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Organizing Principles of Human Cortical Development—Thickness and Area from 4 to 30 Years: Insights from Comparative Primate Neuroanatomy

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The human cerebral cortex undergoes a protracted, regionally heterogeneous development well into young adulthood. Cortical areas that expand the most during human development correspond to those that differ most markedly when the brains of macaque monkeys and humans are compared. However, it remains unclear to what extent this relationship derives from allometric scaling laws that apply to primate brains in general, or represents unique evolutionary adaptations. Furthermore, it is unknown whether the relationship only applies to surface area (SA), or also holds for cortical thickness (CT). In 331 participants aged 4 to 30, we calculated age functions of SA and CT, and examined the correspondence of human cortical development with macaque to human expansion, and with expansion across nonhuman primates. CT followed a linear negative age function from 4 to 30 years, while SA showed positive age functions until 12 years with little further development. Differential cortical expansion across primates was related to regional maturation of SA and CT, with age trajectories differing between high- and low-expanding cortical regions. This relationship adhered to allometric scaling laws rather than representing uniquely macaque–human differences: regional correspondence with human development was as large for expansion across nonhuman primates as between humans and macaque.

Keywords: area, cortex, development, evolution, thickness

Introduction

The human brain undergoes a protracted development, which is traceable postnatally well into the third decade (Tamnes et al. 2010; Lebel and Beaulieu 2011; Petanjek et al. 2011; Raznahan, Shaw, et al. 2011; Grydeland et al. 2013). This is unparalleled in comparison with other primates (Rilling 2014). Human cortical development is highly heterogeneous across regions (Shaw et al. 2008), with structural maturation proceeding in a coordinated manner across large-scale neural networks (Raznahan, Lerch, et al. 2011; Raznahan et al. 2012; Alexander-Bloch et al. 2013; Walhovd et al. 2014). These coordinated changes are related to development of cognitive abilities (Shaw et al. 2006), and evolutionary adaptation has been suggested as one principle governing the changes (Shaw et al. 2008), but limited evidence exists. Intriguingly, Hill et al. (2010) showed that cortical surface area (SA) in regions that appear expanded in humans, relative to the macaque monkey, tend to be those that change the most between human infancy and adulthood. It is unclear whether these relationships can be found for surface expansion alone, or also apply to cortical thickness (CT) (Gogtay et al. 2004), as these measures are shaped by independent genes (Rakic 1988; Panizzon et al. 2009) and neurobiological events (Rakic et al. 2009) and may even be negatively correlated in adults (Hogstrom et al. 2013).

It also remains to be established whether the overlap between human cortical development and interspecific cortical expansion, assessed based on macaque–human comparisons, is a result of human-specific evolutionary adaptations, or can be explained largely due to allometric scaling laws—i.e., that regional differences in cortical size can be predicted from increases in brain size (Rilling 2014). For example, it has been suggested that the relative immaturity of high-expanding areas at birth reflects evolutionary pressure for these areas to develop postnatally, taking advantage of interactions with the environment (Hill et al. 2010; Petanjek and Kostović 2012; Raznahan et al. 2012).

If regional differences in cortical size from a smaller to a larger brained nonhuman primate can predict regional differences in brain growth in humans, then the "evo–devo" relationship can be said to adhere to more general allometric scaling laws. Alternatively, the observed relationship between macaque–human differential cortical expansion and cortical development could represent more specific adaptations, which evolved relatively late in human evolution in parallel with the evolution of a larger complement of higher order association areas (Hill et al. 2010).

In the present study, we calculated age functions across development from 4 to 30 years based on cross-sectional brain scans from 331 participants. We then tested how the estimated developmental trajectories of CT, SA and cortical volume (CV) varied across the cortex as a function of cortical expansion in humans relative to interspecific expansion, namely comparisons between multiple primates—macaque vs. humans, marmoset vs. macaque, and marmoset vs. capuchin (Van Essen and Dierker 2007; Hill et al. 2010; Chaplin et al. 2013). We hypothesized that regions that are selectively larger in humans compared with nonhuman primates would show more protracted developmental curves for SA, and to a lesser degree CT.

We further tested whether the relationship between interspecific cortical expansion and human cortical development changed as a function of age, and hypothesized that the relationship would be strongest in younger children, when cortical development proceeds at a higher pace, and when the proportion of cortical variability stemming from early events during neurogenesis may be greater (Rakic 2009).

