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RESEARCH ARTICLE

Multiple Components of Phylogenetic Non-stationarity in the Evolution of Brain Size in Fossil Hominins

José Alexandre Felizola Diniz-Filho[1] · Lucas Jardim[2] · Alessandro Mondanaro[3] · Pasquale Raia[3]

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Abstract

One outstanding phenotypic character in *Homo* is its brain evolution. Pagel (Morphology, shape and phylogeny, CRC Press, Boca Raton, 2002) performed a phylogenetic analysis of the evolution of cranial capacity (as a surrogate of brain size) in fossil hominins, finding evidence for gradual evolutionary change with accelerating rate. Since Pagel's pioneering investigation, the hominin fossil record expanded backward in time, new species were added to our family tree, different phylogenetic hypotheses were advanced, and new phylogenetic comparative methods became available. Therefore, we feel it is timely to repeat and expand upon Pagel's seminal paper by including such material and applying novel methodologies. We fitted several evolutionary models to the endocranial volume (ECV) for 21 fossil hominins (including Pagel's original analyses) and estimated phylogenetic signal using different approaches, while accounting for phylogenetic uncertainty. We then applied the phylogenetic signal-representation curve to the data to look for non-stationarity (discontinuities, rate shifts, or presence of different evolutionary patterns in different parts of the phylogeny) in brain size evolution. Our analyses show that, in principle, Pagel's findings are robust to the addition of new data and phylogenetic uncertainty and confirm both the strong phylogenetic signal in brain size and acceleration of ECV evolutionary rates towards the present. However, non-stationarity was also detected in about 11% of the simulations, with two significant evolutionary discontinuities occurring close to the origin of the *H. sapiens* lineage (*H. sapiens, H. neanderthalensis, H. heidelbergensis* and *H. antecessor*) and along the phyletic line leading to *H. floresiensis*. This study calls upon further investigation of these important moments in *Homo* evolution, in order to understand the processes underling each of these shifts in brain size evolutionary regimes.

Keywords Phylogenetic comparative methods · Evolutionary models · Endocranial volume · Non-stationarity · Adaptive evolution · Hominins

Introduction

One of the most outstanding character changes in the evolution of *Homo* is the acquisition of a very large brain, which in our own species is more than three times as large as in our closest living relatives, the chimpanzees (González-Forero and Gardner 2018; Herculano-Houzel 2012). Brain power is what makes us different from any other living being. This makes it unsurprising that the pattern of brain size evolution in the human lineage attracts so much scientific interest. Recent analyses of such pattern span across several hierarchical levels, from refined quantitative genetic analyses at the population level, to the comparison of closely-related species' brain size (e.g., Diniz-Filho and Raia 2017; Gómez-Robles et al. 2017; Grabowski 2016; Montgomery et al. 2010; Schroeder and von Cramon-Taubadel 2017; von Cramon-Taubadel 2014).

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✉ José Alexandre Felizola Diniz-Filho
diniz@ufg.br

[1] Departamento de Ecologia, Instituto de Ciências Biológicas (ICB), Universidade Federal de Goiás (UFG), Goiânia, GO, Brazil

[2] Programa de Pós-Graduação em Ecologia & Evolução, ICB, UFG, Goiânia, GO, Brazil

[3] Dipartimento di Scienze della Terra, dell'Ambiente e delle Risorse, Università di Napoli Federico II, Napoli, Italy

Other studies discussed the adaptive mechanisms and potential constraints operating upon brain size variation among hominins (e.g., Leonard et al. 2003; Fischer and Mitteroecker 2015; Gómez-Robles et al. 2013; Herculano-Houzel 2012; Holloway 2015; Montgomery et al. 2016; Navarrete et al. 2011; Schoenemann 2013; Du et al. 2018).

One of the earliest, landmark applications of phylogenetic comparative methods to the evolution of human brain size was performed by Pagel (2002), who fitted several evolutionary models to the human phylogenetic tree, to find that brain size in hominins followed a gradual (i.e., not punctuated) trend towards larger size, that accelerated towards the recent (see Pilbeam and Gould 1974; Ruff et al. 1997 for non-phylogenetic studies reaching similar conclusions). However, since Pagel's pioneering analysis, a wealth of new data and approaches have become available. First, the human fossil record fleshed out thanks to the discoveries and descriptions of at least five new species (*Homo floresiensis, Homo naledi, Australopithecus sediba, Orrorin tugenensis*, and *Sahelanthropus tchadensis*). In addition, several forms of *Homo erectus* are now recognized by different authors (see Baab 2016a for a recent review) which means the information at hand is now much expanded over Pagel's study. Secondly, *Orrorin* and *Sahelanthropus* stretched back the origin of the hominin lineage to some 7 million years ago, that is much earlier than the root of the hominin tree set at about 3.1 mya in Pagel's study. The increased number of species in the hominin tree implies the number of possible alternative phylogenetic trees expanded considerably. It is now clear that human evolution cannot be described according to any simplistic sequence of chronologically disjunctive species evolving by simple anagenesis (e.g. as in the sequence *H. habilis-H. erectus-H. heidelbergensis* and finally *H. sapiens;* see Foley et al. 2016). Also, currently known species cannot be satisfactorily aligned in a "comb-like" phylogeny, as in Pagel's (2002) paper (e.g., Argue et al. 2017; Dembo et al. 2015, 2016). The fossil record tells that there were often a number of coexisting species, and survival of "primitive" and small-brained forms into the recent, such as *H. naledi* and *H. floresiensis* (see Montgomery 2018).

All these factors suggest caution when dealing with any phylogenetic reconstruction of the human tree and, more importantly, that brain size evolution must have proceeded according to a complex, non-linear pattern of evolution (Du et al. 2018). Working in a non phylogenetically-explicit context, the analyses by Pilbeam and Gould (1974) and Ruff et al. (1997) already pointed out that brain size evolution did not follow the same scaling patterns in *Homo* and *Australopithecus* (with early *H. habilis* and *H. rudolfensis* assuming an intermediate position; see Ruff et al. 1997) and that some stasis in relative brain size accrues to hominin brain evolution in between 1.5 and 0.5 mya.

