Coevolution of brain size and longevity in parrots

Simeon Q. Smeele[1,2,3,4,*], Dalia A. Conde[6,4,5], Annette Baudisch[4], Simon Bruslund[7,8], Andrew Iwaniuk[9], Johanna Staerk[4,5,6], Timothy F. Wright[10], Anna M. Young[11], Mary Brooke McElreath[1,2,^], Lucy Aplin[1,12,^]

[1] Cognitive & Cultural Ecology Research Group, Max Planck Institute of Animal Behavior, Radolfzell, Germany

[2] Department of Human Behavior, Ecology and Culture, Max Planck Institute for Evolutionary Anthropology, Leipzig, Germany

[3] Department of Biology, University of Konstanz, Konstanz, Germany

[4] Interdisciplinary Centre on Population Dynamics, University of Southern Denmark, Odense, Denmark

[5] Department of Biology, University of Southern Denmark, Odense, Denmark

[6] Species360 Conservation Science Alliance, Bloomington, USA

[7] Vogelpark Marlow gGmbH, Marlow, Germany

[8] European Association of Zoos and Aquaria, Parrot Taxon Advisory Group, Amsterdam, The Netherlands

[9] Department of Neuroscience, University of Lethbridge, Lethbridge, Canada

[10] Biology Department, New Mexico State University, Las Cruces, USA

[11] Department of Biology and Earth Science, Otterbein University, Westerville, USA

[12] Centre for the Advanced Study of Collective Behaviour, University of Konstanz, Konstanz, Germany

^ Joint senior authors

* Corresponding author: ssmeele@ab.mpg.de

Abstract

Parrots are well-known for their exceptionally long lives and cognitive complexity. While previous studies have demonstrated a correlation between longevity and brain size in a variety of taxa, little research has been devoted to understanding this link in parrots. Here we employed a large-scale comparative analysis that investigated the influence of brain size and life history variables on patterns of longevity. Specifically, we addressed two hypotheses for evolutionary drivers of longevity: the *Cognitive Buffer Hypothesis*, which proposes that increased cognitive abilities enable longer life spans, and the *Expensive Brain Hypothesis*, which holds that the increase in life span is caused by prolonged developmental time of and increased parental investment in, large brained offspring. We estimated life expectancy from detailed zoo records for 133,818 individuals across 244 parrot species. Using Bayesian structural equation models, we found a consistent correlation between relative brain size and life expectancy in parrots. This correlation was best explained by a direct effect of relative brain size. Notably, we found no effects of developmental time, clutch size, or age at first reproduction. Our results provide support for the *Cognitive Buffer Hypothesis,* and demonstrate a principled Bayesian approach that addresses data uncertainty and imputation of missing values.

Keywords

Psittaciformes; life expectancy; cognitive evolution; Bayesian structural equation model; cognitive buffer hypothesis; expensive brain hypothesis

Introduction

Evolutionary theories of ageing predict the inevitability of senescence in most iteroparous multicellular organisms (1–4). However, recent studies have highlighted the diversity of patterns and timing in which different taxa experience senescence, revealing species-specific patterns of longevity linked with allometry and life history variables (5,6). Generally, larger bodied species tend to live longer (7), but longevity is also associated with other variables such as diet, latitude and sociality (8,9). Perhaps of most recent interest, brain size has been correlated with longevity across diverse taxa ranging from amphibians (10) to primates (11). However, the causal pathways for this relationship between brain size and longevity are not yet well established.

Three non-mutually exclusive hypotheses have been proposed to explain the correlated evolution of larger brains and longer lifespans. First, the *Cognitive Buffer Hypothesis* posits that increased cognitive flexibility enabled by a relatively larger brain allows species to solve problems that would otherwise increase their extrinsic mortality, hence allowing for increased longevity (12). Second, the *Delayed Benefits Hypothesis* reverses the directionality of this argument, positing that longevity drives the evolution of larger brains. In other words, long-lived species evolve larger brains because they can benefit most from the cognitive machinery that supports learning (13). These first two hypotheses can be difficult to disentangle in comparative studies, as they both predict a direct association between relative brain size and longevity. Third, the *Expensive Brain Hypothesis* argues that there is an indirect association between brains and longevity, with an investment in expensive brain tissue slowing down the pace of life through increased developmental time and increased parental investment per offspring (14). Previous work in mammals, amphibian and birds has found mixed support for all three hypotheses (15,16). For example, Isler et al. (14) showed that larger brained, monotokous (single offspring per reproduction), precocial mammals had longer developmental periods. This longer developmental period led to a prolonged life span; in other words, the effect of brain size on longevity was indirect. In contrast, Jiménez-Ortega et al. (17) showed both a direct and an indirect effect of absolute brain size on lifespan in birds, with larger brained species also living longer independently from their developmental period.