Finally, by examining the relationships between human cortical development and interspecific cortical expansion, we

aimed to elucidate the question of whether human cortical expansion during ontogenetic development is likely to be explained by uniquely human trait adaptations, or rather adheres to general allometric laws of scaling of brain size across primate species. Specifically, we hypothesized that if this represents human-specific adaptations, the data would show that the pattern of human cortical development more closely relates to the differential expansion between other primates to human, than between other primates species of different brain size. However, if the anatomical overlap were mostly a function of allometric scaling laws, we would expect a similar overlap with the pattern of expansion across other nonhuman primate species of different brain sizes.

Materials and Methods

Sample

A total of 331 children, adolescents, and young adults (157/174 males/females) aged 4.1 to 30.9 years (M = 14.4, SD = 6.6) were drawn from 3 Norwegian studies coordinated from the *Research group for Lifespan Changes in Brain and Cognition, LCBC* at the Department of Psychology, University of Oslo, Norway (The Norwegian Mother and Child Cohort Neurocognitive Study (MOBA)/ Neurocognitive Development/ Cognition and Plasticity Through the Life-span). Details of the samples are described in Table 1.

The studies were approved by the Regional Committee for Medical and Health Research Ethics. Participants were recruited through newspaper advertisements, and local schools and workplaces. Written informed consent was obtained from all participants older than 12 years of age and from a parent/guardian of volunteers under 18 years of age. Oral informed consent was obtained from all participants under 12 years of age. Participants had no self- or parent-reported history of neurological or psychiatric disorders, chronic illness, premature birth, learning disabilities, or use of medicines known to affect nervous system functioning. They were further required to be right-handed, speak Norwegian fluently, and have normal or corrected to normal hearing and vision (participants recruited for the MoBa study were not excluded based on handedness, 1 left handed, 4 ambidextrous). All participants' scans were examined by a neuroradiologist, and deemed free of significant injuries or conditions.

Participants above the age of 6.5 years were tested using the Wechsler Abbreviated Scale of Intelligence (WASI) (Wechsler 1999), and all participants scored above 80 on full-scale intelligence quotient (IQ). Participants below 6.5 years completed the vocabulary, similarities, block-design, and matrix subtests of the Wechsler Preschool and Primary Scale of Intelligence (WPPSI-III) (Wechsler 2002), all with mean scaled scores above 6.

MRI data Acquisition

Imaging data were acquired using a 12-channel head coil on a 1.5-Tesla Siemens Avanto scanner (Siemens Medical Solutions, Erlangen, Germany) at Oslo University Hospital Rikshospitalet. The pulse sequences used for morphometric analysis were 2 repeated 3D T_1-weighted magnetization prepared rapid gradient echo (MPRAGE), with the following parameters: Repetition time 2400 ms, echo time 3.61 ms, inversion time 1000 ms, flip angle 8°, matrix 192 × 192, field of view 192. Each volume consisted of 160 sagittal slices with voxel sizes 1.25 × 1.25 × 1.2 mm. Scanning time for each of these sequences was 7 min, 42 s. For the children between 4 and 9 years old in the MoBa sample, we used a parallel imaging technique (iPAT), acquiring multiple T_1 scans within a short scan time, enabling us to discard scans with residual movement and average the scans with sufficient quality. Previous studies have shown that accelerated imaging does not introduce significant measurement bias in surface-based measures when using FreeSurfer for image analysis, compared with a standard MPRAGE protocol with otherwise identical voxel dimensions and sequence parameters (Wonderlick et al. 2009), which is in accordance with our own analyses. The protocol also included a 25-slices coronal T_2-weighted fluid-attenuated inversion recovery sequence (TR/TE = 7000–9000/109 ms) to aid the neuroradiological examination.

Table 1
Sample characteristics

Sample	*N*	Age, mean	Range	Male/female
1	83	6.6	4.1–9.3	38/45
2	187	14.5	8.2–21.6	93/94
3	61	24.6	20.0–30.9	26/35
Total	331	14.4	4.1–30.9	157/174

1: The Norwegian Mother and Child Cohort Neurocognitive Study. 2: Neurocognitive Development. 3: Cognition and Plasticity Through the Life-span.

