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Maternal investment, life histories, and the evolution of brain structure in primates

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Maternal investment, life histories, and the evolution of brain structure in primates

Abstract

Life history is a robust correlate of relative brain size: larger-brained mammals and birds have slower life histories and longer lifespans than smaller-brained species. The Cognitive Buffer Hypothesis (CBH) proposes an adaptive explanation for this relationship: large brains may permit greater behavioural flexibility and thereby buffer the animal from unpredictable environmental challenges, allowing for reduced mortality and increased lifespan. In contrast, the Developmental Costs Hypothesis (DCH) suggests that life-history correlates of brain size reflect the extension of maturational processes needed to accommodate the evolution of large brains, predicting correlations with pre-adult life history phases. Here we test novel predictions of the hypotheses in primates applied to the neocortex and cerebellum, two major brain structures with distinct developmental trajectories. While neocortical growth is allocated primarily to pre-natal development, the cerebellum exhibits relatively substantial post-natal growth. Consistent with the DCH, neocortical expansion is related primarily to extended gestation while cerebellar expansion to extended post-natal development, particularly the juvenile period. Contrary to the CBH, adult lifespan explains relatively little variance in whole brain or neocortex volume once pre-adult life history phases are accounted for. Only the cerebellum shows a relationship with lifespan after accounting for developmental periods. Our results substantiate and elaborate on the role of maternal investment and offspring development in brain evolution, suggest that brain components can evolve partly independently through modifications of distinct developmental phases, and imply that environmental input during post-natal maturation may be particularly crucial for the development of cerebellar function. They also suggest that relatively extended maturation times provide a developmental mechanism for the marked expansion of the cerebellum in the apes.

Introduction

Extended lifespan is one of the most consistent correlates of large brain size across mammal species (e.g. (1, 2)). Despite being first identified over 100 years ago (1) and confirmed by multiple comparative analyses since (e.g. (2, 3)), the biological significance of the brain size-lifespan correlation remains uncertain. The Cognitive Buffer Hypothesis (hereafter "CBH"), posits that larger brains bestow behavioural flexibility, which in turn reduces mortality by enabling individuals to adjust to environmental contingency and

38   unpredictability, and that this reduction in mortality facilitates longer lifespans (1, 2, 4, 5). A variant of the
39   CBH proposes an alternative causal scenario in which longer lifespans create conditions favouring the
40   evolution of enlarged brains, because species with longer reproductive periods have greater opportunities to
41   reap the benefits of investment in learning during development (4), termed the "delayed benefits" hypothesis
42   (3, 5), e.g. (6). Under the umbrella of the CBH, various elements of behavioural flexibility are emphasised as
43   beneficial for survival in variable environments, including innovation (4), complex foraging strategies (7) and
44   social learning (8). Common to all these ideas, however, is the prediction that across species, there should be
45   a direct association between relatively enlarged brains and longer lifespans (5).

46   In addition to adaptive benefits, however, it is increasingly recognised that large brains impose costs, which
47   may provide a sufficient explanation for the positive correlation between brain size and lifespan. The
48   Developmental Costs Hypothesis (hereafter "DCH") (9), together with the related 'Maternal Energy'
49   hypothesis (10, 11) proposes that life history correlates of brain size reflect the need to extend development
50   and maternal investment in order to build a large brain. Previous comparative analyses have provided support
51   for this hypothesis: across mammals, pre-natal brain growth correlates specifically with gestation duration,
52   while post-natal brain growth correlates specifically with lactation duration. Once these effects are accounted
53   for, the brain size-lifespan correlation becomes non-significant, suggesting it is a side-effect of developmental
54   costs (9). Positive correlations between adult brain size and pre- and post-natal developmental periods in birds
55   are also consistent with the DCH (12). An additional 'costs-based' hypothesis, the 'Expensive Brain'
56   framework, proposes a trade-off between the energetic costs of large brains and reproduction, such that large-
57   brained species must spread higher costs of reproduction over longer lifespans (11). Common to both the
58   Expensive Brain and Developmental Costs Hypotheses is the prediction that the brain size-lifespan correlation
59   is indirect, mediated by relationships of both variables to protracted developmental periods.

