Relaxed genetic control of cortical organization in human brains compared with chimpanzees

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Abstract

The study of hominin brain evolution has largely focused on the neocortical expansion and reorganization undergone by humans as inferred from the endocranial fossil record. Comparisons of modern human brains with those of chimpanzees provide an additional line of evidence to define key neural traits that have emerged in human evolution and that underlie our unique behavioral specializations. In an attempt to identify fundamental developmental differences, we have estimated the genetic bases of brain size and organization in chimpanzees and humans by studying phenotypic similarities between individuals with known kinship relationships. We show that, while heritability for brain size and organization is high in chimpanzees, cerebral cortical

anatomy is substantially less genetically heritable than brain size in humans, indicating greater plasticity and increased environmental influence on neurodevelopment in our species. This relaxed genetic control on cortical organization is especially marked in association areas, and likely related to underlying microstructural changes in neural circuitry. A major result of increased plasticity is that the development of neural circuits that underlie behavior is more intensively modeled by the environmental, social and cultural context in humans than in other primate species, thus providing an anatomical basis for behavioral and cognitive evolution.

Key words

Brain evolution, plasticity, hominins, neocortex, altriciality

Significance statement

Despite decades of research, we still have a very incomplete understanding of what is special about the human brain compared to the brains of our closest fossil and living relatives. Parsing the genetic versus environmental factors that govern the structure of the cerebral cortex in humans and chimpanzees may shed light on the evolution of behavioral flexibility in the human lineage. We show that the morphology of the human cerebral cortex is substantially less genetically heritable than in chimpanzees and therefore more responsive to be molded by environmental influences. This anatomical property of increased plasticity, which is likely related to the human pattern of development, may underlie our species' capacity for cultural evolution.

\body

Compared with nonhuman primates, human brains are significantly enlarged, reorganized, and have a disproportionately expanded neocortex (1–3). The fossil evidence demonstrates that these changes occurred in the hominin lineage over the last ~ 6-8 Myrs (4–9) in parallel with modifications to neurodevelopmental rates (10–13). Although some of these changes have been linked to certain genetic variants in the human lineage [either shared with other late hominin species or exclusive to modern humans (14, 15)], exploring brain evolution in hominins is challenging due to the limitations of the endocranial fossil record (4, 5). Comparisons of chimpanzee and human brains are therefore essential to reveal neural traits that differ between both species, which underlie their behavioral specializations and must have evolved after they split from their last common ancestor.

Human behavioral and cognitive development is highly dependent on cultural influences and social learning (16, 17). Notably, modern human behavioral adaptations to live in diverse habitats depend on skills and information learned from others (18). Regarding nonhuman primates, several studies have demonstrated better performance of enculturated great apes in different tasks related to physical and, especially, social cognition (19), which underscores the importance of environmental influences in shaping behavior. These observations are congruent with experimental studies in mouse models showing that variation in sensory experience early in postnatal life causes reorganization of neural circuits that underlie behavior (20). However, the clear differences in behavioral and cognitive development between enculturated apes and humans point to particular neural specializations that make the human brain —but not the brain of great apes— extremely responsive to exogenous influences. In this light, several comparative

studies have shown molecular and microstructural specializations in the human brain that point to an increased level of synaptic plasticity (21, 22), which might be linked to increased learning abilities.

The potential role that changes in life history and developmental patterns may have had in human brain evolution has been highlighted in paleoanthropology and primatology (10, 13). It is generally assumed that the extended period of growth and delayed maturation of humans in the context of a complex social environment is related to our species' cognitive specializations (13). It remains to be clarified, however, if the human brain is indeed more extensively modeled by environmental factors than the brain of our closest living and fossil relatives. In the current study, we evaluated heritability for brain size and cortical organization in chimpanzees and humans to assess the relative contribution of genes and environment to neural development. Heritability is defined as the proportion of total phenotypic variance in a population that has a genetic basis. The heritability of traits can be calculated from phenotypic similarities between individuals with different degrees of genetic similarity.