Phylogenetic comparative methods work by describing patterns of trait evolution in reference to alternative, low-dimensional heuristic evolutionary models (i.e., Hansen and Martins 1996; see; Pennell and Harmon 2013 for a review). However, it is now well-recognized that trait evolution often is much more complex, with evolutionary rates changing across the phylogeny, forming complex non-stationary patterns in a phylogeny (sensu Diniz-Filho et al. 2010). It is now possible to take such complexity into account, developing models that allow evolutionary rates and phenotypic means change across the tree (Beaulieu et al. 2012; Butler and King 2004; Castiglione et al. 2018; Diniz-Filho et al. 2012; Eastman et al. 2011; Hansen et al. 2008; O'Meara et al. 2006; Rabosky et al. 2014). Exploratory statistical methods, such as the PSR curve, allows detecting where non-stationarity is located on the tree (Diniz-Filho et al. 2012, 2015). This approach may be particularly well-suited to the study of hominin brain size evolution, as this has often been described as a complex pattern which is hard to reduce to any low-dimensional evolutionary model (Falk et al. 2000; Grabowski 2016; Shultz and Maslin 2013) and further complicated by the unique suite of functions *Homo* brain acquired, which suggests population level processes within *Homo* cannot be extended to the entire hominin tree, which would be problematic anyway (see Jablonski 2017a, b for a recent review).

With so much new material and approaches to explore, it is surprising that the recent literature on *Homo* brain size evolution has not focused even more directly on the issue of the tempo and mode of brain size evolution under a phylogenetically-explicit context. For instance, two recent papers fitted evolutionary models to brain size variation in fossil hominins using distinct data and approaches. Gómez-Robles et al. (2017) analyzed endocranial and dental size and shape variation in hominins by fitting a multiple-variance Brownian motion model to reconstruct ancestral states and compare observed and neutral divergence along distinct parts of the tree. They showed that brain size evolutionary rates depart from neutral evolution and accelerate towards the present (as found by Pagel 2002 original paper). Du et al. (2018) fitted time-series to brain size data at progressively less inclusive (lower) taxonomic levels (so they approach is phylogenetically implicit), which allowed decoupling anagenetic and cladogenetic components of change. They showed that local episodes of directional selection coupled with periods of stability in mean brain size (stasis, in the context of punctuated equilibrium theory, or random drift either) describe cranial capacity evolution in hominins better than any simpler acceleration model. Du et al. (2018) also pointed out that changes in brain size are better explained by anagenetic trends than by lineage sorting (although this last process may be also important along some branches of the phylogeny). Because this very complex pattern occurs over a short time

scale, the overall trend mimics gradual evolution, especially when multiple lineages are pulled together. These findings cast doubts on Pagel's original finding, and further call for explicit non-stationary models to be tested.

Here we revisited patterns of brain size evolution in fossil hominins, using the new phylogeny provided by Dembo et al. (2015, 2016) as a backbone but accounting for phylogenetic uncertainty by taking the stratigraphic range of species in consideration (Bapst 2012). We fitted several evolutionary models to endocranial volume (ECV) and repeated Pagel's (2002) original analyses after including new data and estimated the intensity of the phylogenetic signal using different approaches. Moreover, we also applied the phylogenetic signal-representation (PSR) (Diniz-Filho et al. 2012) curve to look for non-stationarity (discontinuities, rate shifts and different evolutionary models pertaining to different parts of the phylogeny), developing a new approach to search for non-stationarity that allowed us distinguishing three major shifts in human brain size evolution taking into account phylogenetic uncertainty.

Methods

Data

Brain size data, expressed as Endocranial Volume (ECV, in cc) were assembled from several sources (e.g., Schoenemann 2013; Rightmire 2013; Holloway 2015; Berger et al. 2015; Grabowski et al. 2015, 2016; Grabowski 2016; Hawks et al. 2017; Holloway et al. 2018; see electronic supplementary material Tables S1 and S2). In each simulation (see below), we added a random value to the log-transformed mean ECV, sampled from a normal distribution with mean 0 and standard deviation of 0.12 (which corresponds to a coefficient of variation of about 12% on the original scale; see Charvet et al. 2013), accounting for uncertainty in mean estimation of brain size for each species. Following Du et al. (2018), we focused on absolute, rather than relative (to body size) brain size, both because relative brain size introduces additional measurement error to the analyses (because of the uncertainty in body size estimates) and because of the biological significance of absolute brain size (i.e. using ECV as a proxy) as a surrogate for cognitive power (see Herculano-Houzel 2012; Gómez-Robles et al. 2017). Moreover, as recently pointed out by Grabowski (2016), adaptive evolution by natural selection for increasing brain size is sufficient to explain both brain size and body size (by correlated evolution) along most, if not all, major transitions in human evolution.

We used as our backbone phylogeny the consensus hominin tree in Dembo et al. (2015, 2016), but collapsed nodes with low support (Fig. 1a). Starting from this