60   To date, tests of these hypotheses have examined whole brain size. However, the brain is composed of
61   functionally and anatomically heterogeneous structures which show heterochronicity in their developmental
62   scheduling (13–16), influenced by structure-specific genes (17). The DCH therefore predicts that different
63   brain structures should have specific developmental correlates across species. A comparative analysis in
64   primates provides general support for this idea, finding that some brain components correlate more strongly
65   with lifespan and some with age at first reproduction (1). However, this study did not examine specific
66   developmental periods relevant to different aspects of brain growth, nor did it explicitly consider contrasting
67   predictions made by costs-based and adaptive hypotheses. Furthermore, it did not control for phylogenetic
68   non-independence in comparative data (1).

69   Here, we test the predictions of the DCH and CBH by examining life history correlates of brain size and the
70   size of two major brain structures which together make up a substantial proportion of total brain size, and
71   which have expanded relative to other structures during primate evolution: the neocortex and cerebellum (18).
72   These two structures have to some extent evolved in a coordinated fashion, congruent with their anatomical

73   and functional connectivity (19) but also partly independently, in a mosaic fashion, with a notable acceleration

74   in the rate of cerebellar expansion in the ape clade (20). These patterns of coordinated and mosaic brain

75   component evolution are reflected in patterns of change at the molecular level, with similar numbers of

76   changes in genes annotated during neocortical and cerebellar ontogeny in non-ape anthropoids, but more

77   changes in cerebellar than neocortical genes during ape evolution (17). These phenotypic and genetic patterns

78   imply that developmental mechanisms can be adjusted to facilitate a complex pattern of both coordinated and

79   mosaic evolution of the cerebellum and neocortex.

80   Neurodevelopmental studies on humans and other primates suggest that one ontogenetic mechanism

81   facilitating divergent evolution of these two structures is a substantial difference in the allocation of growth

82   to pre- versus post-natal phases. While growth and neurogenesis of the neocortex is predominantly pre-natal,

83   the cerebellum exhibits relatively rapid and prolonged post-natal neurogenesis and volumetric growth (21,

84   22). At birth, the human cerebellum is approximately 25% of its volume at two years of age, increasing by

85   240% in the first year post-natally, whilst the neonatal neocortex is already 46% of its volume at two years,

86   increasing by a relatively modest 88% in the first year after birth (21). In terms of neurogenesis, the cerebellum

87   is unusual in that the large majority (85%) of human cerebellar granule cells - the most numerous class of

88   neurons in the brain - are generated post-natally (22). Post-natal growth of the human cerebellum is

89   particularly extended relative to the cortex, attaining its peak volume at around 13.5 years (23). By contrast,

90   the cortex reaches this milestone almost 5 years earlier at approximately 8.7 years (24).

91   Based on the literature discussed, we derive and test the following predictions. The CBH predicts that overall

92   brain volume will be positively associated with lifespan, even when accounting for the effects of other life

93   history phases. While previous tests of the CBH have focused on whole brain volume, some authors have

94   suggested it may apply specifically to the neocortex, on the assumption that this region is particularly

95   implicated in aspects of behavioural flexibility (5) such as innovation (25, 26), while the cerebellum not been

96   predicted to play a major role. The DCH, in contrast, predicts that lifespan will not be positively associated

97   with brain or brain component volumes after accounting for developmental effects. Further, owing to the

98   differential allocation of developmental costs to pre- versus post-natal phases between the neocortex and

99   cerebellum respectively, the DCH predicts that post-natal developmental periods (lactation duration and

100 juvenile period) will be more strongly associated with cerebellum than with neocortex volume, and vice versa,

101 pre-natal life history phases (gestation) will be more strongly associated with neocortex than with cerebellum

102 volume. Given the markedly protracted post-natal development of the cerebellum, the DCH implies that the

103 evolution of extended post-natal development in primates may be explained by the developmental costs

104 associated with growing and maturing a large cerebellum in particular. Therefore the DCH also predicts that

105 apes, a group with substantially expanded cerebella (20), will have significantly longer post-natal

106 developmental periods than other primates, after accounting for allometric effects.