Image Analysis

Each MPRAGE was visually inspected, and rated for movement and artifacts on a scale from 1 to 3 (1: excellent, 2: some movement/artifacts, 3: major movement/artifacts). Only participants with at least 2 acquisitions rated excellent were included in the analyses, and if the participants had more than 2 scans rated excellent, the 2 best acquisitions were used. Twenty-two participants did not have 2 acquisitions rated excellent, and were excluded from the study. The datasets were processed and analyzed at the Neuroimaging Analysis Lab, University of Oslo, with FreeSurfer 5.1 (http://surfer.nmr.mgh.harvard.edu/). This procedure yields a measure of CT, SA, and CV for each person at each point on the reconstructed surface (Dale and Sereno 1993; Dale et al. 1999; Fjell et al. 2010), and is capable of detecting submillimeter differences between groups (Fischl and Dale 2000). SA maps of the gray and white matter boundary were computed for each subject by calculating the area of every triangle in a cortical surface tessellation. The triangular area at each point in native space was compared with the area of the analogous points in standard space (fsaverage) to give an estimate of SA expansion or contraction continuously along the cortical surface (Dale et al. 1999; Fischl et al. 1999). Maps were smoothed using a circularly symmetric Gaussian kernel across the surface with a full-width at half-maximum (FWHM) of 30 mm, and averaged across participants using a nonrigid high-dimensional spherical averaging method to align cortical folding patterns (Fischl et al. 1999). This procedure provides accurate matching of morphologically homologous cortical locations among participants on the basis of each individual's anatomy while minimizing metric distortion. The cortical surface was then parcellated according to procedures described previously (Fischl et al. 2004; Desikan et al. 2006).

Existing and publicly available maps of cortical expansion between the macaque monkey and humans (Van Essen and Dierker 2007; Hill et al. 2010) were downloaded, and combined with expansion maps between marmoset and macaque and between marmoset and capuchin (Chaplin et al. 2013). The maps were created with surface-based registration methods and based on a combination of functional and structural homologies, described elsewhere (Orban et al. 2004). The 3 maps of cortical expansion were registered to the same template brain that was used for visualization of the human surface data ("fsaverage," distributed with FreeSurfer).

We also obtained maps of T_1w/T_2w ratio in a sample of 85 children and adolescents aged 8–20. The maps were created by first registering the T_2w images to the T_2w images using *bbregister*, a part of FreeSurfer, before the transform was applied using FSL's *applywarp*. The T_2w volume was then divided on the T_2w volume, and T_1w/T_2w values were extracted for each vertex at a distance of 0.2 mm into the cortex. The resulting maps were averaged across participants to yield a measure of mean intracortical myelin. The method and sample is described in detail elsewhere (Glasser and Van Essen 2011; Grydeland et al. 2013).

Statistical Analysis

Initially, we tested for interactions between sex and age on all three cortical measures, CT, SA, and CV. When no significant interactions

were found when corrected for false discovery rate (<0.05), we entered sex as a covariate in all subsequent vertex-wise general linear model (GLM) analyses, but did not perform separate analyses on the effect of sex. We chose to control for sex, as this variable is highly related to increased SA and CV in males across the age range. While brain development is also related to puberty and testosterone levels (Raznahan et al. 2010; Nguyen et al. 2013), IQ (Shaw et al. 2006), and might be related to socioeconomic status (SES) and body mass index (BMI) (Lawson et al. 2013), we did not obtain measures of puberty, testosterone, SES, or BMI across all participants, and the IQ measures were not directly comparable across the age range. We correlated age with the 3 cortical measures, CT, SA, and CV separately. This was done first in the complete sample, before we created 3 groups based on age; subsequently, we performed the corresponding correlation analyses in each of the 3 cohorts.

To delineate age trajectories, a nonparametric local smoothing model, the smoothing spline, implemented in Matlab, was fitted to the data. We have previously shown that this approach gives less biased solutions than the more commonly employed higher order polynomial functions $y = C + \beta_1 \text{age} + \beta_2 \text{age}^2$ for mapping curvilinear trends (Fjell et al. 2010, 2014). We employed this method both subject-wise on the parcellation data and for the creation of surface-based vertex-wise maps of estimated yearly change of CT, SA, and CV.

