives of all eight major genera (i.e., those with at least ten species) and  
239 over half of the extant parrot species. The shortest-lived genera were the small-bodied  
240 *Psittaculirostris* and *Chamosyna*, e.g., with a life expectancy of less than 2 years for *Psittaculirostris*  
241 *desmarestii*. The longest-lived genera were the large-bodied *Ara* and *Cacatua*, e.g., with a life  
242 expectancy of more than 35 years for *Ara macao* (full distribution of values across the phylogenetic  
243 tree is shown in Figure 2). Similarly, there was large variability in other covariates, e.g., with brain size  
244 ranging from 1 to 22 grams, and age of first reproduction ranging from 7 months to 6 years. There  
245 was a strong phylogenetic signal in life expectancy (Figure 2b), however, covariance was generally  
246 low between species that belonged to different genera (Figure 3c).

247



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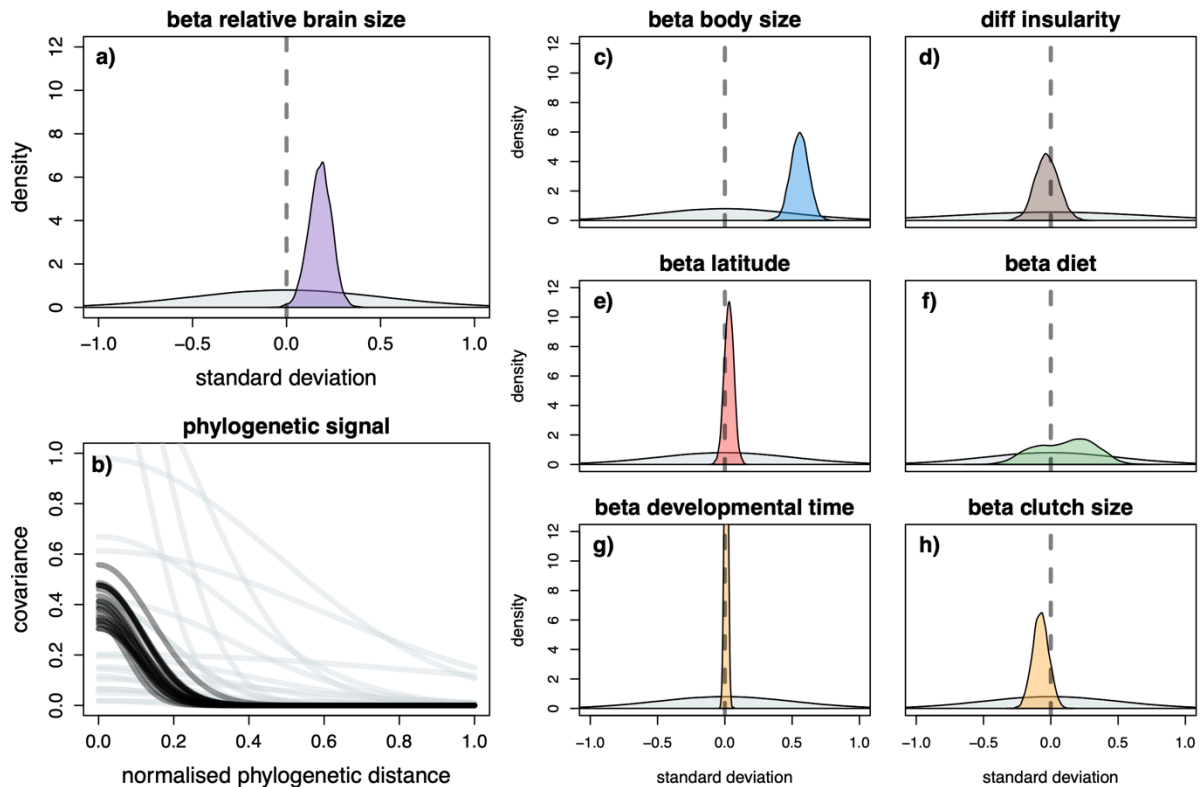
249 **Figure 2.** Phylogenetic tree of the 217 parrot species included in the study. Branches are coloured according to  
 250 life expectancy (see density plot in bottom right), and phylogeny is based on Burgio et al. (43). Genera are  
 251 named if they contain at least two species. For a version with all species named see Supplemental Figure S1.