107

108 **Methods**

109

110 **Brain volume data**

111 We obtained whole brain, neocortex and cerebellum volumes (in mm³) for anthropoid species from an existing
112 compilation (20), together with additional data for non-anthropoids compiled by the same authors (originally
113 from (27, 28)), for 55 species. We analysed overall brain volume in addition to structure volumes to allow
114 comparisons with previous work which has primarily investigated the whole brain (e.g. (1, 2, 8, 9)), and to
115 give context to structure analyses in terms of how relationships between structure sizes and life history may
116 be related to overall brain size. We did not use measures from a recent comparative dataset based on MRI
117 scans (29) due to incompatible measures of neocortex volume (neocortical grey matter only in (29) versus
118 whole neocortex volume in (20)) (see (30)).

119

120 **Life history data**

121 We obtained life history data from the PanTHERIA (31) and AnAge databases (32). For most life history
122 traits – body mass (grams), gestation length (days), weaning age (days) and age at first birth (days) – we
123 prioritised data from PanTHERIA, supplementing missing values with data from AnAge where possible.
124 AnAge provides age of female sexual maturity rather than age of first birth estimates, but the two are very
125 closely correlated among species with data for both variables (PGLS: β=0.85, p<0.001, λ=0.00, n=43). For
126 lifespan data (estimated as maximum longevity), we prioritised records from AnAge due to higher data quality
127 and longer estimates for many species compared with other datasets (33). Longevity records were converted
128 from years (AnAge) or months (PanTHERIA) to days for comparability between datasets and with other life
129 history traits. Some of these life history variables represent phases of life nested within one another (i.e.
130 weaning age within age at first birth, age at first birth within lifespan). To avoid autocorrelation when
131 including multiple life history predictors in the same model, we calculated two additional life history variables
132 that do not overlap with any other for use in analyses: juvenile period and adult lifespan. We calculated
133 juvenile period length by subtracting weaning age from age at first birth, and adult lifespan by subtracting age
134 at first birth from maximum longevity.

135

136 After matching species across different datasets and to the 10ktrees primate phylogeny (34), the main sample
137 contained 48 species (excluding humans) with complete data on all life history and brain volume variables.
138 This dataset is available in the Electronic Supplementary Material (ESM data).

139

140 **Statistical analyses**

141   *Phylogenetic comparative methods*

142

143   We tested predictions using comparative statistical methods that account for the influence of phylogeny,

144   specifically phylogenetic generalized least squares (PGLS) regression using functions from the caper R

145   package (35). We used a consensus phylogeny from 10ktrees (34). Pagel's lambda (λ), a measure of

146   phylogenetic signal, was estimated by maximum likelihood. All continuous variables were log10 transformed

147   to reduce positive skew and improve fit to statistical assumptions. In all analyses, we treated brain or brain

148   structure volumes as the outcome variable and life history traits as the predictors. Additionally, in all analyses

149   we included body mass to control for allometric scaling of both brain structure volumes and life history traits

150   with body size (36–38). For analyses of structure volumes, we did not attempt to additionally control for

151   remaining brain volume for both theoretical and statistical reasons. The DCH rests on the assumption that

152   additional neural tissue, relative to body mass (reflective of energetic capacity), requires longer developmental

153   periods (9), and therefore does not make direct predictions about the size of brain components relative to one

154   another. We also focused on the size of brain components relative to body mass in order to facilitate direct

155   comparisons with prior tests of the DCH and CBH which have examined whole brain relative to body mass

156   (e.g. (2–4, 8, 9)). Further, measures of remaining brain volume are too highly correlated with body mass to

157   obtain confident estimates of the independent contributions of both remaining brain and body size to structure

158   volumes. Variance inflation factors (VIFs) were at least 15 when both body mass and remaining brain volume

159   were included as predictors of individual structures, exceeding commonly used thresholds for problematic

160   levels of collinearity (usually 5 or 10, (39)).