Parrots (Psittaciformes) are famous for both their long lives and complex cognition (18,19), with lifespans and relative brain size on par with primates (20). Indeed, recent studies on the genetics of longevity and cognition in parrots have revealed positive selection on lifespan-prolonging genes, as well as genes related to increased cognitive abilities and cell repair (21–23). Parrots are also morphologically and ecologically diverse, with an extensive global distribution of almost 400 species, ranging in size from adult yellow-capped pygmy parrots (*Micropsitta keiensis*, from 12 g) to kakapo (*Strigops habroptilus*, up to 3000 g) (24). In the first comparative study to examine longevity in parrots, Munshi-South et al. (18) used maximum longevity records from 162 species, and found that both diet and communal roosting were correlated with longevity, with granivorous and communal roosting species living the longest on average. While not considering longevity, the potential drivers of the evolution of brain size in neotropical parrots were explored in Schuck-Paim et al. (25), finding that brain size is associated with environmental and seasonal variability. Finally, highlighting the importance of life history variation, Young et al. (26) found that longer lived parrots were more likely to be threatened. However, perhaps surprisingly, little research effort has been invested in understanding the link between longevity and brain size in parrots.

One of the greatest challenges for comparative life history studies is sourcing good quality data (27). For instance, the above studies all depended on maximum (or median) recorded lifespan, many used regressions on residuals (see e.g., DeCasien et al. (16)) and some only included absolute brain size (see e.g., Jiménez-Ortega et al. (17)). Maximum recorded lifespan can be a problematic measure because it represents the longest-lived known individual and is therefore highly sensitive to sample size. Making matters worse, how much sample size influences results depends on the pattern of age-related mortality itself (28). For species where most individuals die around the same age, smaller samples are more likely to approximate maximum longevity than in species with many extreme ages of death. Therefore, preferable to a single-point measure is a measure that accounts for all information available. Life expectancy is such a measure and has been found to be the most appropriate measure of pace of life (29). It calculates the average age at death based on information across the full age range and therefore takes into account all available information. While life expectancy can be sensitive to both intrinsic and extrinsic sources of mortality, the use of captive records allows the removal of extrinsic sources of mortality as much as possible, thereby focusing on senescence. It is also thought to be the best measure of pace of life (29). Yet even when using captive data, other variables and shared evolutionary history create confounds that need to be addressed within a multivariate framework. A principled way to decide which covariates to include is the use of Directed Acyclical Graphs (DAG) (30,31). Based on a specific hypothesis, a DAG represents all potential causal paths in the system by arrows. Conditional on the DAG being true, the backdoor criterion informs which variable should be included and which should not be included.

Here, we present a phylogenetic comparative analysis focused on brain size and its effects on longevity in parrots. First, we estimate life expectancy from Species360's Zoological Information Management System (ZIMS) with records of 133,818 individuals across 244 parrot species. We then test for a correlation between life expectancy and relative brain size after removing the effect of covariates. Third, we used a DAG to distinguish between two possible pathways for this correlation. The *Cognitive Buffer Hypothesis* predicts a direct effect of relative brain size on life expectancy, with larger brained species living longer (12), while the *Expensive Brain Hypothesis* predicts that the effect of brain size on life expectancy is indirect, emerging from increased developmental time and parental investment per offspring (14). In this case, we expect that any relationship between brain size and life expectancy will be reduced when also including parental investment (clutch size) and developmental time in the model. While the *Delayed Benefits Hypothesis* would also predict a direct relationship between relative brain size and longevity (13), it would argue for reversed directionality (extended longevity leads to larger brain sizes). As this hypothesis was evoked to explain hominid evolution with multiple overlapping generations, and we are explicitly focusing on variance in longevity across species, we did not examine it further in this analysis. Overall, our study demonstrates a robust methodology for comparative life history analysis using a comprehensive measure of life expectancy in a Bayesian statistical framework. Moreover, it provides the most comprehensive analysis of life expectancy and longevity in Psittaciformes to date, and contributes to a broader understanding of this understudied group.

Materials and Methods

Estimating life expectancy

We obtained data on birth and death dates from Species360's ZIMS. After cleaning (see Supplementary Methods) we included records for 133,818 individuals across 244 species. To estimate life expectancy, we implemented Bayesian Survival Trajectory Analysis (BaSTA, (32)), which allowed us to make inferences on age-specific survival based on census data when ages of some individuals are unknown. The model, implemented in R (33), uses a Markov Chain Monte Carlo (MCMC) algorithm with Metropolis-Hastings sampling of mortality parameters and latent times of birth. Here, we used a Siler hazard model (34) for each species, given by

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

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

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

Life expectancy at birth is calculated as

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

We used the Gelman-Rubin statistic (Rhat, (35)) to determine if models converged and visually assessed the traces and model goodness of fit. When models did not converge, they were rerun with longer burn-in and more iterations. If models clearly did not fit the data, the results were excluded. This was the case for 27 out of 244 species. In most cases this was due to issues with data quality (e.g., when the number of individuals without a recorded date of death was too high).