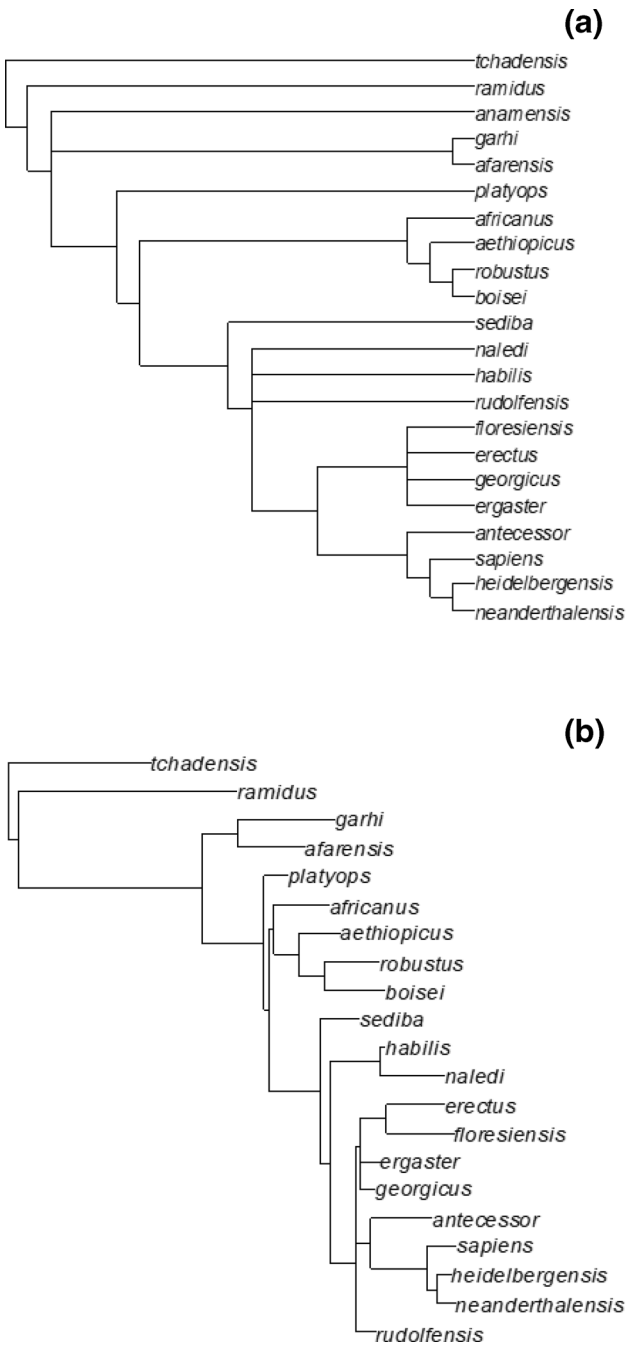

Fig. 1 **a** Basic relationship among 21 fossil hominin species, based on Dembo et al. (2015, 2016); **b** A phylogeny showing an example on how the polytomies are resolved and how the tree is scaled based on the temporal span of each species, in keeping with Bapst (2012)

"base-phylogeny", we used the *paleotree* package by Bapst (2012) to produce 1000 alternative trees, using the temporal fossil range of the species as time span, obtained mainly from Wood and Lonergan (2008) and Wood (2010), but updated for a few species (e.g., Hublin et al. 2017; van den Bergh 2016; Dirks et al. 2017). When only point estimates are available (i.e., *H. georgicus*, around 1.7 my), we assigned

a small variation of about 10% around these estimates. We set the speciation and extinction rates to be 0.1 and 0.05, respectively, and the sampling rate at 0.5 as in *paleotree* (see Fig. 1b for an example). Preliminary tests showed that changing these parameters did not qualitatively affect our results. All comparative analyses (below) were repeated by using the replicated trees, which incorporates phylogenetic uncertainty associated with branch lengths and low-support nodes. For *Homo erectus* sensu lato (s.l.), we used means of individual specimens based on Schoenemann (2013) and Rightmire (2013) and specific reviews (e.g., Bruner et al. 2015; Baab 2016a). We opted to do so because information for this species is often clumped (as *Homo erectus sensu lato*, or African *Homo erectus* and Asian *H. erectus*), and we had to match it with Dembo's et al. (2015, 2016) tips of the phylogeny, which includes several species or forms within the *H. erectus* hypodigm (i.e., *H. erectus, H. ergaster* and *H. georgicus*, referring to classical forms of Asian—Java and China-, African and the older and morphologically distinct Dmanisi fossils, respectively).

Evolutionary Models

We started by fitting alternative evolutionary models to brain size data, including the same set of models as in Pagel (2002). We used the *fitContinuous* function in the R package *Geiger* (Harmon et al. 2008) to fit the following models: Brownian motion (BM), Ornstein–Uhlenbeck (OU), Early Burst (EB), trend (T), lambda (λ), kappa (κ), delta (δ) and white noise (random) evolutionary models. The idea underlying fitting these models is to scale the branch lengths of the phylogenies according to expected variances of quantitative trait evolution and then use a maximum-likelihood inference to select which model fits best trait covariation among species (Hansen and Martins 1996, Pagel 1999; O'Meara et al. 2006, Harmon et al. 2008). The Lambda (λ) model specifically fitted by Pagel (2002), assumes species traits evolve gradually with constant evolutionary rates, and is widely used as a metric for phylogenetic signal (see Freckleton et al. 2002, Hansen et al. 2008). Higher values of Lambda (i.e., close to 1) indicate that species phenotypic variance is proportional to time to coevolution. The Delta model (fits a δ parameter, called by Pagel (2002) a "path-length scaling parameter"), also models a gradual evolution, but evolutionary rates may change over time, either accelerating ($\delta > 1$) or decelerating ($\delta < 1$). Finally, the Kappa model (κ parameter) allows trait to evolve by a continuum of anagenetic ($\kappa = 1$) and cladogenetic ($\kappa = 0$) processes. In a purely cladogenetic process all trait evolution concentrates at speciation, expressing a perfect punctuated equilibrium model (Pagel 1999, 2002, Hansen and Martins 1996). Due to the small sample size and the non-ultrametric tree, fitting more complex models with multiple adaptive peaks (i.e., OUWie; Beaulieu et al. 2012) did not work out and failed to converge in most phylogenies, except for the simplest model of Brownian with distinct evolutionary rates in the two groups (i.e., with a shift in the node representing the origin of *Homo*), which is included among the models we tested. This model represents changes in adaptive landscape enabling traits to evolve at different rates than their ancestors.

Model fit was compared using Akaike Information Criterion (AICc), with distinct models assigned if $\Delta\text{AICc} > 2$ in respect to smallest AICc. Akaike weighting was obtained by relative values of $\exp(-0.5 \times \Delta\text{AICc})$, expressing the probability that the selected model is the best among the candidate models, given the data (Burnham and Anderson 2002).

As pointed out above, the δ model fitted by Pagel (2002) allows testing for acceleration in evolutionary rates, but it is now also possible to evaluate whether acceleration is related to higher diversification rate (i.e., speciation minus extinction) of lineages that vary in a quantitative trait (e.g., ECV) and there are currently very sophisticated models for this, in particular BAMM (Bayesian Analysis of Macroevolutionary Mixtures; Rabosky 2014). However, these approaches usually require an ultrametric tree, so they will not work in phylogenies with fossils. Thus, we applied here an alternative and simpler approach proposed by Freckleton et al. (2008), in which diversification rate at each tip of the phylogeny is approximated by the number of nodes divided by the length of the tree, from the root up to each species. This species-specific rate can then be correlated with the trait of interest (i.e., ECV) using a phylogenetic regression, and Freckleton et al. (2008) used a phylogenetic generalized least-squares (PGLS) to test the relationship (using the fitted evolutionary models described above to incorporate species non-independence in the model residuals). Here we also applied the phylogenetic eigenvector regression (PVR) to decouple the effects of phylogenetic similarity and diversification rates explaining variation in ECV along the species (Desdevises et al. 2003; see below).