161

162   We tested predictions by examining coefficients reported in global models and using model comparison to

163   identify more parsimonious models. For each brain volume measure, we first fit a global model including all

164   four life history traits plus body mass as predictor variables. Model performance was deemed acceptable for

165   all global models based on visual examination of diagnostic plots. VIFs for the global model ranged from 2.04

166   to 5.20, indicating moderate to potentially problematic levels of collinearity (although thresholds vary widely

167   in practice) (39). Then for each brain measure, we created a candidate set of models using functions from the

168   R package MuMIn (40). Candidate sets consisted of the full model, a null allometric (body mass only) model

169   and models containing all possible combinations of 1-3 life history predictors (total N=16 models for each

170   structure). Body mass was included in all candidate models to account for allometric relationships with brain

171   volumes and life history variables. Comparing PGLS models is complicated by the effect of phylogeny, since

172   both changes in the predictor variables and the influence of phylogeny can affect model fit. Therefore, to

173   simplify interpretation of life history effects, we fixed λ across all models in the candidate set for a given

174   structure, to the same value as that estimated by maximum likelihood from the global model (as recommended

175   in (35)). We used Bayesian Information Criterion (BIC) rather than AIC(c) scores to rank models, as the

176   former applies a higher penalty for additional parameters and is thus better suited to identifying the most

177   parsimonious model (41). We report selected effect sizes to aid interpretation of the relative effects of life

178   history phases, to illustrate comparisons that are particularly important in relation to predictions. We compare

179   effects only between growth periods (i.e. gestation, lactation and juvenile period) as these comparisons are

180   most biologically meaningful, and only when at least one of these variables has a significant or marginal effect

181   ($p < 0.10$) in global models. Effect sizes for selected parameters were estimated by raising 10 to the power of

182   their coefficients from the global models, which gives the amount of change in the dependent variable for a

183   unit change a given predictor variable on the same scale as the data (e.g. number of additional mm^3 in

184   neocortex volume for an additional day of gestation), assuming the effects of all other predictors are held

185   constant.

186

187   *Differences between apes and other primates*

188   We assessed the potential influence of apes (hominoidea) on the relationship between the cerebellum and life

189   history variables in two different ways. First, we tested for differences in life history traits relative to body

190   mass between apes and non-apes by fitting models predicting each life history trait in turn from body mass

191   and a factor representing membership of the ape clade (following (20) which used this approach to examine

192   such 'grade shifts' in cerebellar evolution). Here, we compared models in which either both slopes and

193   intercepts, or intercepts only, are allowed to vary between apes and non-apes, using BIC scores. Second, we

194   re-ran the global cerebellum model removing ape species (n=5) from the sample. To establish whether any

195   differences in results were due specifically to removing apes versus reduced statistical power, we also re-ran

196   the model 1000 times, removing 5 random non-ape species from the sample at each iteration. If the

197   relationship between cerebellum volume and a particular life history variable is strongly contingent on the

198   apes, we should expect that it generally remains significant when 5 random non-ape species are removed, but

199   not when the 5 apes are removed.

200

201   **Results**

202

203   **Figure 1** summarises the results of global models for all brain volume measures. Global models are reported

204   in full in **Tables 1-3** while model selection tables are included in the Electronic Supplementary Material

205   (**Tables S1-S3**).

206

207

208   **Cognitive Buffer Hypothesis**

209

210   Contrary to the predictions of the Cognitive Buffer Hypothesis, we do not find a strong effect of adult lifespan

211   on whole brain volume in the global model, in which the effects of pre- and post-natal development are taken

212   into account (**Table 1**). In model comparisons, adult lifespan is retained in the best-supported model for

213   overall brain volume, but the second-ranked model, containing gestation length only, is similarly well-

214   supported (**Table S1**). We also find little support for the predictions of the CBH as applied to the neocortex.

215   Gestation length is the strongest predictor of neocortex volume in the global model, while adult lifespan has

216   little effect (**Table 2**). In model comparisons, adult lifespan is absent from the three highest-ranking models

217   (**Table S2**). We do, however, find a significant effect of adult lifespan on cerebellum volume in the global

218   model, after accounting for developmental periods (**Table 3**). In model comparisons, adult lifespan is included

219   in the model with the lowest BIC score for cerebellum volume (**Table S3**), although the second-ranked,

220   similarly supported model does not contain adult lifespan.

