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**

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., Gonzáles-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
$$\mu(x) = \exp[a_0 - a_1 x] + c + \exp[b_0 + b_1 x],$$

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

155   Life expectancy at birth is calculated as

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

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).
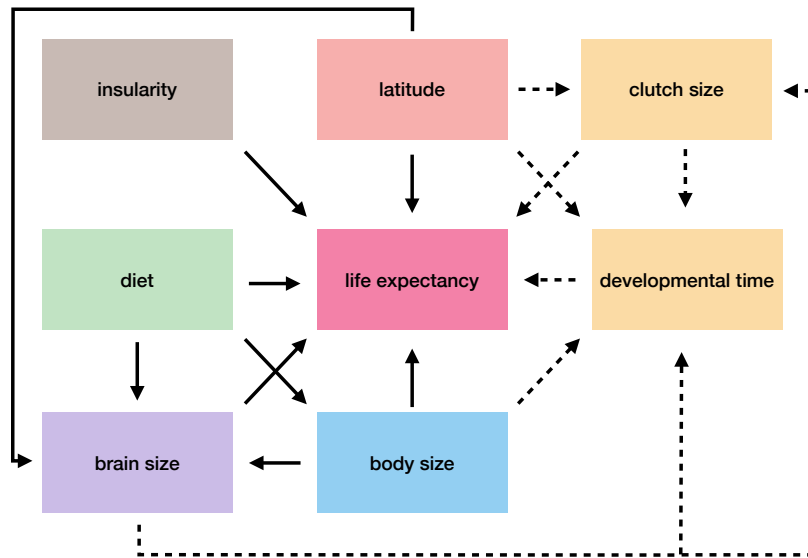
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192   **Figure 1** *Directed Acyclic Graph of the potential causal pathways that could drive parrot life expectancy. Colours*
193   *represent different covariate groups and are kept consistent throughout the manuscript. Solid lines represent*
194   *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 ~ 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 ~ 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 *Charmosyna*, 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).

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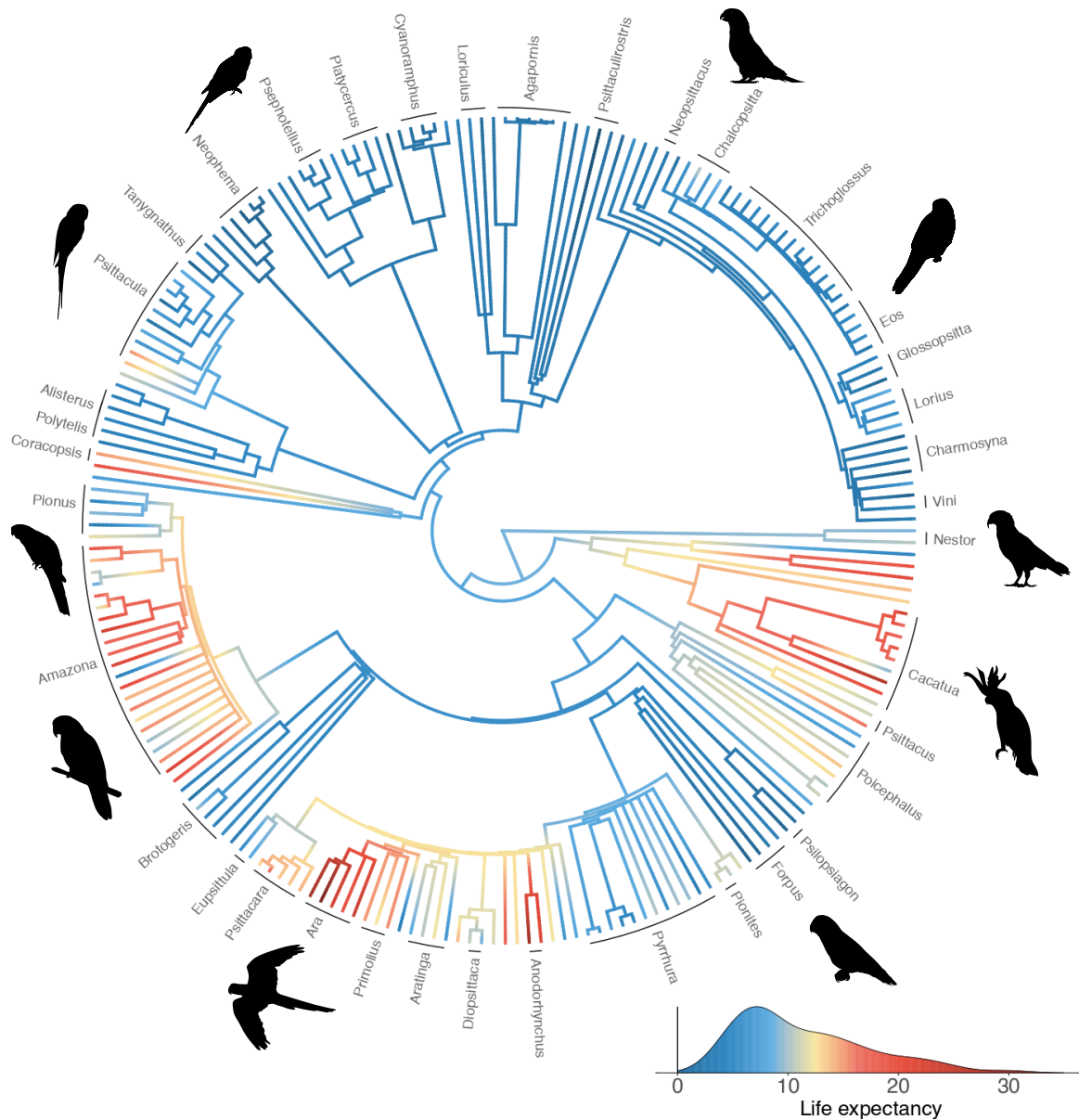
Figure 2. *Phylogenetic tree of the 217 parrot species included in the study. Branches are coloured according to life expectancy (see density plot in bottom right), and phylogeny is based on Burgio et al. (43). Genera are named if they contain at least two species. For a version with all species named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