First, maps of estimated mean yearly cortical change (SA and CT) for the three age groups described above (4–10, 10–17, 17–30 years) were correlated vertex-wise with the maps of cortical expansion between macaque and human, and the mean of the expansion maps between the monkeys (marmoset/capuchin and marmoset/macaque). Second, the map of expansion between macaque and human was z-transformed, yielding a mean of 0 and a standard deviation of 1. Two regions of interest (ROIs) were created, representing areas of relative high or low cortical expansion between macaque and human, by identifying continuous sets of vertices meeting threshold criteria of ±0.5 SD, and covering a minimum area of 2000 vertices in the z-transformed maps. Mean CT and SA from the 2 ROIs were extracted for every subject, which we in turn fitted the above-mentioned smoothing spline model to, and computed estimated yearly rate of change.

Finally, we tested how a measure representing intracortical myelin content was related to CT in the 2 ROIs defined above, representing regions of high and low interspecific cortical expansion. Using maps of the mean ratio between T_1-weighted and T_2-weighted image intensities in a sample of 8–20 year olds ($n = 85$), we tested whether a measure of intracortical myelin differed between the high- and low-expanding areas with a permutation-based approach. To account for spatial dependence between neighboring vertices, we iterated a t-test of the intracortical myelin value from 200 randomly chosen vertices in each of the ROIs, repeated 5000 times.

Results

Global Analyses

Mean CT, total SA, and total CV across the whole cortex were plotted against age, and a smoothing spline function fitted to the data (Fig. 1). Interestingly, CT showed a continuously negative trajectory throughout the age range of the sample (4–30 years), with no tendencies for increases at any age. In contrast, SA was positively related to age until about 12 years, before the curve flattened, indicating little SA differences after early teenage. CV, as a product of CT and SA, showed an initial positive age relationship, before decreasing through the rest of the age range. Corresponding data for individual ROIs can be found in supplementary Figure 1. There were no significant sex differences in mean CT, but boys had greater total SA and CV than girls ($P < 0.001$).

Vertex-wise Analyses

Correlating age with CT, SA, and CV vertex-wise revealed highly significant relationships over most cortical vertices (Fig. 2) when we entered the whole sample in the analysis ($P < 0.05$, corrected). When we split the sample into 3 age cohorts (Table 2), negative correlations between age and CT were found in all 3 groups across widespread cortical areas, while no positive correlations were seen. Significant positive age–SA correlations were found across the cortex in the youngest age group, while a more mixed pattern was seen for the 2 older groups. CV showed a mixed pattern of positive (mainly lateral temporal and an area around the central sulcus) and negative correlations (occipital and medial orbitofrontal cortex) in the youngest group, negative correlations covering most of the cortical surface in the middle group, before weaker and more scattered negative correlations in the oldest group were seen. There were no significant interactions between sex and age on CT, SA, or CV.

Next, we fitted a smoothing spline function to each vertex of the cortical surface in order to calculate age functions (estimated "rate of change") in CT, SA, and CV continuously across the age range (Fig. 3). CT showed the most negative age function in the posterior parietal, occipital, and orbitofrontal cortices from 4 years (estimated >1.5% annually), which gradually leveled off through childhood and adolescence, yielding a