221

222   **Developmental Costs Hypothesis**

223

224   In the global model, gestation length is the strongest predictor of whole brain volume, while lactation and

225   juvenile period have negligible effects (**Table 1**). In model comparisons, the two highest-ranked models retain

226   gestation length, but not lactation duration or juvenile period (**Table S1**). Effect sizes from the model predict

227   that brain volume increases by $3.18mm^3$ for each additional day of gestation, while by $1.17mm^3$ for each

228   additional day of lactation, for a species with average life history traits and body mass. Gestation length is the

229   only near-significant predictor of neocortex volume in the global model, while lactation and juvenile period

230   have little to no effects (**Table 2**). Gestation length is the sole predictor included in the top-ranked model for

231   neocortex volume, although the second highest ranking model contains both gestation length and weaning age

232   (**Table S2**). Effect sizes predict a greater increase in neocortex volume for each additional day of gestation

233   ($4.11mm^3$) than lactation ($1.30mm^3$), all else being equal. Conversely, cerebellum volume significantly

234   increases with juvenile period in the global model, while gestation and lactation have negligible effects (**Table

235   3**). Juvenile period is retained in the 5 top-ranked models for cerebellum volume, and is the sole predictor in

236   the second-highest ranked model (**Table S3**). Effect sizes from the global model suggest that for an average

237   species, every additional day of life prior to first reproduction is associated with an increase of $1.85mm^3$ in

238   cerebellum volume. We obtain estimates of zero phylogenetic signal in global models of cerebellum volume

239   (**Table 3**). While a lack of phylogenetic signal is unexpected for evolutionarily conserved traits such as brain

240   structure volumes, we show in supplementary analyses that this is unlikely to be the result of statistical

241   artefacts (**ESM Appendix**).

242

243

244 *Differences between apes and other primates*

245 Relative to their body sizes, apes have significantly longer lactation and juvenile periods, and marginally

246 longer adult lifespans, than non-apes (**Table 5, 6, 7; Figure 2, 3**). In contrast, apes do not significantly differ

247 from non-apes in relative gestation time (**Table 4**). For all life history variables, BIC scores favoured intercept-

248 only models over those in which both slopes and intercepts were allowed to vary between the clades (**Table**

249 **8**). When re-running the global cerebellum model without apes (n=43), the association between juvenile

250 period and cerebellum volume becomes non-significant (**Table 9**). However, removing 5 randomly selected

251 non-ape species from the sample also often results in a weakened relationship: p-values for juvenile period

252 are 0.05 or greater in 64.7% of 1000 iterations. This suggests that the relationship between juvenile period

253 and cerebellum volume is not solely contingent on the ape clade.

254

255 **Discussion**

256

257 Rather than a general extension of lifespan in large brained species, we find that specific aspects of life history

258 are correlated with the volumes of different structures according to their developmental trajectories. Our

259 results are therefore primarily consistent with predictions of the Developmental Costs and Expensive Brain

260 hypotheses rather than the Cognitive Buffer Hypothesis, in that correlations between lifespan and brain size

261 or neocortex volume appear to be by-products of the relationship of these structures with developmental

262 periods. In support of the Developmental Costs Hypothesis more specifically, we find that brain structures

263 with different emphases on pre-versus post-natal growth show predicted associations with those periods of

264 investment. Maternal investment, specifically pre-natal investment, has an independent relationship with the

265 relative volume of the neocortex. In contrast, cerebellum volume has an independent positive correlation with

266 juvenile period length, congruent with the idea that interaction with the environment during maturation

267 provides crucial input to the development of this structure, through play, for example. In summary, the

268 correlation between brain size and life history in primates may require no specific adaptive explanation,

269 instead reflecting the developmental mechanisms by which enlarged brains and brain components evolve.

270

271 **Cognitive buffer hypothesis**

272

273 We do not find evidence of a strong, direct correlation between brain size and lifespan, contradicting a central

274 prediction of the CBH. Rather, the association between the two is weak when controlling for other life history

275 phases. This finding is consistent with the interpretation that the brain size-lifespan correlation is confounded

276 by the duration of maternal investment, as previously found across mammals (9) and more recently confirmed

277 for primates in particular (42). We also find no evidence to support a direct association between lifespan and

278 neocortex volume, contradicting the idea that the neocortex plays a key role in extending lifespan via

279 behavioural flexibility (5). We do find, however, an independent positive association of the cerebellum with

280 adult lifespan, unanticipated by prior literature on the CBH, which has focused on whole brain or neocortex

281 volume. Our findings could therefore be construed as consistent with a cognitive buffering effect specifically

282 of the cerebellum. In support of this interpretation, the cerebellum is increasingly recognized to play a role

283 not only in fine motor control and coordination but a wide diversity of cognitive functions including working

284 memory, planning and decision-making (18). However, this finding is also consistent with the 'Neuronal

285 Investment' hypothesis, which posits that longer-lived animals require larger brain volumes to compensate

286 for longer periods of decline in neuronal function over their lifetimes (5). The cerebellum in particular may

287 be implicated due its potentially greater susceptibility to neuronal loss with age compared with other structures

288 (5). Further evidence, most crucially a relationship between cerebellum volume and survival, is thus required

289 to distinguish between different explanations for this finding.