Phylogenetic Eigenvectors and the Components of Non-stationarity

We used phylogenetic eigenvector regression (PVR) (Diniz-Filho et al. 1998, 2014 for a review of applications in paleobiology, and; see; Guénard et al. 2013 for a related approach) to analyze phylogenetic signal and phylogenetic non-stationarity in ECV. The basic idea with PVR is to convert a pairwise patristic distance among species, derived from a phylogeny, into a series of phylogenetic eigenvectors by using Principal Coordinate Analysis (PCOA) (see Legendre and Legendre 2012). Single eigenvectors describing the most important phylogenetic relationships associated with variation in species' data were defined by significant ($P < 0.05$) correlation with ECV and could be used as explanatory

variables in a multiple regression model (with EVC as the response). The coefficient of determination (R^2) of PVR is then an estimate of the magnitude of phylogenetic signal in the data, expressing how much phenotypically similar closely related species are (Diniz-Filho et al. 2012). Also, PVR residuals indicate which species have unusual ECV given its phylogenetic position.

We also compared PVR's R^2 with Blomberg's K (Blomberg et al. 2003), which is another commonly used metric for phylogenetic signal. Blomberg's $K = 1.0$ indicates that a trait is evolving as expected by Brownian motion, in which trait covariance among species matches the branch lengths of the phylogeny (thus equivalent to $\lambda = 1$). Values of Blomberg's $K < 1$ indicates smaller than expectated traits covariance (i.e. low phylogenetic signal), whereas a $K > 1$ indicates that trait diverges more than expected by Brownian motion.

We also used PVR to correlate ECV with diversification using the approach proposed by Freckleton et al. (2008) described above, but using the phylogenetic eigenvectors as covariates. We then used a partial regression (Legendre and Legendre 2012) to estimate the amount of variation in ECV that could be explained by diversification rates along the branches of the phylogeny independent of the phylogenetic structure in data, expressed by the phylogenetic eigenvectors (see Desdevises et al. 2003).

The main limitation of PVR is the proper selection of eigenvectors, which may be problematic under Brownian motion exactly because phylogenetic eigenanalysis returns vectors of low discriminatory power (Rohlf 2001; Freckleton et al. 2011). Diniz-Filho et al. (2012) showed that a plot of R^2 of multiple PVRs, each one describing trait evolution by a sequentially increasing number of eigenvectors, provides a continuous description of how trait changes along the phylogeny. This 'phylogenetic signal-representation' (PSR) curve is linear under Brownian motion (for the same reason as with the low discrimination between phylogenetic eigenvectors). Deviations from linearity thus indicate deviation from Brownian motion (for instance, the pattern becomes curvilinear under OU or AC/DC models). Moreover, the PSR curve may reveal more complex forms, under non-stationary patterns of trait evolution, with shifts in R^2 occurring when specific eigenvectors (which associate to specific nodes in the tree) are added to the multiple regression model. Because, as pointed out above, eigenvectors describe different parts of the phylogeny, mapping the eigenvector in which the shift is detected allows detecting parts of the phylogeny where rate deviates from the neutral expectations. The statistical significance of the increase in R^2 can be evaluated by randomization, contrasting real differences in R^2 (ΔR^2) with the expected distribution of ΔR^2 obtained under Brownian motion (Diniz-Filho et al. 2015).

However, because of the stochasticity generated by using Bapst (2012) approach to generate many alternative phylogenies, as well as small random values added to ECV, it is necessary to develop a new approach describing such potential shifts over multiple, topologically-inconsistent phylogenies. To this aim, we counted the proportion of simulations where significant (at $P < 0.05$) shifts were detected, based on 1000 iterations, and, if this was the case, retained the eigenvector involved in the shift. This eigenvector consistently relates to a node parent to the same group of species across phylogenies, so that it is always possible to compare eigenvectors from multiple phylogenies (which therefore describe differences in the evolutionary rate among different regions of the phylogeny). To compare the similarity among the non-stationary eigenvectors, we used several ordination and clustering techniques, including K-means clustering and Metric Muldimensional Scaling (i.e., converging to an optimized Principal Coordinate Analyses) (Legendre and Legendre 2012). This allows understanding similarity patterns among the non-stationary phylogenetic eigenvectors (i.e., those related to the shifts), providing thus a synthetic description of the phylogenetic eigenvectors that captured shifts in trait evolution that can be mapped on the base phylogeny.

All analyses were performed using several packages in the R plataform 3.5.1 (R Core Team 2018), with data and script available in the electronic supplementary material.

Results

In some 56% of the simulations, the best-fit model for ECV was Delta, with average AIC weight equal to 0.372. The median value of δ was 2.782, with 95% non-parametric confidence interval ranging from 1.543 to 2.999 (Fig. 2a; see electronic supplementary material Fig. S1 for a visual evaluation of the branch length distribution of this best fit model). The second-best model was the double-peak Brownian motion (in 30% of the simulations), with a mean AIC weight equal to 0.261. The other two model parameters tested by Pagel (2002) were λ, whose mean value across simulations was 0.979 (95% CI ranging from 0.879 to 1.000) (Fig. 2b) and κ, which has a mean of 0.581 across the simulations, but with a widely bimodal distribution with peaks at 0 and 1 (i.e. the lower and upper bounds, respectively; Fig. 2c). The number of simulations with $\kappa > 0.9$ (upper bound) is larger (ca. 31.2%) those with $\kappa < 0.1$ (about 18%).

According to the PVR models, on average 64% of variation in ECV is explained by phylogenetic eigenvectors (Fig. 3a), and the PVR's R^2 is significantly correlated with Blomberg's K ($r = 0.366$; $P < 0.01$), whose median value was equal to 1.52 (Fig. 3b). In general, these two methods (as well as a λ close to 1) reveal a strong phylogenetic signal in data. From the PVR, the largest negative residual in ECV was estimated for *H. floresiensis* (standardized residuals