Life-history covariates

We collected body mass data from ZIMS. Additional body mass measurements were included from the literature if no captive records were available for a species (27). We then used a Bayesian multi-level model to extract species-level averages and standard errors (see Supplemental Methods more details). Brain mass was collected by AI, from Iwaniuk et al. (36), from Schuck-Paim et al. (25) and from Ksepka et al. (37), and similarly to body size, we fitted a Bayesian multi-level model to extract species-level averages and standard errors. We also collected data for six additional potential explanatory variables, based on previously proposed causal relationships with life expectancy: diet (estimated protein content of main food items) (18), insularity (whether a species includes a continental range or not) (18), maximum latitudinal range (as a proxy for environmental variability) (38), clutch size (39), developmental time (from the start of incubation until fledging) and age of first possible reproduction (AFR) (14). Diet, insularity, maximum latitude range, clutch size and developmental time were collected from the literature. When data were not freely available, we collected estimates directly from experts (see Supplemental Methods for the details). Finally, AFR is unknown for the large majority of parrot species. We therefore estimated it directly from the distribution of first breeding records in the ZIMS, using the 5% percentile. To control for possible issues arising from low sample sizes, we restricted this analysis to species with at least 30 breeding individuals.

We used a DAG (see Figure 1) to decide how to incorporate variables in the statistical models, accounting for their influences on each other in proposed causal pathways. It is important to note that evolutionary time is not included explicitly in the DAG, thus arrows can potentially go in both directions, representing evolutionary feedbacks. However, in our view, it represents the most principled representation of the potential causal relationships for evolution of longevity in parrots, based on available data and current knowledge. Although not depicted in the DAG, phylogenetic co-variance was assumed to influence all variables and was included in all analyses using the L2-norm and the phylogenetic tree from Burgio et al. (40).

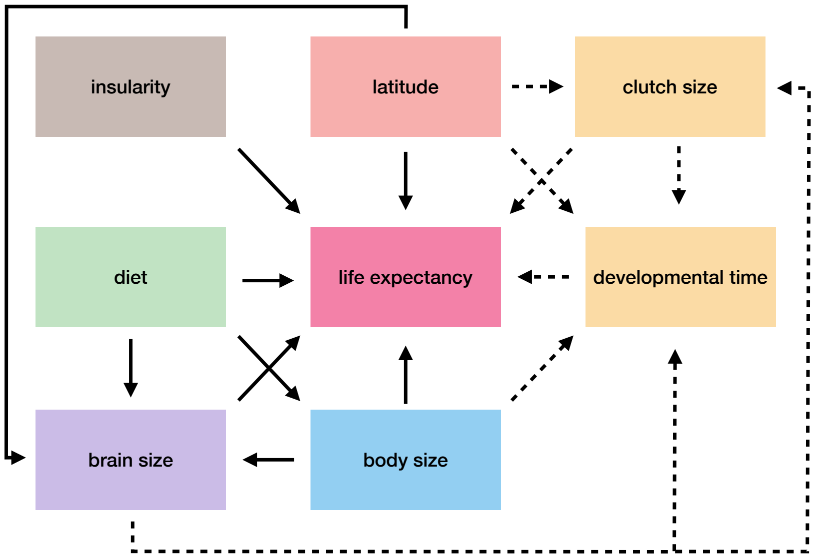

Figure 1 Directed Acyclic Graph of the potential causal pathways that could drive parrot life expectancy. Colours represent different covariate groups and are kept consistent throughout the manuscript. Solid lines represent assumed causal effects in all models. Dashed lines represent assumed causal relationships in model 2 and 3.

Statistical analysis

To test for a correlation between life expectancy and relative brain size, we first constructed a Bayesian structural equation model with life expectancy as the main variable to be explained by relative brain size and four other potential covariates. We included a total of 360 species for which at least one variable was known. The structure of this first model was as follows: LE ~ I + BO + RB + LA + D, where LE = standardised log life expectancy, I = insularity (binary), BO = standardised log body mass, RB = relative brain size, LA = standardised maximum latitude range and D = protein content diet (ordinal). Relative brain size was calculated as: BR – pBR, where BR = standardised log brain mass and pBR = predicted brain mass from a second model that ran simultaneously: pBR ~ BO. This is similar to residual brain size in multiple regressions, but since both models are evaluated at each step of the sampling, information flows in both directions and measurement error is modelled correctly (41). We included standard error around the mean for life expectancy, body mass and brain mass. We also included a phylogenetic variance-covariance matrix based on the phylogenetic distances calculated from Burgio et al. (40), using the L2-norm. For each variable with missing data, missing values were imputed using a multinormal distribution with mean and standard deviation based on the observed data, variance-covariance based on the phylogenetic signal and means further informed by the causal relationships outlined in Figure 1. For a detailed version of the model see Supplemental Methods.