The studied sample included magnetic resonance imaging (MRI) scans of 206 chimpanzees and 218 humans. A well-documented pedigree is available for the chimpanzees, whereas the human sample includes monozygotic twins, non-monozygotic twins and non-twin siblings. MRI scans were used to measure brain volume and to reconstruct three-dimensional models of the cortical surface. Cortical organization was characterized through a set of anatomically homologous landmarks (Fig. S1, Table S1 and *SI Text*), which were analyzed using linear distances (Fig. S2 and Table S2) and a geometric morphometric approach (*Datasets S1 and S2*). All measurements

were obtained after each individual brain was scaled to a common size through Procrustes superimposition, removing the effects of differences in overall brain size. Consequently, distances in our analyses do not reflect absolute size, but relative lobe proportions and sulcal measurements. This homology-based method allows for comparability across species in spite of differences in cortical anatomy and variation in scanning procedures. Also, this approach captures important information about the position and orientation of different cortical regions that is overlooked when focusing on volumes or surface areas of these regions. Additionally, this landmark-based approach avoids intensive automatic processing of anatomical data, which has been demonstrated to have a significant effect on neuroanatomical studies (23). Because of the importance of differential cortical expansion and reorganization in both evolution and development (24), we selected variables related to the morphology of sulci across the cerebral cortex. Sulcal variation shows a close correspondence with primary sensory and motor cytoarchitectonic areas (25), but a more variable correspondence with high-order association areas in both chimpanzees and humans (25, 26). In humans, sulcal morphology shows a high degree of interindividual variability that is linked to differences in functional networks and long-range corticocortical connectivity (27), whereas lobe- or region-specific volumetric measures and cortical thickness have been shown to be less variable and highly heritable (28).

Results

Our findings demonstrate that humans show very high heritability for brain size, which is consistent with previous studies (28) (Fig. 1A and Table S3). Chimpanzees show significant heritability for brain size, although substantially lower than humans (Fig. 1A and Table S3). Several reviews and meta-analyses have demonstrated that twin-based studies inflate heritability

116 estimates as compared to family- or pedigree-based studies (28), which is likely related to the
117 higher heritability for brain size observed in the human sample (see also *SI Text*). Cerebral lobe
118 dimensions show significant and relatively high heritability in both chimpanzees and humans
119 (Fig. 1B and Table S4). Sulci that demarcate cerebral lobe subdivisions, such as the central
120 sulcus and the Sylvian fissure, also have significant heritability in both species (Fig. 1C and
121 Table S4), which points to strong genetic control of lobar organization. However, other sulci
122 within cortical association regions show significant heritability only in chimpanzees, but not in
123 humans (Fig. 1C and Table S4). Low heritabilities in human sulci within higher-order
124 association regions suggest a greater degree of plasticity in brain architecture that is not observed
125 in chimpanzees. Genetic correlations in both species between variables tend to be low, although
126 there are exceptions that include some lobe dimensions (Fig. S3, Tables S5 and S6), which
127 reflect the inverse relationship between relative proportions of cerebral lobes.
128
129 Principal components analyses of shape variation within each species show different patterns of
130 divergence with respect to genetic similarity in chimpanzees and humans. In chimpanzees,
131 mother-offspring pairs, which share 50% genetic similarity, show less shape divergence than
132 half-sibling pairs, which share on average 25% genetic similarity (Figs. 2A and 2B). In humans,
133 however, 50% decrease in genetic similarity is not associated with an increase in shape
134 differences: monozygotic twins, who share 100% genetic similarity, show the same degree of
135 shape variation as non-monozygotic twins and non-twin siblings, who share on average 50%
136 genetic similarity (Figs. 2D and 2E). These differences are further reflected in the substantially
137 higher heritabilities observed in chimpanzees for principal components of shape variation than in
138 humans. Chimpanzees show significant heritability in the first ten principal components, which

correspond to the main patterns of shape variation (Fig. 3A and Table S7). The main pattern of variation in chimpanzee brains, summarized by PC1, corresponds to differences in the general proportions of the brain, which vary from long and narrow to short and broad (Fig. 2C). This component of anatomical variation shows a highly significant heritability of 0.59 ($p<0.001$) (Fig. 3A and Table S7). Subsequent principal components also show significant and relatively high heritabilities, with a weighted mean (weighted by the proportion of variance explained by each PC) of 0.48 (Fig. 3A and Table S7). Notably, the heritabilities for shape variation approach the same degree of heritability for overall brain size in chimpanzees. Humans, however, show non-significant heritabilities in several of these components, including PC1 (Fig. 3B and Table S7). In the human sample, the main pattern of shape variation corresponds to differences within perisylvian areas that involve reorientation of the Sylvian fissure and reorganization of the superior temporal sulcus (Fig. 2F). This pattern of variation has a non-significant heritability of 0.21 ($p=0.142$) (Fig. 3B and Table S7), which indicates that this aspect of interindividual variability in sulcal morphology of humans is under relaxed genetic control. The weighted mean heritability for the first ten principal components of cortical shape variation in humans is 0.35 (Fig. 3B and Table S7), which is less than half the heritability for brain size. Temporal and inferior parietal regions, the variation of which is associated with the lowest heritability values, are involved in cognitive functions in humans that include language, attention and memory (29). Our findings highlight the importance of cortical plasticity as a foundation for the emergence of high-order cognitive functions (29), as environmental influence on areas dedicated to these functions is substantially greater in humans than in chimpanzees.