252

253 Model 1 (without developmental time and parental investment) as well as model 2 and 3 (including  
 254 these potential indirect paths) had similar estimates for the direct effect of relative brain size. As  
 255 expected, body size was strongly and positively correlated with life expectancy (see Figure 3c for  
 256 model 2, Supplementary Results for model 1 and 3). Relative brain size also had a small, but  
 257 consistently positive, effect on life expectancy ( $\beta = 0.22$  in model 1,  $\beta = 0.18$  in model 2 and  $\beta = 0.16$   
 258 in model 3; overlap with zero  $< 0.03$  for all models; Figures 3a, 4). Of the other life history variables  
 259 included, none appeared to have a large effect on life expectancy (see Figure 3d-h). In particular,

260 model 2 showed no effect of developmental time ( $\beta = 0.01$ , overlap with zero > 0.22) or clutch size ( $\beta$   
261 = -0.08, overlap with zero > 0.88) on longevity, and there was no clear effect of AFR on longevity in  
262 model 3 ( $\beta = -0.11$ , overlap with zero > 0.88). However, it should be noted that these models were  
263 designed to test the effect of relative brain size, so other parameter estimates should be interpreted  
264 with caution (46).

265

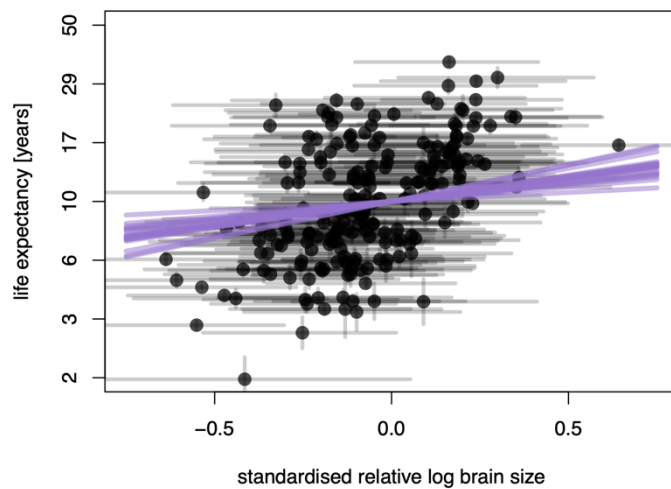


266

267 **Figure 3.** Parameter estimates for model 2. For results of model 1 and 3 see Figure S2 and S4. Grey density  
268 plots and lines are the regularising priors. Coloured areas are the posterior densities for the parameter estimates  
269 controlling the effect of the covariates on life expectancy. Black lines are 20 samples of the posterior for the  
270 phylogenetic covariance. For insularity the difference between islandic and continental species is shown.

271

272



273

274 **Figure 4.** Standardised relative log brain size vs life expectancy for model 2. Black points represent 217 species  
275 where life expectancy was available, vertical black lines represent the SE for life expectancy, horizontal black  
276 lines represent the 89% percentile intervals for standardised relative log brain size. Purple lines represent 20  
277 samples from the posterior for the slope (beta) of the effect of standardised relative log brain size on life  
278 expectancy.