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

Results

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

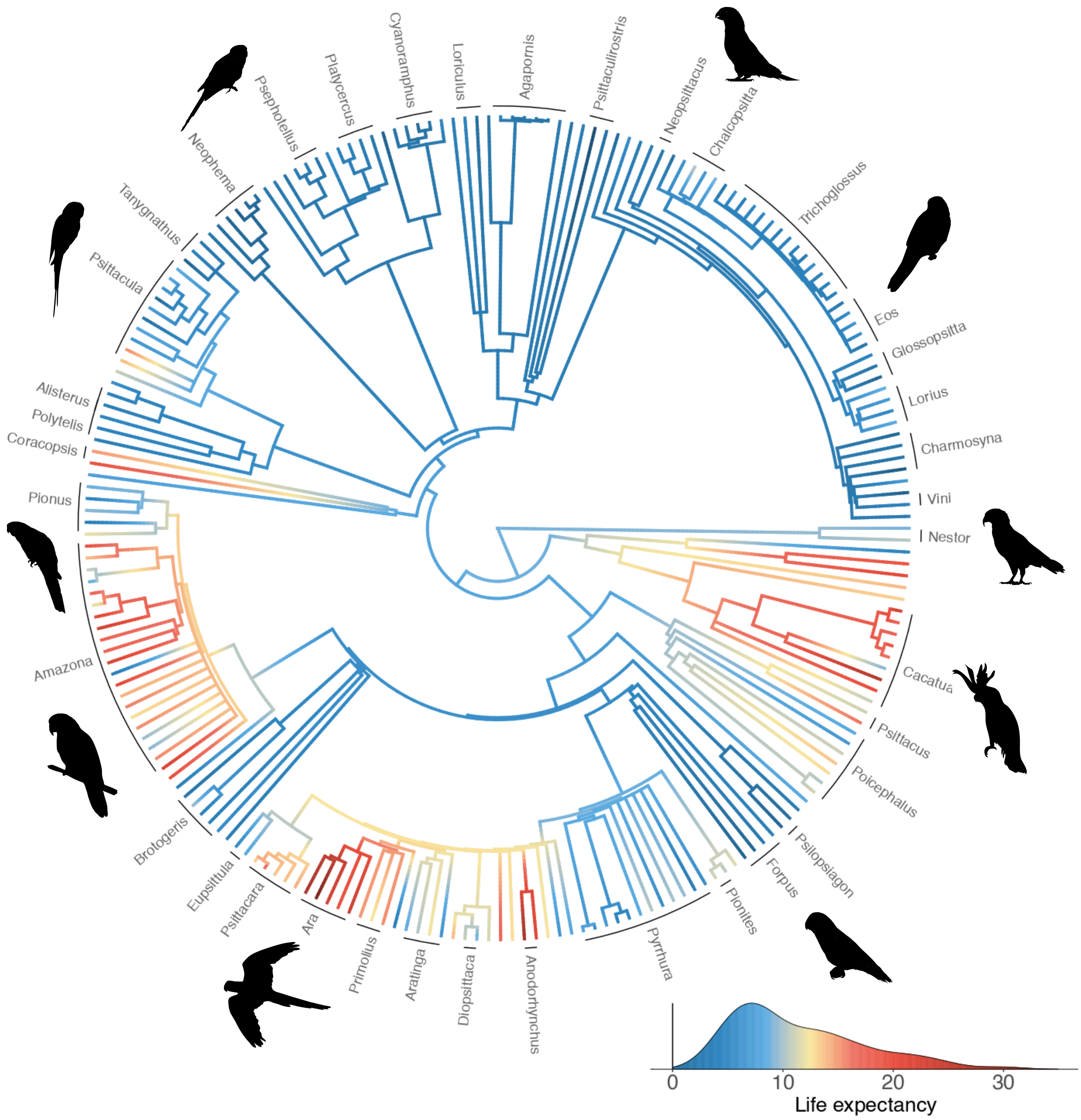

Figure 2. *Phylogenetic tree of the 217 parrot species included in the study. Branches are coloured according to life expectancy (see density plot in bottom right), and phylogeny is based on Burgio et al. (40). Genera are named if they contain at least two species. For a version with all species named 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 (β = 0.22 in model 1, β = 0.18 in model 2 and β = 0.16 in model 3; Figures 3a, 4). Of the other life history factors included, none appeared to have a large effect on life expectancy (see Figure 3d-h). In particular, model 2 showed no effect of developmental time (β = 0.01) or clutch size (β = -0.05) on longevity, and there was no clear effect of AFR on longevity in model 3 (β = -0.11). However, it should be noted that these models were designed to test the effect of relative brain size, so other parameter estimates should be interpreted with caution (42).