Discussion

Differences in population structure between chimpanzee and human samples make it necessary to restrict comparisons of heritability values within each species separately. Furthermore, heritability values are characteristic of given populations and particular environmental conditions, requiring caution in making cross-species and cross-study comparisons. Nonetheless, within-species differences are marked in our analyses. Specifically, while chimpanzees are characterized by similar heritability levels for brain size and cortical morphology, humans show a much higher heritability for brain size than for cortical organization, indicating elevated plasticity in our species for the latter. This interpretation is further supported by previous findings demonstrating that human brains exhibit a higher level of fluctuating asymmetry in cortical association areas compared with chimpanzees (30). The lack of clear homology between humans and chimpanzees in some sulci of the inferior frontal and occipital lobes (see *SI Text*) is notable and reflects the higher variability of the human brain. As humans do not have clear fronto-orbital and lunate sulci, our analyses in the human sample focused on alternate sulci and landmarks that can be most reliably identified in the inferior frontal region and in the parieto-occipital boundary. Those sulcal dimensions still show substantially lower heritability in humans than developmentally and evolutionarily primary sulci such as the central sulcus and the Sylvian fissure.

Studies of cortical development in humans have shown differential regional enlargement, which has been suggested to reflect extended maturation and complexity of dendritic and synaptic architecture in association areas (24). Lateral temporal, lateral parietal, dorsal and medial prefrontal regions show the greatest degree of expansion from birth to adulthood, and it has been suggested that cortical circuits in these regions may be more sensitive to postnatal experience

(24). Heritability patterns observed in chimpanzees and humans in the present study are consistent with the proposition that humans have evolved relaxed genetic control on cortical organization, especially in areas related to higher-order cognitive functions. Although particular plastic changes are not themselves heritable, the level of developmental plasticity in different traits can have a genetic basis and, therefore, be evolvable, and it may respond to both artificial and natural selection (31–33). A high level of cortical plasticity means that neural circuits that are responsible for behavior are formed under a complex array of environmental influences that directly shape those networks, thus providing a neurobiological basis for socially- and culturally-mediated behavioral evolution.

A causal factor driving the highly plastic nature of the human brain is likely the underdeveloped or altricial condition of humans at birth (34), which requires a relatively larger fraction of brain maturation to occur postnatally. Humans have evolved a secondary altricial pattern of development from the more precocial pattern that characterizes other living primates (34), which might be related to obstetrical (35) or metabolic constraints (36). Regardless of the initial causal factor, once established, an altricial pattern of development may have provided fundamental selective advantages through the opportunity for postnatal maturation and associated increased learning abilities to allow human offspring to incorporate cultural information through social transmission mechanisms.

The increase in brain size that is observed during hominin evolution may have created the opportunity for a more extended postnatal period of brain maturation, thus promoting a synergistic interaction between an increased computational capacity [larger brains (3) with

208 expanded neocortices (1) and more neurons (37)] and the ability to form connections in a plastic,
209 environment-dependent manner. This model therefore predicts that hominin species with a large
210 brain size and modern body proportions likely also had an altricial pattern of development, a
211 prolonged postnatal period of brain maturation and an increased level of cerebral cortical
212 plasticity. While several studies of brain growth in *H. erectus*, which is the first hominin species
213 characterized by these anatomical traits, indicate that this species likely had a pattern of brain
214 development intermediate between those of chimpanzees and modern humans (11, 38), it has
215 also been suggested by other analyses that *H. erectus* and *H. sapiens* shared similar
216 developmental patterns (39). Either way, secondary altriciality seems to have been characteristic
217 of different species of the genus *Homo*, and to have evolved at least in the last common ancestor
218 of Neanderthals and modern humans (11). In that case, and in spite of the differences in the
219 evolution and development of endocranial shape between these species (6, 12), they may have
220 shared the anatomical bases for social learning and cultural accumulation that are related to
221 human cognitive evolution. Our results showing relaxed genetic control of cortical anatomy in
222 human brains compared with chimpanzees point to the fundamental role of developmental
223 plasticity in increasing learning abilities and allowing behavioral flexibility in late hominins, thus
224 providing a link between biological evolution and cultural evolution.
225
226 **Materials and Methods**
227 **Samples and MRI scans.** A sample of 206 chimpanzee (79 males, 127 females, age range 8-53)
228 and 218 human (87 males, 131 females, age range 22-30) MRI scans was used. The number of
229 human individuals was chosen to approximately match the number of available chimpanzee
230 scans. Chimpanzees used in this study were housed at the Yerkes National Primate Research