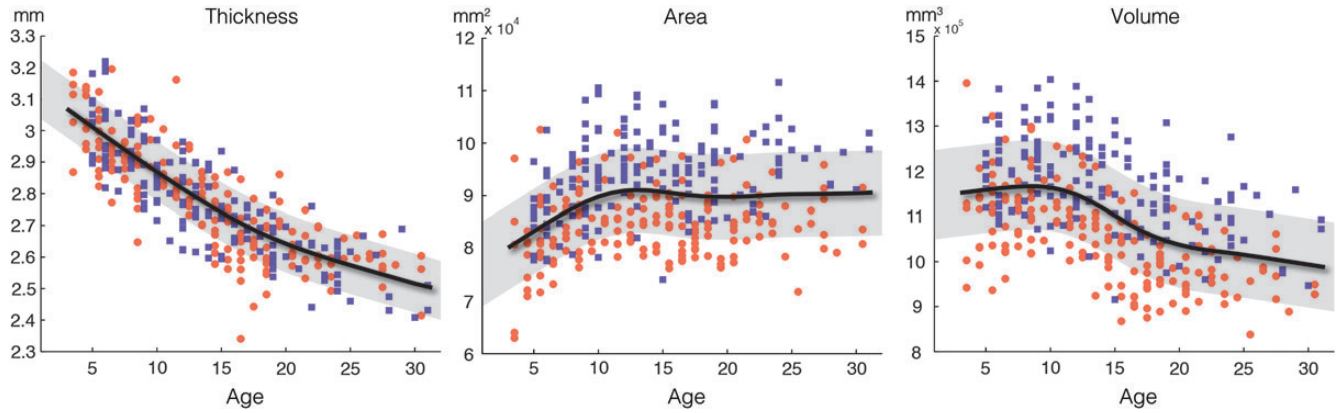

Figure 1. Scatterplots and smoothing spline fitted trajectories of mean cortical thickness (left), total surface area (middle), and cortical volume (right) across the cerebral cortex. Boys in blue squares, girls in red circles. 95% confidence interval for the whole range in gray .

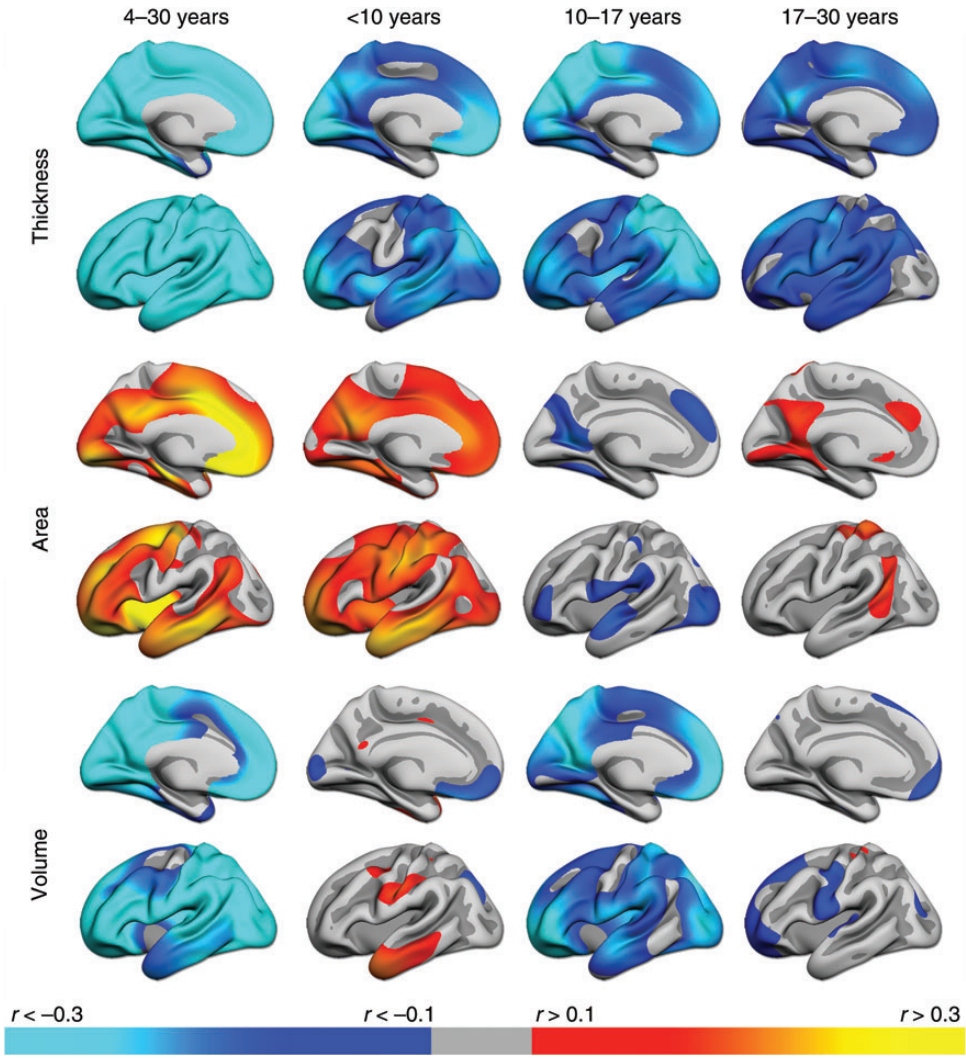

Figure 2. Age correlated with cortical thickness, area and volume. Left to right; correlations in the complete sample, participants aged 4 up to 10, participants aged from 10 up to 17, participants aged from 17 to 30 years. Only significant correlations after employing FDR corrections are showed. Cyan represents >0.3 negative correlations, yellow represents >0.3 positive correlations.