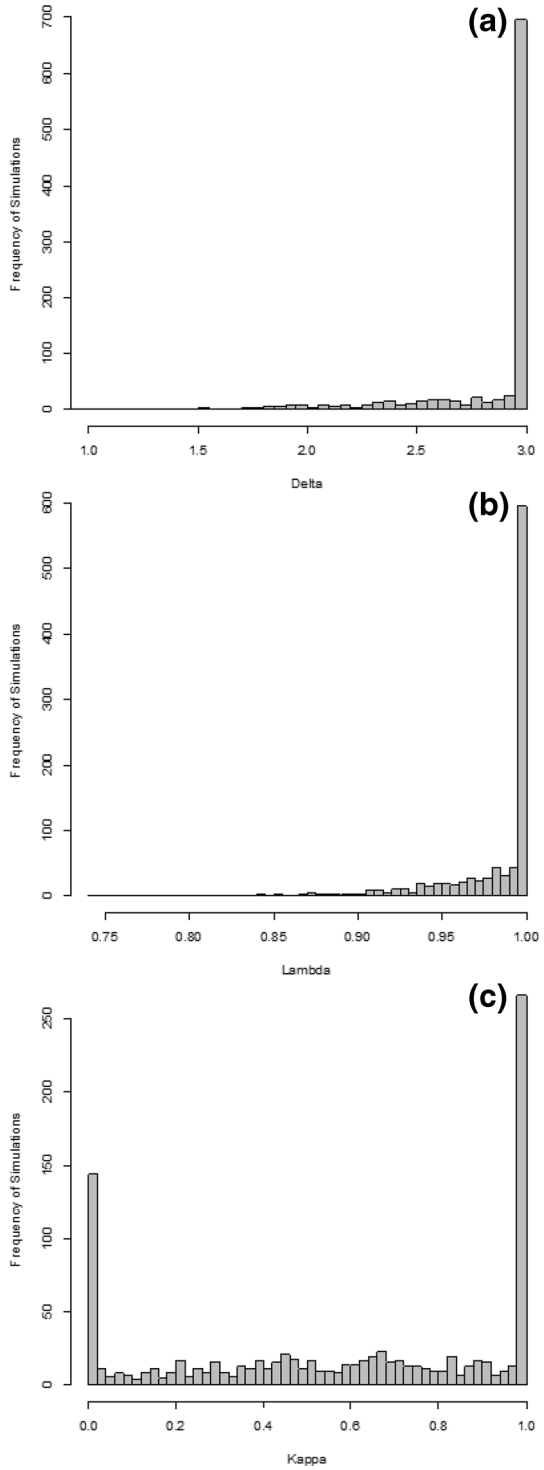

Fig. 2 Distribution of the parameters in the evolutionary models originally fitted by Pagel (2002) [Delta (**a**), Lambda (**b**) and Kappa (**c**)] obtained here, analyzing brain size evolution on 1000 simulated phylogenies (see Fig. 1)

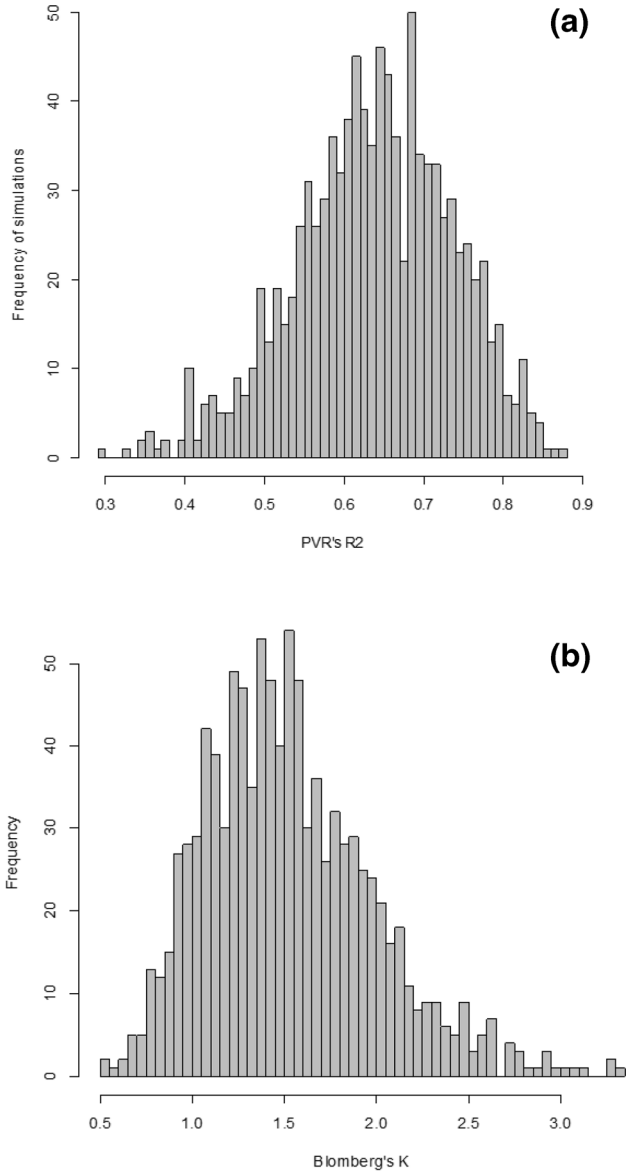

Fig. 3 Distribution of PVR's R^2 (**a**) and Blomberg's K (**b**) for brain size evolution based on 1000 phylogenies

equal to − 0.61) in 85% of the simulations. The smallest residuals were found for ECV in *A. sediba* in 10% of the simulations (mean residual of − 0.34), and for *H. naledi* 3% of the times (mean residual of − 0.28). This indicates that, taking phylogenetic uncertainty into account, these three species tend to have smaller brains than expected by their phylogenetic position across all simulations. The largest PVR residuals in ECV, on the other hand, appears for *H. neanderthalensis* in 26% of the times, followed by *H. sapiens* and *H. erectus* in 22% and 14% of the simulations, respectively.

Freckleton's et al. (2008) approach did not allow detecting, in principle, a strong significant relationship between diversification rates and ECV. Results using original's Freckleton's et al. (2008) PGLS indicate a significant correlation between ECV and diversification in about 23% of

the simulations based on delta transformations of branch lengths, with a median $R^2 = 17\%$ (Fig. S2A). The median correlation between diversification rates for species and ECV is in general relatively high ($R^2 = 0.399$), but the distribution of partial regression R^2 for the unique effects of diversification (i.e., after accounting for phylogenetic structure) are close to zero, strongly right-skewed (see Fig. S2B in the electronic supplementary material). However, there is a large overlap between diversification per species and phylogenetic structure estimated by phylogenetic eigenvectors, so it is difficult to decouple these two components (phylogenetic similarity among species and diversification trends along each branch). The mean unique component of phylogenetic structure independent of diversification is 18% (Fig. S2C).

The average PSR curve (Fig. 4), with 95% confidence intervals, indicates a pattern in which evolution occurs faster than expected by Brownian motion (consistently with Pagel's (2002) results and with the mean Blomberg's $K > 1$; see Diniz-Filho et al. 2012). However, in 11% of the simulations, significant non-stationary patterns were detected by the PSR curves, indicating localized shifts in ECV evolution and that changes occurred at different rates in different parts of the phylogeny.