279

## 280 Discussion

281

282 Using an extensive database from captive parrots, our study showed a clear and positive correlation  
283 between relative brain size and life expectancy in parrots. We further tested two hypotheses to  
284 explain this observed correlation between relative brain size and life expectancy: the *Cognitive Buffer*  
285 *Hypothesis* (15) and the *Expensive Brain Hypothesis* (18). Our results best supported a direct  
286 relationship between larger brains and longer life expectancy, as predicted under the *Cognitive Buffer*  
287 *Hypothesis*. It should be noted that this result is also consistent with the *Delayed Benefits Hypothesis*  
288 (17). We would, however, also expect a strong effect of diet on life expectancy, since this hypothesis  
289 argues that long life spans allow species to invest more time in learning foraging skill which requires  
290 larger brains, and only pays off with an extended juvenile period. To fully explore this hypothesis we  
291 would need data on post-fledging parental care and future studies could additionally try to use  
292 process-based approaches (where evolution is modelled explicitly), such as generative inference (47)  
293 or Bayesian ancestral state reconstruction (48) to disentangle the direction of causality. We found no  
294 evidence that the relationship between relative brain size and life expectancy was explained by the

295 need for longer development times (here measured by incubation to fledging time, and by age of first  
296 reproduction), or by increased parental investment (here represented by clutch size), as predicted by  
297 the *Expensive Brain Hypothesis*. Interestingly, our results differ from a previous study in parrots by  
298 Munshi-South et al. (19). This study found that the protein content of diets and communal roosting  
299 best explained variation in maximum longevity. Data on sociality is largely lacking for parrots, so we  
300 did not test for an effect of sociality, but we found no effect of diet. However, Munshi-South et al. did  
301 not consider brain size in their analysis. Since diet potentially determines whether and how quickly  
302 brains can grow (49), protein intake could still have an indirect effect on longevity via its potential link  
303 with brain size.

304

305 The lack of support for the *Expensive Brain Hypothesis* is contrary to previous studies in primates  
306 (11,50), other mammals (29,51), and amphibians (10), all of which show a positive correlation  
307 between developmental time or AFR and life expectancy. However, it is in line with previous work  
308 examining the evolution of longevity in birds (14). To explain this discrepancy between birds and  
309 mammals, Isler et al. (16) suggested that bird species with allomaternal care (care provided for  
310 mother or offspring by either the father or helpers) can provide enough nutrition for relatively larger  
311 brained offspring without the need to prolong developmental periods or reduce clutch size to an extent  
312 that would lead to the co-evolution of increased lifespans. All parrots have relatively large brain sizes  
313 compared to most other birds, and all parrot species exhibit biparental care. Almost all parrots are  
314 also cavity nesters. Cavity nests are less vulnerable to predation, and often have extensive nest  
315 defence strategies, and so can have relatively relaxed selective pressure on fledging times as  
316 compared to open-cup nesters (52). Perhaps the combination of these factors provides enough  
317 flexibility to deal with heightened nutritional demands of rearing large-brained offspring without  
318 selection on developmental times. This does not, however, diminish the importance of cognitive  
319 development in parrots. The extended juvenile periods observed in many parrot species of up to six  
320 years may provide enhanced opportunities for social learning, as proposed for another large-brained  
321 bird taxon, the corvids (53). This hypothesis remains to be tested in parrots.

322

323 To our knowledge this is the first study of life expectancy and/or brain size that uses a bespoke  
324 Bayesian model to include: 1) uncertainty about variable estimates; 2) imputation of missing values;  
325 3) a principled representation of relative brain size; and 4) phylogenetic signal. We believe this  
326 method has some major advantages. Most notably, we could estimate both life expectancy and its  
327 uncertainty in each species. This allowed us to fully exploit the fact that we have a hundred-fold more  
328 data for some species, instead of relying on a single point estimate of maximum longevity as in  
329 previous studies of longevity in parrots (19,27). We also imputed life expectancy for species which  
330 have no data. This is likely to be important in most datasets to account for biased data collection, but  
331 it is especially important when using data from captivity, because zoos do not randomly pick species  
332 to be included in their population, but have a general bias toward larger and longer-lived species (54).  
333 Complete case analysis will introduce bias in this case (55) and we therefore chose to impute missing  
334 values. The use of DAGs and structural equation models is very similar to path analysis. The main  
335 advantage of our implementation is that it allows for a statistically robust definition of relative brain  
336 size and can handle uncertainty and missing data. Our model structure can be easily adapted to  
337 impute any continuous variable.