231 Center (YNPRC) in Atlanta, GA, and at the University of Texas MD Anderson Cancer Center
232 (UTMDACC) in Bastrop, TX. They were scanned using a 3T scanner (Siemens Trio, Siemens
233 Medical Solutions, Malvern, USA) or a 1.5T scanner (Phillips, Model 51, Philips Medical
234 Systems, N.A., Bothell, Washington, USA). Technical details regarding scanning procedures and
235 processing can be found in ref. 40. Scanning procedures in chimpanzees were approved by the
236 Institutional Animal Care and Use Committees at YNPRC and UTMDACC, and also followed
237 the guidelines of the Institute of Medicine on the use of chimpanzees in research. No paternity
238 tests were conducted for the purposes of this study, but a well-documented pedigree is available
239 for these chimpanzees, which includes information on mother, father and offspring identity for
240 many individuals. This chimpanzee population has been used previously in quantitative genetic
241 studies of behavioral phenotypes (41, 42). Human MRI scans were obtained from the Human
242 Connectome Project (HCP) database (43). Individuals were scanned with a Siemens Skyra 3T
243 scanner. Technical details regarding scanning procedures and processing in human subjects can
244 be found in refs. 43 and 44. Consent from human participants was obtained in the context of the
245 Human Connectome Project, and data use terms for open and restricted data were accepted and
246 observed as per HCP requirement (45). The HCP database includes monozygotic twins, non-
247 monozygotic twins and non-twin siblings. In order to maximize sample size and minimize inter-
248 population variability due to genetic ancestry, which has been recently proposed to correlate with
249 general brain anatomy (46), white (self-defined) individuals were selected, as they are more
250 numerous in the HCP database than individuals with other ancestries.
251
252 **3D reconstructions and landmarks.** Three-dimensional models of the cortical surface were
253 reconstructed from MRI scans using BrainVisa software (47) for chimpanzees and FreeSurfer

254 software (48) for humans (3D models were directly obtained from the HCP database for the
255 human sample). Thirty-two anatomically homologous landmarks (16 bilateral landmarks) were
256 placed on the intersections and extreme points of the most constant sulci in the chimpanzee
257 cortical surface (30, 49) (Fig. S1 and Table S1). The same sulci were used to identify equivalent
258 anatomically homologous landmarks in human brains (but see *SI Text*). Because of the
259 anatomical complexity of the human cortical surface, which makes it difficult to identify some
260 sulci, landmark placement was aided by a comparison with automatically parcellated models.
261 These parcellated models, obtained with FreeSurfer software version 5.3.0 according to the
262 Desikan surface atlas (50), are provided in the HCP database. In comparison with other studies
263 of heritability in brain structure, our study can be considered a minimal-processing approach.
264 The use of anatomically homologous landmarks makes our study reliant on anatomical criteria
265 rather than on processing steps that have been demonstrated to have a significant effect on the
266 evaluated phenotypes (23).
267
268 **Brain volume measurement and linear distances.** Brain volumes were obtained from the HCP
269 database for humans, which were obtained in turn from the FreeSurfer structural pipeline (48). In
270 chimpanzees, brain volumes were obtained from BrainVisa (47) masks. Potential differences in
271 values obtained from both approaches do not impact our results because both species were not
272 compared to each other in the same analyses.
273
274 Linear distances between landmarks were calculated in *Mathematica* (Wolfram Research).
275 Euclidean distances between landmarks were measured after Procrustes superimposition [which
276 entails a translation, scaling and rotation of configurations until distances between homologous