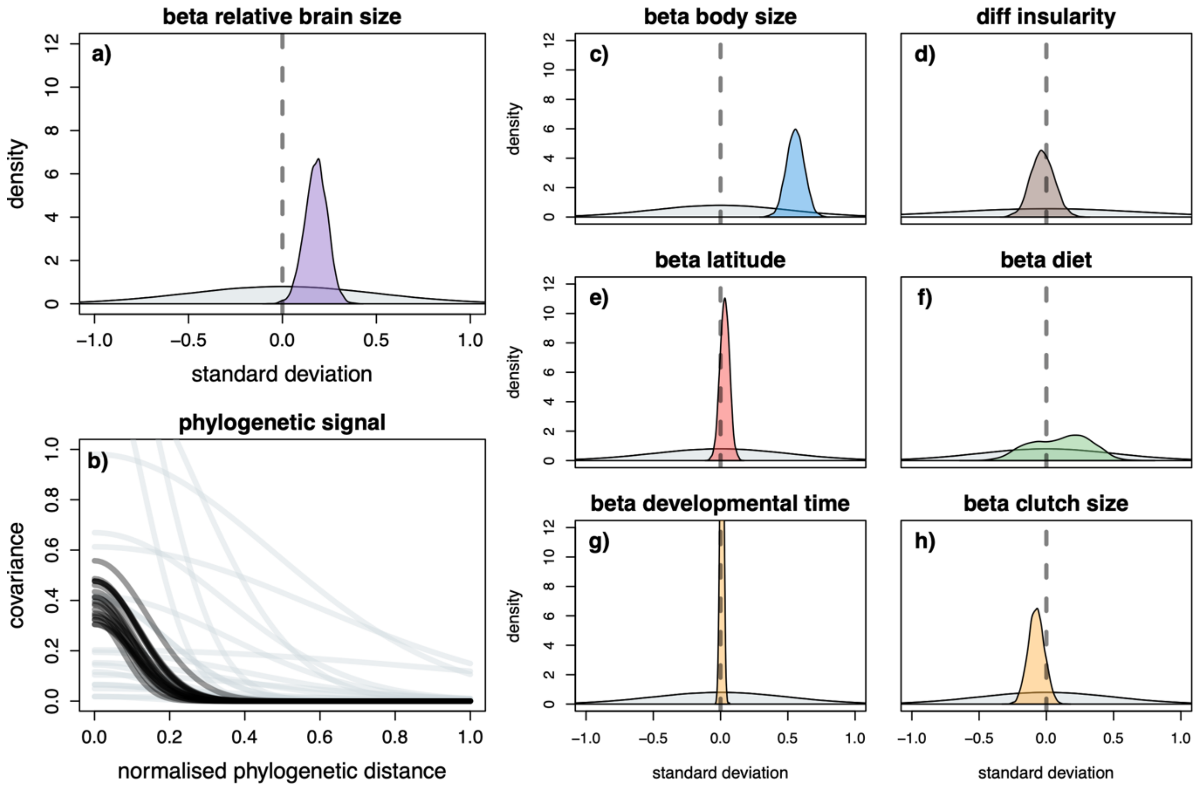

Figure 3. *Parameter estimates for model 2. For results of model 1 and 3 see Figure S2 and S4. Grey density plots and lines are the regularising priors. Coloured areas are the posterior densities for the parameter estimates controlling the effect of the covariates on life expectancy. Black lines are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

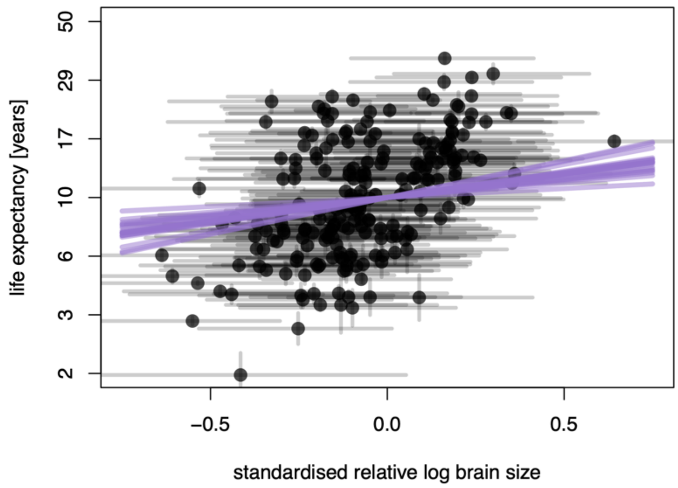

Figure 4. *Standardised relative log brain size vs life expectancy for model 2. Black points represent 217 species where life expectancy was available, vertical black lines represent the SE for life expectancy, horizontal black lines represent the 89% percentile intervals for standardised relative log 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 (12)* and the *Expensive Brain Hypothesis (14).* 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 (13).* These hypotheses could not be disentangled in this analysis, as both predict a direct relationship between life expectancy and cognition, albeit with reversed directionality. Future studies could additionally try to use process-based approaches (where evolution is modelled explicitly), such as generative inference (43) or Bayesian ancestral state reconstruction (44) to disentangle the direction of causality. However, we found no evidence that the relationship between relative brain size and life expectancy was explained by the need for longer development times (here measured by incubation to fledging time, and by age of first reproduction), or by increased parental investment (here represented by clutch size), as predicted by the *Expensive Brain Hypothesis*. Interestingly, our results differ from a previous study in parrots by Munshi-South et al. (18). This study found that the protein content of diets and communal roosting best explained variation in maximum longevity. Data on sociality is largely lacking for parrots, so we did not test for an effect of sociality, but we found no effect of diet. However, Munshi-South et al. did not consider brain size in their analysis. Since diet potentially determines whether and how quickly brains can grow (45), protein intake could still have an indirect effect on longevity via its potential link with brain size.