290 While we find little support for the CBH in terms of a direct link between overall brain size or neocortex size

291 and lifespan in primates, our findings do not exclude the possibility that this hypothesis is supported by other

292 lines of evidence, and in other taxonomic groups. For example, primate species with larger brains experience

293 less variation in net energy intake than expected based on environmental seasonality compared with smaller-

294 brained species, consistent with a cognitive buffering effect (43). Prior comparative work suggests that the

295 extent to which brain size correlates with lifespan may vary between clades, finding greater support for the

296 prediction among haplorrhine than strepsirrhine primates (1). A more recent comparative study finds support

297 for the correlation within primates and rodents, but not other mammalian orders (3). Neither of these studies,

298 however, accounted for the effects of developmental periods and therefore do not directly test the DCH. In

299 birds, cognitive buffering effects are consistent with the findings that relatively large-brained species have

300 lower adult mortality rates (44), experience more environmental variation (45) and have more stable

301 populations in variable environments (46). Directly comparable tests of the DCH in primates and birds would

302 therefore be a productive avenue of future research.

303

304

Developmental costs hypothesis

306

307 Our findings suggest that the relationship between lifespan and both overall brain volume and neocortical

308 volume in primates is a by-product of maternal investment, primarily in pre-natal, rather than post-natal,

309 offspring development. This result likely reflects the predominant role of pre-natal investment in neocortical

310 growth specifically, given that this structure accounts for such a large proportion of overall brain volume.

311 Consistent with this interpretation, and as predicted from the fact that neurogenesis and a relatively large

312 proportion of neocortical growth is completed before birth, the life history variable most strongly associated

313 with adult neocortex size was gestation length. Our results suggest that evolutionary expansion of the

314 neocortex is supported primarily by increased pre-natal investment, while cerebellar expansion requires

315 greater investment in post-natal development. Despite the shared functional roles and correlated evolution of

316 the cerebellum and neocortex (18–20, 47–49), a degree of independent, or mosaic, evolution of the two

317 structures has been documented (20). Our results thus provide developmental mechanisms for the expansion

318 of the neocortex and cerebellum, complementing evidence of distinct genetic mechanisms supporting such

319 mosaic evolution (17). The patterns may be yet more complex, however: since the neocortex is composed of

320 many heterogeneous systems, an interesting avenue for future work would be to investigate whether specific

321 neocortical components or tissue types correlate with different aspects of maternal investment. Indeed,

322 developmental scheduling varies across the neocortex, with occipital grey matter maturing earlier than that in

323 the prefrontal cortex (50). Those specific areas and tissues which continue to grow post-natally may therefore

324 be associated with post-natal developmental phases including lactation and juvenile period.

325 Adult cerebellum volume correlated positively with post-natal (juvenile) development, after accounting for

326 variation in other life history phases. This pattern fits with evidence indicating late volumetric growth and

327 maturation of this structure, extending through infancy and beyond in humans (23, 51, 52). At a cellular level,

328 the post-natal genesis of the majority of cerebellar granule cells followed by synaptogenesis indicates high

329 functional plasticity during this time, making environmental stimuli potentially critical in cerebellar

330 maturation (22). Further evidence for the importance of environmental input in cerebellar development

331 includes the low heritability of cerebellum volume compared to that of other brain structures (53), and effects

332 of an impoverished post-natal environment on the volume of superior-posterior cerebellar lobes (54). Infancy

333 and juvenility are periods of social learning, practice and play in an environment of reduced risk (55).

334 Behaviourally, play is correlated with cerebellum volume (56) and with the volume of structures comprising

335 the cortico-cerebellar system (57) across primates, and within species there are concurrent increases in the

336 rate of play and formation of cerebellar synapses during post-natal development (58). The correlation between

337 play and both post-natal brain growth and behavioural flexibility in primates is thus likely to involve cerebellar

338 maturation (59, 60). Converging lines of evidence therefore suggest that many environmental influences on

339 post-natal learning and development may be mediated by effects on the cerebellum.