When analyzing the simulations in which PSR indicates non-stationarity, the eigenvectors describing the shifts are isolated and ordered through MDS (Fig. 5). A broken-stick criterion only the first two axes indicate significant patterns

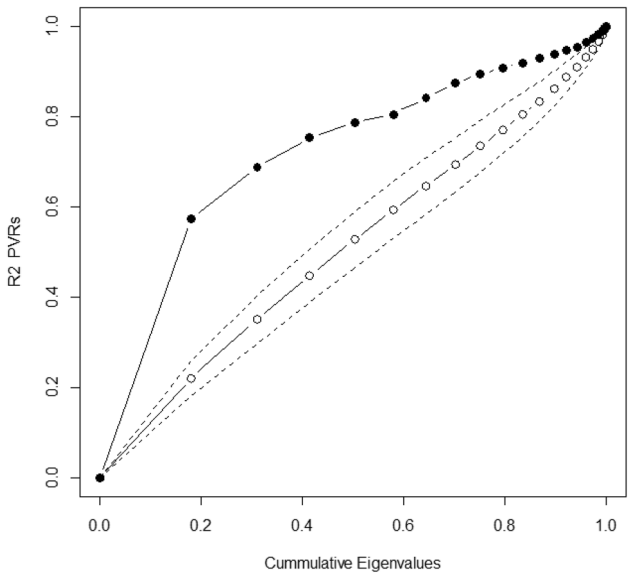

Fig. 4 The mean PSR curve for brain size evolution based on 1000 simulations (circles) and the expected pattern under 1000 simulation of Brownian motion on each phylogeny (crosses), as well as 95% confidence intervals (dashed line). The observed PSR curve lies significantly above the curve for simulated Brownian motion, so it indicates accelerated evolution, coherent with Blomberg's K and delta parameter above 1

of variation among species (in terms of non-stationarity). We found out that the first component explains 45.6% of the variance in the distances among non-stationary eigenvectors. This eigenvector highlights the difference between the clade including *H. antecessor, H. heidelbergensis, H. neanderthalensis* and *H. sapiens*, with higher values for the last three species, from the other species (Fig. 5a). The second component of the MDS, explaining 22.5% of the non-stationary variation, indicates a shift between *H. floresiensis* and *H. erectus* (Fig. 5b). Results from K-mean clustering also indicate similar patterns when comparing non-stationary phylogenetic eigenvectors, with only two clusters (see Fig. S3 in the electronic supplementary material, which also reveal a contrast between *H. heidelbergensis* and *H. neanderthalensis* within the first component of non-stationarity in Fig. 5a).

Discussion

Evolutionary Models and Phylogenetic Signal

There are three main insights emerging from our revisit to Pagel's (2002) original approach which are usefully interpreted in the light of recent findings coming from other studies (Gómez-Robles et al. 2017; Du et al. 2018) addressing questions similar to ours. These are the temporal variation in ECV evolutionary rates, the mode of evolution (i.e., whether gradual versus punctuated patterns are apparent), and the importance of anagenesis as opposed to cladogenesis in understanding brain evolution in hominins.

The analyses presented in Pagel's (2002) seminal paper are, in principle, robust to the inclusion of new data and to phylogenetic uncertainty. We found the δ model to fit the ECV data best. Pagel's (2002) δ estimate of 3.01, which is very close to our own mean estimate (2.78) and indicates that ECV evolved faster towards the present. The data also display a strong phylogenetic signal (supported by both PVR's high R^2 and Blomberg's *K* statistics), with λ tending to 1.0 in most simulations. This is close to Pagel's finding as well. In addition, Blomberg's *K* statistics and the overall shape of the PSR curve (i.e., which lays above the line expected under Brownian motion) also support that evolution in ECV is faster than expected under a pure Brownian motion model. These results are consistent with recent findings by Du et al. (2018) using a different approach to model temporal trends in ECV at different phylogenetic scales. More importantly, they agree with earlier findings by Gómez-Robles et al. (2017) who explicitly showed, using rates calculated from ancestor estimates under Brownian motion, that this acceleration occurs along some particular branches in the human tree, especially those leading to the later species of *Homo* (mainly from *H. heidelbergensis* towards *H. sapiens* and *H.*