277 landmarks are minimized following a least squares criterion (51)] to remove differences in
278 general size. Individuals were scaled so that centroid size, defined as the squared root of the sum
279 of the squared distances between each landmark and the centroid of the configuration, was 1 in
280 all individuals. Variation in original dimensions (as measured in individuals' native space before
281 Procrustes superimposition) and in dimensions obtained after Procrustes superimposition is
282 shown in Fig. S2. Asymmetric variation was removed by averaging left and right values because
283 our aim was to assess general patterns of heritability in cortical anatomy without regard to side-
284 specific differences. Inter-landmark linear distances included two types of variables. The first
285 one corresponded to dimensions of the major cerebral lobes, which were defined as: superior and
286 inferior frontal lengths, superior and inferior parietal lengths, temporal and occipital lengths
287 (Table S2). The second group of variables corresponded to linear approximations of the lengths
288 of major cortical sulci, including the central sulcus, Sylvian fissure, fronto-orbital sulcus (latero-
289 orbital sulcus in humans), precentral sulcus, superior temporal sulcus and lunate sulcus (parieto-
290 occipital sulcus in humans). Potential concerns regarding the homology of some of these sulci
291 between chimpanzees and humans are discussed in *SI Text*. The first group of linear distances
292 (lobe dimensions) describes the general proportions of cerebral lobes. The second group of linear
293 distances (sulcal dimensions) describes more detailed aspects of cortical organization.
294
295 **Geometric morphometrics.** Asymmetric variation was removed by mirror-imaging and
296 averaging the original and mirrored configurations of landmarks for each specimen (52). As
297 indicated above, variation corresponding to position, orientation and size of individuals in the
298 digitized space was removed through Procrustes superimposition (51). No further affine or non-
299 affine registration was performed in order to maintain and analyze all shape variation in

analyses. Procrustes-superimposed landmark coordinates were subjected to separate principal components analyses for each species, as our major interest was to understand the genetic bases of shape variation within each species. Each principal component corresponds to a set of phenotypically correlated changes in the position of certain landmarks across the whole population, the genetic bases of which were later estimated. Patterns of shape differences between kin-related individuals were visualized by highlighting pairs of individuals with different degrees of genetic similarity in the morphospace formed by PC1 and PC2. In chimpanzees, the ten closest mother-offspring pairs (which share 50% genetic similarity) were represented and compared with the ten closest pairs of half siblings (who share on average 25% genetic similarity). In humans, the ten closest pairs of monozygotic twins (who share 100% genetic similarity) were represented and compared with the ten closest pairs of non-monozygotic twins or non-twin siblings (who share on average 50% genetic similarity). Patterns of shape variation corresponding to extreme values on PC1 and PC2 for each species are represented as well.

Quantitative genetics. A maximum likelihood approach was used to estimate the components of variance of the different evaluated variables as implemented in SOLAR software (53). Narrow sense heritabilities were estimated and their significance was tested using likelihood ratio tests. Following other quantitative genetic studies of brain, endocranial and cranial anatomy in humans and nonhuman primates, age, sex, and the interaction between age and sex were used as covariates (54–56). Overall brain size was also tested as a covariate in analyses of linear distances and principal components of shape. Because linear measures were obtained and geometric morphometric analyses were performed after all individuals were scaled to a common

size through Procrustes superimposition, overall brain size was not expected to have a consistent significant effect. However, it was still tested as a covariate to explore potential allometric trends. Chimpanzees in the sample belong to different colonies and they were scanned with two different types of scanner (the correspondence between these two variables is not complete). For these reasons, these variables (colony and scanner type) were used as covariates in analyses of chimpanzees. When covariates were significant at $P<0.10$ level, they were retained in final models to calculate heritabilities, and they were excluded when not significant. Variables were inverse-normalized before analyses to force normality and avoid high residual kurtosis (56).

Heritability for the first ten principal components of shape variation, which correspond to the ten major patterns of phenotypic variation within each sample, was estimated using the same methodological approach and the same covariates. A clear drop in eigenvalues is observed in PC5 for chimpanzees and in PC3 for humans, but the distribution of variance is in general very homogeneous in both species (see Fig. 3). Because these first PCs explain a rather minor proportion of variance in both species (40.6% in chimpanzees for PC1-PC4 and 23.5% in humans for PC1-PC2), analyses of heritability were extended to the first ten principal components, which explain 68.5% of shape variance in chimpanzees and 62.6% in humans. Subsequent principal components were not included because they explain very minor proportions of shape variation. Univariate estimates of heritability in these principal components were preferred over a fully multivariate approach as described in refs 55 and 56 due to the limited size of our samples. Although these sample sizes are enough to estimate heritabilities, they do not allow for a reliable calculation of genetic correlations and covariances, which makes the estimated genetic covariance matrices unstable.