The lack of support for the *Expensive Brain Hypothesis* is contrary to previous studies in primates (11,46), other mammals (47,48), and amphibians (10), all of which show a positive correlation between developmental time or AFR and life expectancy. However, it is in line with previous work examining the evolution of longevity in birds (17). To explain this discrepancy between birds and mammals, Isler et al. (15) suggested that bird species with allomaternal care (care provided for mother or offspring by either the father or helpers) can provide enough nutrition for relatively larger brained offspring without the need to prolong developmental periods or reduce clutch size to an extent that would lead to the co-evolution of increased lifespans. All parrots have relatively large brain sizes compared to most other birds, and all parrot species exhibit biparental care. Almost all parrots are also cavity nesters. Cavity nests are less vulnerable to predation, and often have extensive nest defence strategies, and so can have relatively relaxed selective pressure on fledging times as compared to open-cup nesters (49). Perhaps the combination of these factors provides enough flexibility to deal with heightened nutritional demands of rearing large-brained offspring without selection on developmental times. This does not, however, diminish the importance of cognitive development in parrots. The extended juvenile periods observed in many parrot species of up to six years may provide enhanced opportunities for social learning, as proposed for another large-brained bird taxon, the corvids (50). This hypothesis remains to be tested in parrots.

To our knowledge this is the first study of life expectancy and/or brain size that uses a bespoke Bayesian model to include: 1) uncertainty about variable estimates; 2) imputation of missing values; 3) a principled representation of relative brain size; and 4) phylogenetic signal. In our opinion, this method has some major advantages. Most notably, we could estimate both life expectancy and its uncertainty in each species. This allowed us to fully exploit the fact that we have a hundred-fold more data for some species, instead of relying on a single point estimate of maximum longevity. We also imputed life expectancy for species which have no data. This is likely to be important in most datasets to account for biased data collection, but it is especially important when using data from captivity, because zoos never randomly pick species to be included in their population, leading to a bias toward larger and longer-lived species (51). Complete case analysis will introduce bias in this case (52) and we therefore chose to impute missing values. Our model structure can be easily adapted to impute any continuous variable.

Our study also departs from most previous studies of longevity by using data from captivity on life expectancy (38,53–55). This provided several important advantages. First, it provided a large sample size, both improving the estimation of life expectancy per species and allowing us to have a fuller representation of species. Second, captivity reduces external sources of mortality as much as possible (little predation, starvation, etc.). However, captive data poses different challenges. First, similarly to data from the wild, birth and death dates can be missing (e.g., for individuals born in the wild or transferred from institutions that are not part of ZIMS). The BaSTA implementation that we used imputed these missing values, and we believe that our thorough cleaning procedure, coupled with the sheer magnitude of the dataset, means that any gaps, data entry errors or biases should have minimal effect on the life expectancies presented here. Third, there may be differences in causes of death in captivity and the wild, for example if some species are difficult to keep or prone to negative behavioural responses to captivity which is also true for some of the shortest-lived genera included in the study such as *Psittaculirostris* and *Charmosyna* which have been historically difficult to manage in captivity. We dealt with this by excluding potentially problematic species from the initial life expectancy estimations, and instead imputed values in the final model (see Supplemental Methods for details). We can still not be completely sure that the patterns observed in the data are all representative of the evolutionary processes that shaped them, but it is highly unlikely that the clear positive correlation between relative brain size and life expectancy is due to captivity.

Conclusions

Overall, our results are consistent with the *Cognitive Buffer Hypothesis*, suggesting that relatively large brains may have buffered parrots against environmental variability and/or predation threats reducing sources of extrinsic mortality and allowing longer lifespans. This result is consistent with previous studies in other birds, suggesting that common processes may explain longevity in altricial birds. As well as longevity, parrots are famous for their complex cognition. It remains largely unknown what evolutionary processes have driven cognitive evolution in parrots, but given the results of our study, in addition to those of Munshi-South et al. (18), future work should further investigate the potentially complex feedbacks between these two factors and sociality and diet. Unfortunately, longer lived species are also more likely to be threatened (26), showing the vulnerability of this order. Having life expectancy and other life history variables for hundreds of species will hopefully aid in future conservation efforts for this globally threatened order.

Data, code and materials

Data will be made publicly available upon publication. Code is publicly available at https://github.com/simeonqs/Coevolution_of_brain_size_and_longevity_in_parrots.

Competing interests

The authors have no competing interests with this study.