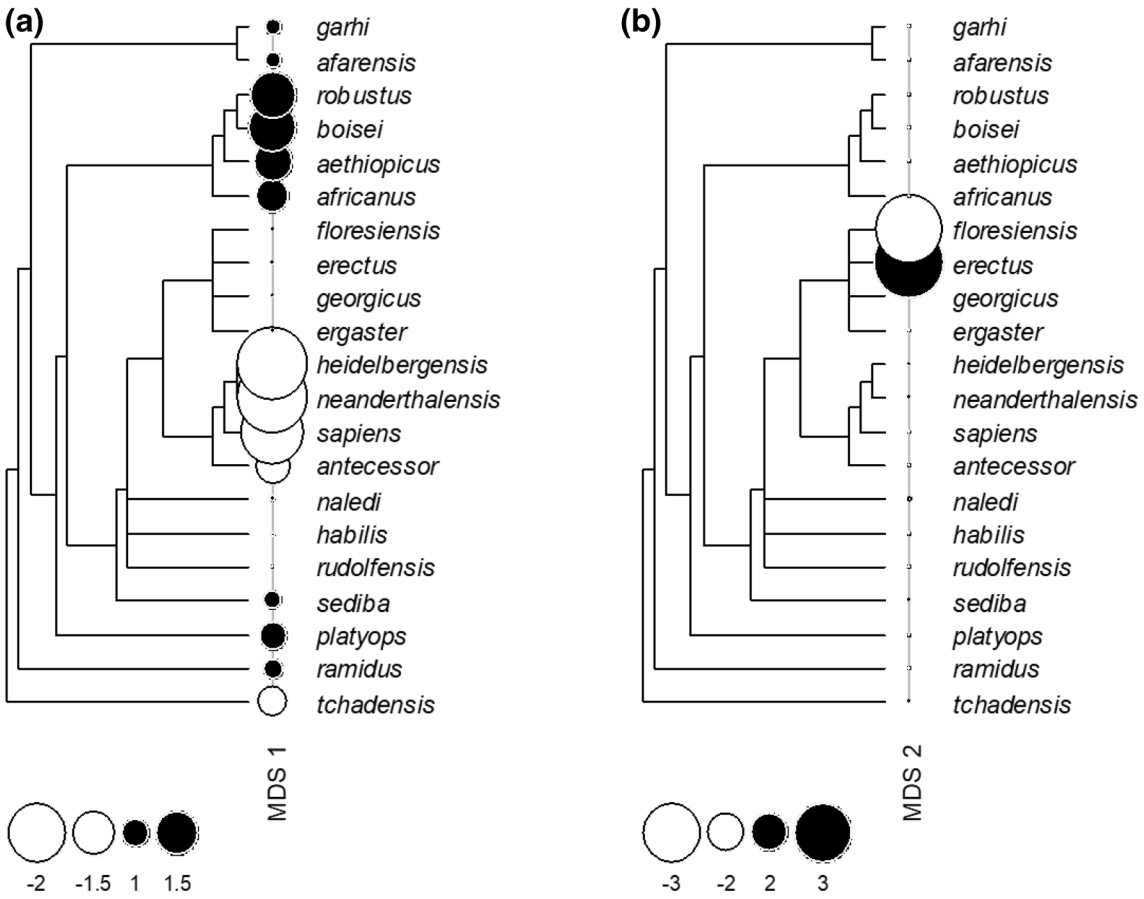

Fig. 5 The two principal coordinates of non-stationarity in brain size evolution, mapped on the basic phylogeny. **a** The shift in the lineage leading to the "modern" humans (*H. heidelbergensis, H. neanderthalensis* and *H. sapiens*), that explains 50.2% of the variation in distance among non-stationarity axes in the PSR curves; **b** the shift between *H. floresiensis* and *H. erectus*, that explains 15.9% of non-stationarity

neanderthalensis). It is important to realize that in Pagel's (2002) original analyses the ECV data were fitted by a directional model as well, whereas in our analysis no significant trend was observed (the trend model performed best in as few as 2% of the simulations). Even so, because we are analyzing a non-ultrametric phylogeny with extinct lineages, the better fit of a model with a $\delta > 1$ clearly indicates that more recent events in the phylogeny are more important in explaining variation in EVC (see also Fig. 1c).

Differently from Pagel (2002), the κ values estimated across our simulations were strongly bimodal, although in most case closer to 1.0 (i.e., indicating that evolution is not punctuated, again supporting recent analyses by Du et al. 2018). However, in about 21% of the simulations the kappa values were close to 0, which would support a punctuated equilibrium model. This wide and bimodal distribution of κ values estimated for ECV while accommodating for phylogenetic uncertainty is also reflected in the literature, where both punctuated evolution and gradual patterns are almost equally supported, and indicates that the phylogenetic arrangement may be crucial in telling the two patterns apart (see Du et al. 2018 for a review). It is interesting to note that κ is the best fit model in only about 6% of the simulations, but when this happens the fitted parameter tend to zero. Anyway, by considering the best fit of δ in most simulations and the strong phylogenetic signal from Blomberg's *K* and PVR, it is possible to state that our analyses tends to indicative a gradual model for the evolution of ECV.

As pointed out by Du et al. (2018), evolutionary patterns in ECV are better explained by gradual trends and variation within lineages (anagenetic) than by macroevolutionary (i.e., speciation and extinction dynamics; see Jablonski 2017a, b) events, especially using less speciose (i.e., based on a more splitting taxonomy) definition of taxa for analyses. In this case, the idea is that extinction of small-brained lineages reinforces the impression of rate change (i.e., acceleration) of ECV towards the present. Their variance partition suggests that in these cases around 60% of the variation in ECV is due to speciation-extinction dynamics. We did not find a strong correlation between diversification and ECV,

with very low unique components in partial PVR. Yet, partial PVR also reveals that there is a strong overlap between diversification rate within lineages and their phylogenetic structure in explaining ECV variation. Even so, this high overlap term indicates that we cannot rule out in principle that macroevolutionary sorting lead to higher ECV towards the present, which is coherent with Freckleton's et al. (2008) PGLS approach, with 22% of the correlations with $P < 0.05$ (despite the relatively low R^2). As pointed out by Du et al. (2018), this leads to a new research avenue to understand how processes driving variation in speciation and extinction dynamics would be affected by ECV along human evolution. Unique effects from phylogenetic eigenvectors (i.e., similarity among phylogenetically related species) explain about 24% of the variation, which is about one-third of the total variation in EVC explained in the full model. Although it is not statistically possible to decouple the overlap term, based on available information, these simple calculations show that unique gradual evolution expressed by similarity among phylogenetically related species does not suffice to explain EVC evolution in hominins. Although unique effects of diversification are low, favoring interpretations of diversification or gradual evolution in overlap is subjective.

Patterns of Phylogenetic Non-stationarity

Although our results for overall evolutionary models and phylogenetic signal in ECV are generally supportive of Pagel's (2002) findings and in line with recent analyses by Gómez-Robles et al. (2017) and Du et al. (2018), additional evidence unique to this study reveal that more complex evolutionary patterns may be present in the data, conditional to the phylogenetic structure underlying species relationships. The multiple PVR curves support the idea that brain size evolution was non-stationary in hominins in about 11% of simulations, with significant shifts occurring at three distinct nodes. This strongly suggests that different processes may relate to different parts of the phylogeny, in keeping with a recent claim in Du et al. (2018). The most important shift in brain size evolution detected by PSR occurs at the basis of the "modern human" lineage (including *H. sapiens, H. neanderthalensis* and *H. heidelbergensis*). Non-stationarity analysis reveals that, considering uncertainty in data, this might be not only a true "acceleration" towards the present, but rather the effect of a non-gradual shift towards larger brain size in the lineage leading to modern humans. Because some recent lineages related to *H. erectus* s.l. (including the small brain *H. floresiensis*; see below), as well as *H. naledi*, persisted until recent times and without possessing a large brain, the acceleration of the entire *Homo* clade becomes unfeasible.

Gómez-Robles et al. (2017), who found that brain size evolution was twice as fast in recent lineages (those leading to *H. sapiens, H. neanderthalensis* and *H. heidelbergensis*) than in any older, which is consistent with the shift described by the non-stationarity in PRS curves described here. This shift was also discussed by González-Forero and Gardner (2018), who showed that a distinctive increase in brain size due to ecological pressure occurred in the ancestor of *H. sapiens* and *H. neanderthalensis*, and not in the older *H. erectus* group. Finally, Neubauer and Hublin (2012) pointed out that a developmental shift occurred in later stages of *Homo* evolution, with extended brain growth explaining a much larger adult brain size (and also the globular form of late *H. sapiens* skulls; see Neubauer et al. 2018).