Table 2
Characteristics of the subject groups used in the correlation analyses displayed in Figure 2

Subgroup	*N*	Age, mean	Range	Male/female
1	110	7.2	4.1–10.1	56/54
2	110	13.8	10.3–17.0	55/55
3	111	22.0	17.1–30.9	63/48

spatially more homogenous pattern of negative effects from the end of the teens. Weaker effects were seen in the pre- and postcentral gyri, and the medial temporal lobes showed small or no effects.

The largest early SA effects were seen in cingulate, insular, and lateral temporal- and prefrontal regions (estimated >1.5% annual increase). In contrast to CT, for which occipital and parietal regions showed the largest effects, relatively small effects in SA were seen in posterior cortical regions. After positive effects for SA to about 12–15 years, effects were relatively stable around zero in most all regions, except small, continued increases in posterior parts of the medial temporal lobe, cingulate cortex, and later precuneus. Approaching the end of the age range, small decreases were also seen around the central sulcus. CV displayed a mixed pattern of positive and negative effects early on, dependent on the relative contributions of CT and SA.

Cortical Expansion Across Primates

To quantify the overlap between regional differences in cortical development and between-species expansion, expansion map between macaque and human, and the mean expansion maps between marmoset and macaque, and marmoset and capuchin, were correlated with maps of human cortical development, vertex by vertex. This yielded overall measures of anatomical correspondence between maps. Both SA and CT development correlated with expansion maps between macaque and humans (SA: $r = 0.13$, CT: $r = 0.12$, $P < 0.05$ by permutation testing). Broken down in the 3 age groups, we observed a tendency for numerically stronger correlations at younger than older ages (Fig. 4). This pattern was present across all primate comparisons (SA: macaque → human; $r =$ 0.19/0.15/0.04 for young/middle/old, respectively, marmoset → [capuchin and macaque]; 0.20/0.22/−0.04. CT: macaque →

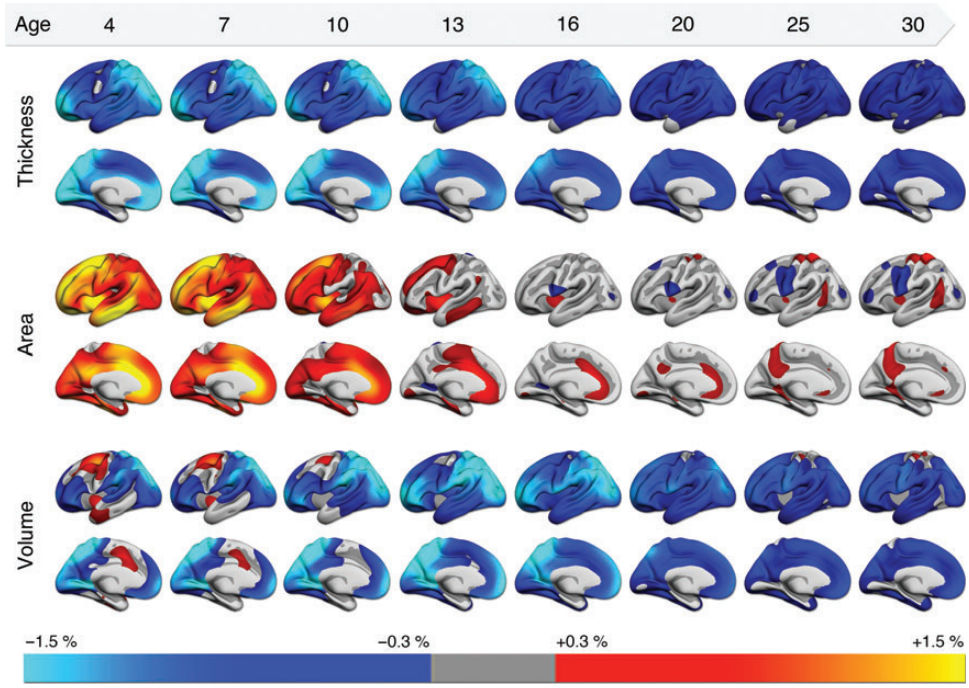

Figure 3. Age functions (annual rate of change in percent) at ages 4, 7, 10, 13, 16, 20, 25, and 30 years, medial and lateral views. Top to bottom; thickness, area, and volume. Cyan represents 1.5% reduction, yellow 1.5% increase per year.