Author Contributions

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

Acknowledgments

We would like to thank Dr. Dieter Lukas, Dr. Cody Ross, Prof. Fernando Colchero, Prof. Richard McElreath and Dr. Rita da Silva for their advice on the analysis. We would further like to thank the more than 1,300 Species360 members for registering their animals in the ZIMS database. We thank Prof. Alejandro Salinas, Geddes Hislop and Johann C. Carstens for contributing data on diet.

Funding

This project was possible thanks to the financial support of the sponsor members of the Species360 Conservation Science Alliance (Copenhagen Zoo, the World Association of Zoos and Aquariums, and the Wildlife Reserves of Singapore), the Interdisciplinary Centre on Population Dynamics and the Biology Department at the University of Southern Denmark. We would like to thank Species360 for granting access to the data under permission number #86892. This work was supported by the Max Planck Society. LMA was funded by a Max Planck Independent Group Leader Fellowship. SQS received additional funding from the International Max Planck Research School for Organismal Biology.

References

1. Medawar PB, University College London. An Unsolved problem of biology; an inaugural lecture delivered at University College, London, 6 December, 1951. London: H.K. Lewis and Co.; 1952.

2. Williams GC. Pleiotropy, natural selection, and the evolution of senescence. evolution. 1957;398–411.

3. Hamilton WD. The moulding of senescence by natural selection. J Theor Biol. 1966;12(1):12–45.

4. Kirkwood TB. Evolution of ageing. Nature. 1977;270(5635):301–4.

5. Jones OR, Scheuerlein A, Salguero-Gómez R, Camarda CG, Schaible R, Casper BB, et al. Diversity of ageing across the tree of life. Nature. 2014;505(7482):169–73.

6. Colchero F, Jones OR, Conde DA, Hodgson D, Zajitschek F, Schmidt BR, et al. The diversity of population responses to environmental change. Ecol Lett. 2019;22(2):342–53.

7. Lindstedt SL, Calder III WA. Body size, physiological time, and longevity of homeothermic animals. Q Rev Biol. 1981;56(1):1–16.

8. Wasser D, Sherman P. Avian longevities and their interpretation under evolutionary theories of senescence. J Zool. 2010;280(2):103–55.

9. Scholer MN, Strimas-Mackey M, Jankowski JE. A meta-analysis of global avian survival across species and latitude. Ecol Lett. 2020;23(10):1537–49.

10. Yu X, Zhong MJ, Li DY, Jin L, Liao WB, Kotrschal A. Large-brained frogs mature later and live longer. Evolution. 2018;72(5):1174–83.

11. Street SE, Navarrete AF, Reader SM, Laland KN. Coevolution of cultural intelligence, extended life history, sociality, and brain size in primates. Proc Natl Acad Sci. 2017;114(30):7908–14.

12. Allman J, McLaughlin T, Hakeem A. Brain weight and life-span in primate species. Proc Natl Acad Sci. 1993;90(1):118–22.

13. Kaplan H, Hill K, Lancaster J, Hurtado AM. A theory of human life history evolution: Diet, intelligence, and longevity. Evol Anthropol Issues News Rev Issues News Rev. 2000;9(4):156–85.

14. Isler K, van Schaik CP. The expensive brain: a framework for explaining evolutionary changes in brain size. J Hum Evol. 2009;57(4):392–400.

15. Isler K, Van Schaik CP. Why are there so few smart mammals (but so many smart birds)? Biol Lett. 2009;5(1):125–9.

16. DeCasien AR, Thompson NA, Williams SA, Shattuck MR. Encephalization and longevity evolved in a correlated fashion in Euarchontoglires but not in other mammals. Evolution. 2018;72(12):2617–31.

17. Jiménez-Ortega D, Kolm N, Immler S, Maklakov AA, Gonzalez-Voyer A. Long life evolves in large-brained bird lineages. Evolution. 2020;74(12):2617–28.

18. Munshi-South J, Wilkinson GS. Diet influences life span in parrots (Psittaciformes). The Auk. 2006;123(1):108–18.

19. Auersperg AM, von Bayern AM. Who'sa clever bird—now? A brief history of parrot cognition. Behaviour. 2019;156(5–8):391–407.

20. Olkowicz S, Kocourek M, Lučan RK, Porteš M, Fitch WT, Herculano-Houzel S, et al. Birds have primate-like numbers of neurons in the forebrain. Proc Natl Acad Sci. 2016;113(26):7255–60.

21. Costantini D, Racheli L, Cavallo D, Dell'Omo G. Genome size variation in parrots: longevity and flying ability. J Avian Biol. 2008;39(4):453–9.

22. Andrews CB, Gregory TR. Genome size is inversely correlated with relative brain size in parrots and cockatoos. Genome. 2009;52(3):261–7.

23. Wirthlin M, Lima NC, Guedes RLM, Soares AE, Almeida LGP, Cavaleiro NP, et al. Parrot genomes and the evolution of heightened longevity and cognition. Curr Biol. 2018;28(24):4001-4008. e7.