340

341 *Cerebellar expansion and extended maturation time in apes*

342 When apes were removed from the PGLS analyses, the effect of juvenile period duration on cerebellum

343 volume became non-significant. However, the same was true in the majority of cases when 5 random non-ape

344 species were removed from the analyses, suggesting that this may be due to a loss of statistical power rather

345 than contingency of results on the ape clade. We do, however, find that apes have a distinct life history profile

346 compared to other primates, with significantly longer lactation and juvenile periods and marginally longer

347   adult lifespans. Together with prior evidence of accelerated cerebellar expansion in the apes (20), these results

348   suggest that apes may have evolved extended post-natal maturation in part due to the need to invest in

349   development of a large cerebellum and the time required for its experience-dependent maturation. The absence

350   of a difference in relative gestation duration between the apes and other primates further suggests that ape life

351   histories are distinct specifically in terms of their post-natal developmental trajectories. Consistent with this

352   interpretation, cerebellar expansion in the apes is largely driven by enlargement of the cerebellar hemispheres:

353   late-developing structures that are strongly implicated in the organisation and control of complex motor

354   patterns (61, 62). Together, these results may help to explain the combination of unusually large cerebella

355   (20) extended periods of immaturity (63) delayed locomotor independence (64), and high levels of social

356   learning (65), play (66), extractive foraging and tool use (20) that characterises the ape clade. Future

357   comparative analyses could investigate whether similar developmental profiles help explain independent

358   cerebellar expansion in other mammalian lineages, such as elephants and cetaceans (67).

359

360

361   **Conclusion**

362

363   Developmental costs appear to provide the best explanation for the pattern of correlations between primate

364   brain structures and life history. The central prediction of the Cognitive Buffer Hypothesis was not supported

365   by strong, direct associations of lifespan and whole brain or neocortex volume; instead, these structures

366   correlated most strongly with gestation length. The cerebellum does correlate with lifespan after accounting

367   for developmental periods, consistent with an unanticipated cognitive buffering effect of this structure in

368   particular, although alternative explanations are possible. Overall, we provide the first evidence that primate

369   brain components exhibit distinct life history correlates that are congruent with their divergent developmental

370   profiles. These divergent patterns support the view that selection on particular functional capacities can result

371   in mosaic brain evolution mediated by complex developmental mechanisms (68).

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544 **TABLES**

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546 **Table 1: global model results for brain volume**

Parameter	Estimate	S.E.	T-value	p-value
Intercept	0.28	0.72	0.39	0.70
Gestation	0.50	0.28	1.82	0.08
Lactation	0.07	0.07	0.97	0.34
Juvenile period	0.10	0.10	1.00	0.32
Adult lifespan	0.22	0.14	1.59	0.12
Body mass	0.51	0.05	10.10	<0.001

547 *Table 1: Results of the global model for brain volume, predicted by all four life history traits and body mass (N=48, R^2=0.88, λ=1).*

548

549 **Table 2: global model results for neocortex volume**

Parameter	Estimate	S.E.	T-value	p-value
Intercept	-0.19	0.83	-0.23	0.82
Gestation	0.61	0.32	1.93	0.06
Lactation	0.12	0.08	1.39	0.17
Juvenile period	0.08	0.11	0.76	0.45
Adult lifespan	0.18	0.16	1.13	0.27
Body mass	0.53	0.06	9.07	<0.001

550 *Table 2: Results of the global model for neocortex volume, predicted by all four life history traits and body mass (N=48, R^2=0.87, λ=1).*

551

552 **Table 3: global model results for cerebellum volume**

Parameter	Estimate	S.E.	T-value	p-value
Intercept	-0.94	0.65	-1.44	0.16
Gestation	0.08	0.22	0.39	0.70
Lactation	0.13	0.10	1.29	0.20
Juvenile period	0.27	0.13	2.01	0.05
Adult lifespan	0.32	0.16	2.06	0.05
Body mass	0.58	0.05	11.03	<0.001

553 *Table 3: Results of the global model for cerebellum volume, predicted by all four life history traits and body mass (N=48, R^2=0.96, λ=0).*