human; 0.18/0.13/0.06, marmoset → [capuchin and macaque]; 0.29/0.23/0.15). Interestingly, both for SA and CT, the correlations were as strong between human cortical development and nonhuman–primate expansion marmoset → (macaque and capuchin), as between human cortical development and the macaque → human expansion.

Based on the global overlap between development and interspecific expansion identified above, we wanted to map the age trajectories of CT and SA as a function of degree of cortical expansion between macaque and humans. We defined one ROI of relatively low (<−0.5 SD) cortical expansion from macaque to human, including a large cluster covering the occipital lobe and parts of the inferior posterior medial parietal and temporal lobes, as well as a smaller cluster aligning with primary somatosensory cortex (Fig. 5). The definitions of the ROIs were done on expansion maps based on comparisons between macaque and humans independent of the current human sample. Similarly, we defined one ROI with relatively large cortical expansion (>0.5 SD), including one posterior cluster covering the lateral parts of the temporal and parietal lobe, and 2 frontal clusters covering lateral prefrontal cortex and the cingulate regions. Across the age range, high-expanding regions had larger CT ($t = 89.80$, $P < 0.05$) than low-expanding regions. By mapping CT across the age range, low-expanding regions showed larger negative CT effects from the beginning of the age range than high-expanding regions, indicating more rapid early development, before they approached the same level of negative effects at about 20 years. High-expanding regions showed a relatively more protracted trajectory, in the sense that the yearly negative effects were not reduced to the same degree through adolescence compared with the initial levels. Still, net magnitude of development, in terms of thinner cortex, across the age range appeared larger for low versus high-expanding regions. In contrast, absolute positive SA effects were larger in high-expanding areas at the youngest ages, before effects converged toward zero for high- and low-expanding regions from about 20 years. Thus, the opposite pattern of that observed for thickness was seen.

Due to previous research suggesting myelin content to be lower in high-expanding regions (Glasser and Van Essen 2011), and ongoing myelinetaion as one factors affecting cortical thinning in development (Sowell et al. 2003), we quantified intracortical myelin content. The differences in intracortical myelin between high- and low-expanding regions were tested based on T_1w/T_2w ratio maps. A t-test of intracortical values between the 2 ROIs revealed that the low-expanding regions had higher intracortical myelin values than the high-expanding regions (median P-values for 5000 independent t-tests were 2.539×10^{-9} for left, and 8.362×10^{-10} for right hemisphere). The similarities between the T_1w/T_2w ratio maps and macaque → human expansion are depicted in Figure 6.

Discussion

Human brain evolution is characterized by a tremendous expansion in the SA of the human cerebral cortex, together with a much more modest increase in thickness (Rakic 2009). The present results show that regional differences in cortical expansion between macaque and humans are related to development of both CT and SA. Interestingly, the expansion–development relationships were not stronger when regional expansion was calculated based on comparisons between macaque and humans, than between nonhuman primates of different sizes. This indicates that human-specific adaptations may not be responsible for the observed overlap between development and expansion between macaque and human, and suggests that allometric scaling laws that apply to brains of increasing size can better explain the findings.