338  
339 Our study also departs from most previous studies of longevity by using data from captivity on life  
340 expectancy (41,56–58). This provided several important advantages. First, it provided a large sample  
341 size, both improving the estimation of life expectancy per species and allowing us to have a fuller  
342 representation of species. Second, captivity reduces external sources of mortality as much as  
343 possible (little predation, starvation, etc.). However, captive data poses different challenges. First, as  
344 with data from the wild, birth and death dates can be missing (e.g., for individuals born in the wild or  
345 transferred from institutions that are not part of ZIMS). The BaSTA implementation that we used  
346 imputed these missing values, and we believe that our thorough cleaning procedure, coupled with the  
347 sheer magnitude of the dataset, means that any gaps, data entry errors or biases should have  
348 minimal effect on the life expectancies presented here. Second, there may be differences in causes of  
349 death in captivity and the wild, for example if some species are difficult to keep or prone to negative  
350 behavioural responses to captivity which is also true for some of the shortest-lived genera included in  
351 the study such as *Psittaculirostris* and *Chamosyna* which have been historically difficult to manage in

352 captivity. We dealt with this by excluding potentially problematic species from the initial life  
353 expectancy estimations, and instead imputed values in the final model (see Supplemental Methods  
354 for details). We can still not be completely sure that the patterns observed in the data are all  
355 representative of the evolutionary processes that shaped them, but it is highly unlikely that the clear  
356 positive correlation between relative brain size and life expectancy is due to captivity. It could even be  
357 expected that large brained species live shorter in captivity, because of the higher metabolic rates  
358 required to keep the large brain supplied with glucose. This has been shown to be the case within  
359 species in captive guppies (12). Since such an effect would be opposite to the one observed in our  
360 study, its presence would not change the conclusions drawn from our results.

361

## 362 **Conclusions**

363

364 Overall, our results are consistent with the *Cognitive Buffer Hypothesis*, suggesting that relatively  
365 large brains may have buffered parrots against environmental variability and/or predation threats  
366 reducing sources of extrinsic mortality and allowing longer lifespans. This result is consistent with  
367 previous studies in other birds, suggesting that common processes may explain longevity in altricial  
368 birds. In addition to their longevity, parrots are famous for their complex cognition. It remains largely  
369 unknown what evolutionary processes have driven cognitive evolution in parrots, but given the results  
370 of our study, in addition to those of Munshi-South et al. (19), future work should further investigate the  
371 potentially complex feedbacks between these two factors and sociality and diet. Unfortunately, longer  
372 lived species are also more likely to be threatened (27), showing the vulnerability of this order. Having  
373 life expectancy and other life history variables for hundreds of species will hopefully aid in future  
374 conservation efforts for this globally threatened order.

375

## 376 **Data, code and materials**

377 Data, code and materials are publicly available at <https://doi.org/10.5061/dryad.sbccc2fr7x>. A  
378 repository is also publicly available at  
379 [https://github.com/simeonqs/Coevolution\\_of\\_relative\\_brain\\_size\\_and\\_life\\_expectancy\\_in\\_parrots](https://github.com/simeonqs/Coevolution_of_relative_brain_size_and_life_expectancy_in_parrots).

380



381 **Competing interests**

382 The authors have no competing interests with this study.

383

384 **Author Contributions**

385 AMY, DAC, LMA, MBM and SQS conceived the idea. AI, AMY, LA, TFW, SB and SQS collected the  
386 data. SQS analysed the data under supervision from LA and MBM. AB, DAC, LA, MBM and SQS  
387 drafted the initial manuscript, and all authors contributed to writing and editing the final article. All  
388 contributors are listed in alphabetical order.

389

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