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558   **Table 4: comparison of gestation length in apes versus non-apes**

Parameter	Estimate	S.E.	T-value	p-value
Intercept	1.93	0.08	25.55	<0.001
Body mass	0.09	0.02	4.26	<0.001
Ape vs. non-ape	0.08	0.07	1.19	0.24

559   *Table 4: Results of the intercept-only model comparing gestation length relative to body mass in ape versus non-ape species (N=48, R^2=0.34, λ=1).*

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561   **Table 5: comparison of lactation duration in apes versus non-apes**

Parameter	Estimate	S.E.	T-value	p-value
Intercept	0.91	0.15	6.12	<0.001
Body mass	0.40	0.05	8.61	<0.001
Ape vs. non-ape	0.27	0.11	2.46	0.02

562   *Table 5: Results of the intercept-only model comparing lactation duration relative to body mass in ape versus non-ape species (N=48, R^2=0.75, λ=0.05).*

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564   **Table 6: comparison of juvenile period in apes versus non-apes**

Parameter	Estimate	S.E.	T-value	p-value
Intercept	2.32	0.13	18.58	<0.001
Body mass	0.19	0.04	4.84	<0.001
Ape vs. non-ape	0.24	0.09	2.56	0.01

565   *Table 6: Results of the intercept-only model comparing juvenile period length relative to body mass in ape versus non-ape species (N=48, R^2=0.53, λ=0.26).*

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567   **Table 7: comparison of adult lifespan in apes versus non-apes**

Parameter	Estimate	S.E.	T-value	p-value
Intercept	3.56	0.09	40.45	<0.001
Body mass	0.12	0.03	4.51	<0.001
Ape vs. non-ape	0.13	0.07	1.96	0.06

568   *Table 7: Results of the intercept-only model comparing adult lifespan relative to body mass in ape versus non-ape species (N=48, R^2=0.52, λ=0).*

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577 **Table 8: BIC scores for models comparing life history traits between apes and non-apes**

Outcome variable	Intercept-only	Different slopes
Gestation	-121.79	-118.06
Lactation	-11.80	-10.21
Juvenile period	-37.60	-35.26
Adult lifespan	-59.57	-56.47

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580 *Table 8: BIC scores for models comparing gestation, lactation, juvenile period and adult lifespan relative to body size between apes and non-apes. In intercept-only models, only intercepts were allowed to vary between apes and non-apes, while in different slopes models, both intercepts and slopes were allowed to vary between apes and non-apes.*

581

582 **Table 9: global model results for cerebellum volume without ape species**

Parameter	Estimate	S.E.	T-value	p-value
Intercept	-0.67	0.72	-0.92	0.36
Gestation	0.09	0.23	0.38	0.70
Lactation	0.09	0.11	0.79	0.44
Juvenile period	0.25	0.15	1.70	0.10
Adult lifespan	0.28	0.17	1.67	0.10
Body mass	0.59	0.06	10.19	<0.001

583 *Table 9: results repeating the global model for cerebellum volume without ape species (N=43, R^2=0.93, λ=0).*

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601   **FIGURE CAPTIONS**

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603   **Figure 1: effects of life history predictors on brain structure volumes from global models**

604   *Figure 1:* PGLS regression coefficients (points) with 95% confidence intervals (whiskers) for life history predictors of brain structure volumes, from global models.
605   *Dashed vertical lines indicate zero. Each row corresponds to a separate global model in which the brain structure volume on the Y axis is the outcome variable,*
606   *predicted by the four life history traits on the X axis plus body mass (not shown).*

607

608   **Figure 2: relative lactation duration in apes versus non-apes**

609   *Figure 2:* raw data (points) and regression slopes (lines) from a model predicting lactation duration from body mass, fitting separate intercepts for ape (green) and
610   *non-ape species (blue). Apes have significantly longer lactation periods relative to their body size than do non-apes.*

611

612   **Figure 3: relative juvenile period length in apes versus non-apes**

613   *Figure 3:* raw data (points) and regression slopes (lines) from a model predicting juvenile period length from body mass, fitting separate intercepts for ape (green)
614   *and non-ape species (blue). Apes have significantly longer juvenile periods relative to their body size than do non-apes.*

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