Model 1 (without developmental time and parental investment) as well as model 2 and 3 (including these potential indirect paths) had similar estimates for the direct effect of relative brain size. As expected, body size was strongly and positively correlated with life expectancy (see Figure 3c for model 2, Supplementary Results for model 1 and 3). Relative brain size also had a small, but consistently positive, effect on life expectancy ($\beta = 0.22$ in model 1, $\beta = 0.18$ in model 2 and $\beta = 0.16$ in model 3; overlap with zero < 0.03 for all models; Figures 3a, 4). Of the other life history variables 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 (β = 0.01, overlap with zero > 0.22) or clutch size (β

261   = -0.08, overlap with zero > 0.88) on longevity, and there was no clear effect of AFR on longevity in

262   model 3 (β = -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).

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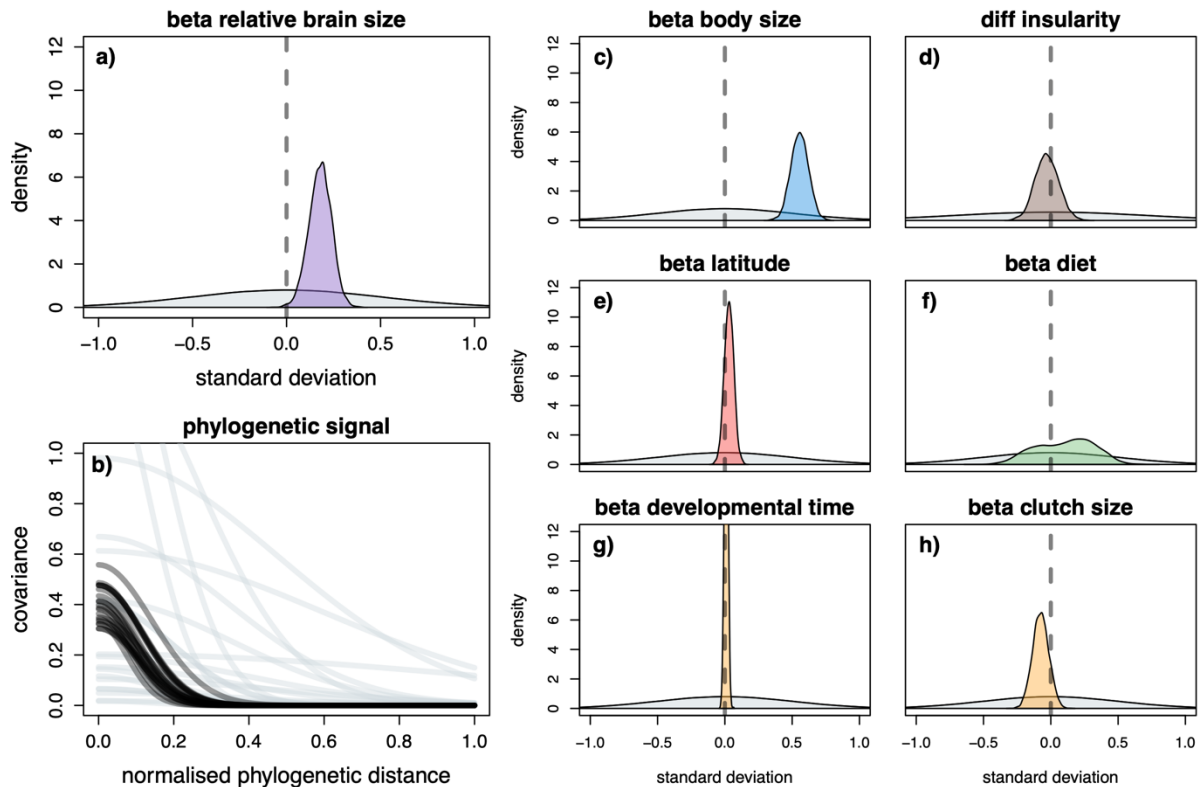
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267   *Figure 3.* *Parameter estimates for model 2. For results of model 1 and 3 see Figure S2 and S4. Grey density*
268   *plots and lines are the regularising priors. Coloured areas are the posterior densities for the parameter estimates*
269   *controlling 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

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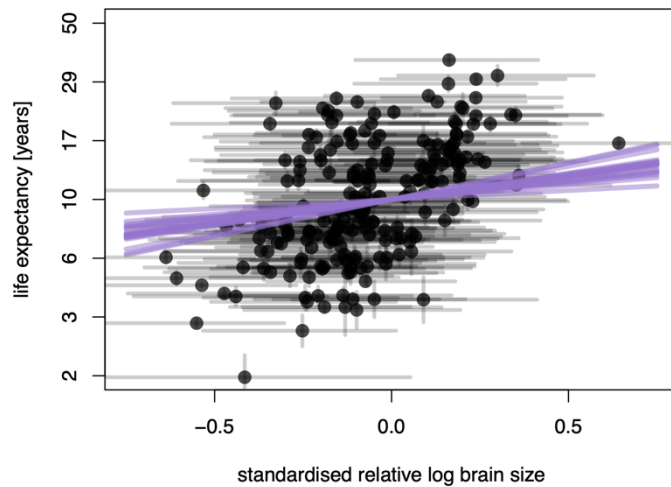
Figure 4. Standardised relative log brain size vs life expectancy for model 2. Black points represent 217 species where life expectancy was available, vertical black lines represent the SE for life expectancy, horizontal black lines represent the 89% percentile intervals for standardised relative log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

Discussion

Using an extensive database from captive parrots, our study showed a clear and positive correlation

between relative brain size and life expectancy in parrots. We further tested two hypotheses to

explain this observed correlation between relative brain size and life expectancy: the *Cognitive Buffer*

Hypothesis (15) and the *Expensive Brain Hypothesis* (18). Our results best supported a direct

relationship between larger brains and longer life expectancy, as predicted under the *Cognitive Buffer*

Hypothesis. It should be noted that this result is also consistent with the *Delayed Benefits Hypothesis*

(17). We would, however, also expect a strong effect of diet on life expectancy, since this hypothesis

argues that long life spans allow species to invest more time in learning foraging skill which requires

larger brains, and only pays off with an extended juvenile period. To fully explore this hypothesis we

would need data on post-fledging parental care and future studies could additionally try to use

process-based approaches (where evolution is modelled explicitly), such as generative inference (47)

or Bayesian ancestral state reconstruction (48) to disentangle the direction of causality. We found no

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 *Charmosyna* 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.sbcc2fr7x. A

378   repository is also publicly available at

379   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.

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