The second component of non-stationarity is the shift between *H. floresiensis* and *H. erectus*, explaining about 22.5% of the variation in the non-stationarity eigenvectors. The case of *H. floresiensis* has been widely discussed in literature. The species was originally described as a dwarfed form of *H. erectus*, and later considered a pathological form of *H. sapiens* (see Eckhardt et al. 2014). Yet, several recent accounts (Baab 2016b; Kaifu et al. 2011) and additional fossil findings (dating the origin of the species back at some 700 ky, van den Bergh et al. 2016) supported the validity of the species. However, there is still some discussions about the ancestor of the species, especially fueled by recent phylogenetic analyses suggesting that *H. floresiensis* could be related to older lineages at the base of the genus *Homo* (e.g., Argue et al. 2017; Dembo et al. 2015, 2016; Trueman 2010; Zeitoun et al. 2016; Schroeder et al. 2017). This phylogenetic relationship would not be plausible given the current biogeographical understanding of the first dispersal out of Africa about 1.8 mya (i.e., assigning *H. floresiensis* to an older ancestor would imply in a much earlier dispersal out of Africa; see Carotenuto et al. 2016). On the other hand, by considering the complexity and wide variation of early *Homo* in Africa and at Dmanisi (Lordkipanidze et al. 2013; Spoor et al. 2015; Schroeder et al. 2017) and the fast potential dispersal out of Africa towards southeast Asia (Hughes and Smith 2008), it is actually difficult to define the particular attributes of the population that gave origin to *H. floresiensis*.

It is interesting to note that this shift refers to the divergence between *H. floresiensis* and *H. erectus*, and the later species here refers to late forms with a relatively large ECV. The non-stationarity analyses suggest that this strong deviation can be characterized by a rate shift in the phylogenetic expectations, which explains the many recent controversies surround this species (see Baab 2016b). But a visual inspection of the patterns shown by phylogenetic eigenvectors reveal that only rarely a significant contrast appears between *H. floresiensis* and other forms of *H. erectus*l.s. (manly *H. ergaster*). Thus, the phylogenetic comparative analyses presented here show that ECV of *H. floresiensis* is smaller than expected by the evolutionary

model mainly if *H. erectus*s.s. is the ancestor. Indeed, the analyses by Diniz-Filho and Raia (2017), based on quantitative genetics of brain size—body size allometry, suggested that the expected ECV of *H. floresiensis* ancestor is about 700–800 cc, consistent to what is found for early *H. erectus s. l.* like *H. georgicus* (and not the large-brained *H. erectus*). Thus, the PVR analyses indicate that *H. floresiensis* has really a small ECV considering its phylogenetic position in the genus *Homo* (i.e., it is the smallest residuals in 85% of the simulations), but this would really indicate a discontinuity in the gradual mode of evolution if it descends from a large-brained form of *H. erectus*. Notice also that although more refined analyses using allometric scaling (Kubo et al. 2013) or quantitative evolutionary genetics models (Diniz-Filho and Raia 2017) showed that brain size in *H. floresiensis* is still a bit smaller than expected by body size reduction, but the evidence for a shift in brain size driving the overall dwarfism process in *H. floresiensis* (see Grabowski 2016) would be hardly detected at macroevolutionary scales, as done here.

Several recent papers pointed out that a true ECV shift coincides with the Out of Africa event (Carotenuto et al. 2016; but see; Lordkipanidze et al. 2013) at the basis of the *H. erectus* l.s. group. This did not appear as a main non-stationarity component in our simulations, although a visual inspection of the patterns in each of the non-stationary eigenvectors revealed a significant contrast between *H. georgicus* and the other species appears in about 10% of the simulations (indicating that this species is actually smaller than expected). Moreover, individual non-stationary eigenvectors also revealed a contrast between *A. sediba* and *H. rudolfensis*, which is also consistent with other analyses that concur with the view that *H. rudolfensis* is a distinct and large-brained species of early *Homo* (Spoor et al. 2015). Even so, the lack of a more general component at the basis of the genus *Homo* may partly depend on the fact the variation in ECV in African forms is too large to differentiate among species basing on this trait alone (Antón et al. 2014; Lordkipanidze et al. 2013; Schroeder and Ackermann 2017; Spoor et al. 2015). This also explains why *H. naledi*, even appearing as a recent species in time, is also not that different from its phylogenetic expectations (see Holloway et al. 2018). Apparently, at that time and in this group at the basis of the genus *Homo* dental and facial traits varied more than any measure of bain mass (Gómez-Robles et al. 2017). These morphological changes would be more clearly associated with strong environmental instability and vegetation shifts in Africa 1.5–2.0 mya (see Maslin et al. 2015), and brain expansion seems to have started later. Schroeder and Ackermann (2017) also point out that in general morphological shape differentiation can be explained by a neutral model, although selection can be detected in a few events, including cranial morphological at the basis of *H. erectus* and dispersal out of Africa, as shown by the second component of phylogenetic non-stationarity here.

Concluding Remarks

Our analyses is coherent with original results to those of Pagel (2002) and, in a more general sense, supportive of previous findings from students of human brain evolution. Yet, we argue this similarity is partly superficial because complex evolutionary dynamics in brain size evolution might actually have taken place (Grabowski et al. 2016 and; Du et al. 2018).

From a methodological point of view, our study shows how PVR can be useful to understand complex evolutionary patterns, including the multifaceted relationship between diversification rates and phylogenetic structure through time and how non-stationarity patterns can be detected by using the PSR approach (see Diniz-Filho et al. 2015). Other approaches available to detect non-stationarity and shifts in phenotypic evolution require a priori hypotheses, based on other ecological or life-history traits to build a model of heterogeneous rates and peak shifts among lineages and subclades (e.g. Beaulieu et al. 2012; Butler and King 2004; O'Meara et al. 2006). Sometimes the effects of these potential drivers of shifts in trait evolution are simply not known, so that an exploratory approach may be helpful first to better fit data and understand patterns (e.g. Castiglione et al. 2018; Eastman et al. 2011; Khabbazian et al. 2016; Uyeda and Harmon 2014) and, secondly, to help developing new hypothesis to further a posteriori testing.

In conclusion, our analyses shows that brain size evolution accelerated towards the present in hominins, occurred in a gradual mode and mainly driven by trends within lineages. Our specific analyses reveal that this acceleration in the evolutionary rate found in Pagel (2002; see also Gómez-Robles et al. (2017) and Du et al. 2018) could well be the result of a particular shift in brain size at the origin of the *Homo sapiens* lineage, including *H. neandethalensis* and *H. heidelbergensis*. The other important shift is the later differentiation of *Homo floresiensis* within *H. erectus* l.s. group. Our analyses thus reinforce recent views that patterns in brain size evolution are more complex than previously assumed and, moreover, that although most changes can be explained by anagenetic trends and within lineage variation due to adaptive evolution, other macroevolutionary events of sorting relating extinction and speciation dynamics are associated with brain evolution.

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