Genetic correlations between lobe and sulcal dimensions. Genetic correlations between linear measures were estimated using bivariate models in which significant covariates for each variable were retained. The genetic correlation between two traits is defined as the association between those traits due to the correlation between the loci controlling both traits. These correlations can arise through linkage disequilibrium or pleiotropy (59), and they are usually considered to constrain evolution and reduce evolvability. As stated above, the reliable estimation of genetic correlations requires very large sample sizes that exceed the number of individuals available to our study. For this reason, genetic correlations between lobe dimensions and between sulcal dimensions are provided in Fig. S3 and Tables S5 and S6, but they should be taken with caution.

Representation of heritabilities. For heritability in linear measures, linear distances were represented and overlaid on 3D models of a representative chimpanzee and human brain. Linear dimensions were color-coded according to their heritability values. Chimpanzee-human differences were maximized by rescaling the color gradient to the minimum and maximum heritabilities observed in our study (0.07 and 0.77, respectively), instead of using the whole range of possible heritabilities (0-1). Heritabilities in patterns of shape variation (principal components) were also color-coded and overlaid on scree plots representing the distribution of variance within each species (heritability range for color code is 0-0.72). Heritabilities that remained significant after correcting for multiple comparisons using a false discovery rate procedure (60, 61) were marked in Figs. 1 and 3. Original P-values obtained for all analyses are listed in Tables S3, S4 and S7.

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538 **Figure legends**

539 **Fig. 1.** Heritability for brain size and lobe and sulcal dimensions. (*A*) Heritability for brain size
540 (brain volume including white and gray matter, but not ventricular spaces) for chimpanzees (left)
541 and humans (right). (*B*) Heritability for cerebral lobe dimensions in chimpanzees (left) and
542 humans (right): SF: superior frontal length; IF: inferior frontal length; SP: superior parietal
543 length; IP: inferior parietal length; T: temporal length; O: occipital length. (*C*) Heritability for
544 sulcal lengths in chimpanzees (left) and humans (right): FOS: fronto-orbital sulcus; LOS: latero-
545 orbital sulcus; PCS: precentral sulcus; CS: central sulcus; SyF: Sylvian fissure; STS: superior
546 temporal sulcus; LS: lunate sulcus; POS: parieto-occipital sulcus. In *B* and *C* lobe dimensions
547 and sulci are color-coded according to heritability values as indicated in the color scale bars.
548 Dimensions and sulci marked with an asterisk show significant heritability after using a false
549 discovery rate approach to control for multiple comparisons. Detailed heritabilities, standard
550 errors and P-values are listed in Tables S3 and S4. In *B* and *C* chimpanzee and human brains are
551 not to scale.

552

553 **Fig. 2.** Principal components analysis of shape variation in chimpanzee and human brains. (*A*
554 and *B*) Principal components analysis of shape variation in chimpanzee brains showing the ten
555 closest mother-offspring pairs (*A*, pink links), which share 50% genetic similarity, and the ten
556 closest pairs of half siblings (*B*, purple links), who share on average 25% genetic similarity. (*C*)
557 Brain models showing shape variation corresponding to positive and negative extremes of PC1
558 and PC2 in chimpanzees. (*D* and *E*), Principal components analysis of shape variation in human
559 brains showing the ten closest pairs of monozygotic twins (*D*, pink links), who share 100%
560 genetic similarity, and the ten closest pairs of non-monozygotic twins or non-twin siblings (*E*,

purple links), who share on average 50% genetic similarity. (*F*) Brain models showing shape variation corresponding to positive and negative extremes of PC1 and PC2 in humans. *C* and *F* include dorsal and lateral views, with right hemispheres represented as opaque models with landmarks and left hemispheres represented as transparent models with overlaid schematic representations of landmark variation. Red is used in brain models to show variation corresponding to negative extremes on PC1 and PC2, and blue is used to show variation corresponding to positive extremes in those PCs.

Fig. 3. Distribution of variance and heritability of phenotypic shape variation. (*A*) Scree plot showing the distribution of shape variance in chimpanzee brains. (*B*) Scree plot corresponding to shape variation in human brains. Heritabilities for the first ten principal components are represented using a color code. Principal components marked with an asterisk show significant heritability after applying a false discovery rate to control for multiple comparisons. Only the first twenty principal components are represented; heritabilities of PC11-PC20 have not been estimated because they account for very minor proportions of variance. Detailed heritabilities, standard errors and P-values are listed in Table S7.