24. Dunning Jr JB. CRC handbook of avian body masses. CRC press; 2007.

25. Schuck-Paim C, Alonso WJ, Ottoni EB. Cognition in an ever-changing world: climatic variability is associated with brain size in neotropical parrots. Brain Behav Evol. 2008;71(3):200–15.

26. Young AM, Hobson EA, Lackey LB, Wright TF. Survival on the ark: life-history trends in captive parrots. Anim Conserv. 2012;15(1):28–43.

27. Conde DA, Staerk J, Colchero F, da Silva R, Schöley J, Baden HM, et al. Data gaps and opportunities for comparative and conservation biology. Proc Natl Acad Sci. 2019;116(19):9658–64.

28. Vaupel JW. Post-darwinian longevity. Popul Dev Rev. 2003;29:258–69.

29. Wrycza TF, Baudisch A. The pace of aging: Intrinsic time scales in demography. Demogr Res. 2014;30:1571–90.

30. Laubach ZM, Murray EJ, Hoke KL, Safran RJ, Perng W. A biologist's guide to model selection and causal inference. Proc R Soc B. 2021;288(1943):20202815.

31. Pearl J, Glymour M, Jewell NP. Causal inference in statistics: A primer. John Wiley & Sons; 2016.

32. Colchero F, Jones OR, Rebke M. BaSTA: an R package for Bayesian estimation of age-specific survival from incomplete mark–recapture/recovery data with covariates. Methods Ecol Evol. 2012;3(3):466–70.

33. R Core Team. R: A language and environment for statistical computing. 2013; Available from: https://www.R-project.org/

34. Siler W. A competing-risk model for animal mortality. Ecology. 1979;60(4):750–7.

35. Gelman A, Carlin JB, Stern HS, Dunson DB, Vehtari A, Rubin DB. Bayesian data analysis. CRC press; 2013.

36. Iwaniuk AN, Dean KM, Nelson JE. Interspecific allometry of the brain and brain regions in parrots (Psittaciformes): comparisons with other birds and primates. Brain Behav Evol. 2005;65(1):40–59.

37. Ksepka DT, Balanoff AM, Smith NA, Bever GS, Bhullar B-AS, Bourdon E, et al. Tempo and pattern of avian brain size evolution. Curr Biol. 2020;30(11):2026-2036. e3.

38. Scholer MN, Strimas-Mackey M, Jankowski JE. A meta-analysis of global avian survival across species and latitude. Ecol Lett. 2020;23(10):1537–49.

39. Ghalambor CK, Martin TE. Fecundity-survival trade-offs and parental risk-taking in birds. Science. 2001;292(5516):494–7.

40. Burgio KR, Davis KE, Dreiss LM, Cisneros LM, Klingbeil BT, Presley SJ, et al. Phylogenetic supertree and functional trait database for all extant parrots. Data Brief. 2019;24:103882.

41. Freckleton RP. On the misuse of residuals in ecology: regression of residuals vs. multiple regression. J Anim Ecol. 2002;542–5.

42. Westreich D, Greenland S. The table 2 fallacy: presenting and interpreting confounder and modifier coefficients. Am J Epidemiol. 2013;177(4):292–8.

43. Kandler A, Powell A. Generative inference for cultural evolution. Philos Trans R Soc B Biol Sci. 2018;373(1743):20170056.

44. Ringen E, Martin JS, Jaeggi A. Novel phylogenetic methods reveal that resource-use intensification drives the evolution of “complex” societies. 2021;

45. Robson S. Breast milk, diet, and large human brains. Curr Anthropol. 2004;45(3):419–25.

46. Powell LE, Barton RA, Street SE. Maternal investment, life histories and the evolution of brain structure in primates. Proc R Soc B. 2019;286(1911):20191608.

47. González-Lagos C, Sol D, Reader S. Large-brained mammals live longer. J Evol Biol. 2010;23(5):1064–74.

48. Barton RA, Capellini I. Maternal investment, life histories, and the costs of brain growth in mammals. Proc Natl Acad Sci. 2011;108(15):6169–74.

49. Martin TE. Nest predation and nest sites. BioScience. 1993;43(8):523–32.

50. Uomini N, Fairlie J, Gray RD, Griesser M. Extended parenting and the evolution of cognition. Philos Trans R Soc B. 2020;375(1803):20190495.

51. Frynta D, Lišková S, Bültmann S, Burda H. Being attractive brings advantages: the case of parrot species in captivity. PloS One. 2010;5(9):e12568.

52. Little RJ, Rubin DB. Statistical analysis with missing data. Stat Anal Missing Data. 2002;

53. Blumstein DT, Møller AP. Is sociality associated with high longevity in North American birds? Biol Lett. 2008;4(2):146–8.

54. Beauchamp G. Group-foraging is not associated with longevity in North American birds. Biol Lett. 2010;6(1):42–4.

55. Arnold KE, Owens IP. Cooperative breeding in birds: a comparative test of the life history hypothesis. Proc R Soc Lond B Biol Sci. 1998;265(1398):739–45.