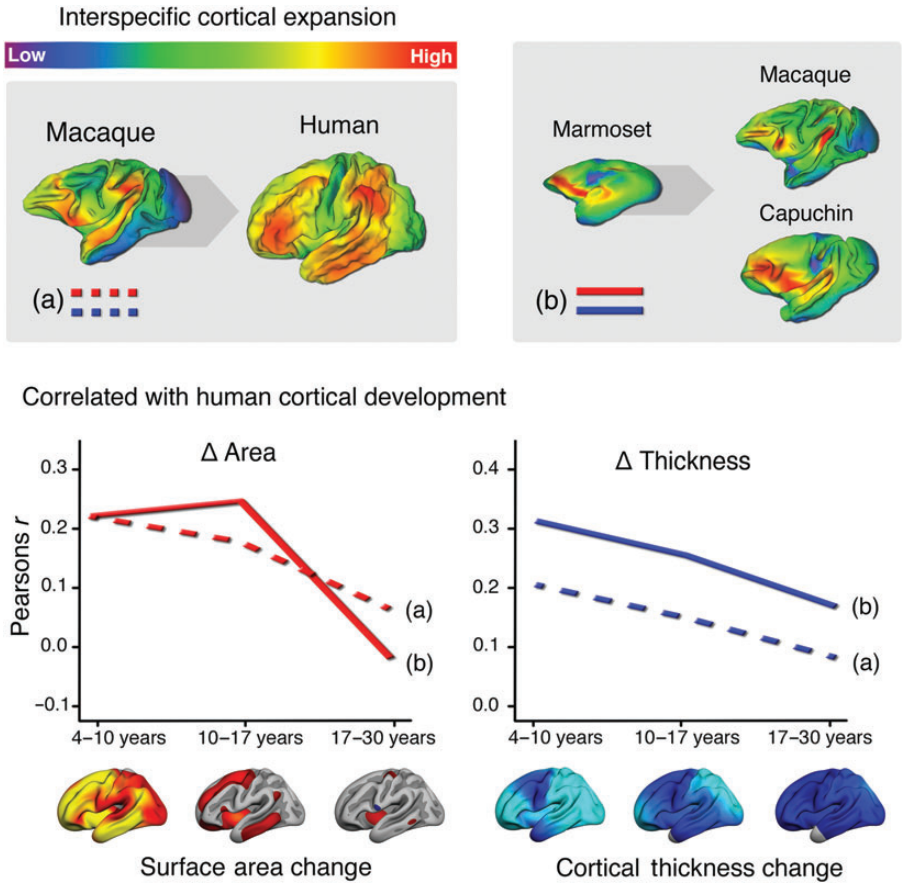

Figure 4. Top row; maps of surface area expansion in humans relative to macaque (left), and nonhuman primate surface area expansion (right). Bottom row; mean vertex-wise correlations between above expansion maps and mean human surface area and cortical thickness development in the three age groups. Left/red; surface area, right/blue; thickness. Dotted lines; macaque → human expansion, solid lines; marmoset → (macaque/capuchin) expansion.

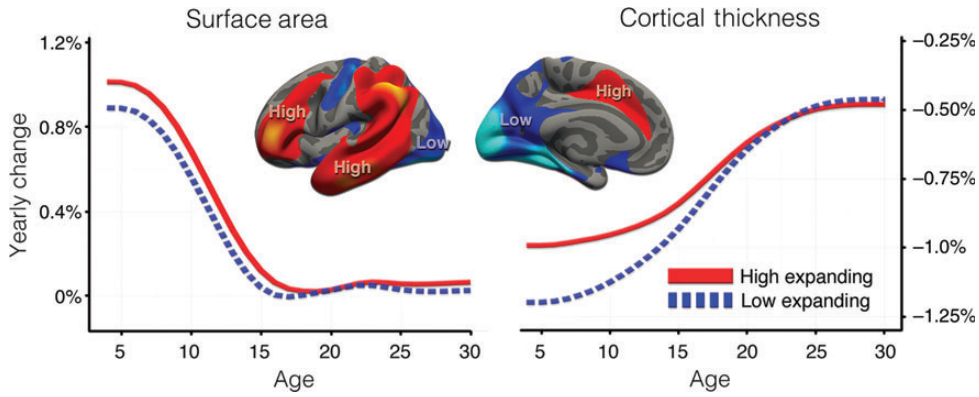

Figure 5. Annualized change in the human sample, in areas of relatively high cortical expansion in human relative to macaque (>0.5 SD, red/yellow) and relatively low expansion (<−0.5 SD, blue/cyan). Left, surface area; right, cortical thickness.

In addition, contrary to what has been the dominating view on maturation of CT (Shaw et al. 2008; Raznahan, Shaw, et al. 2011), we observed that CT showed a monotonically negative age function from 4 years of age, across almost the entire cortex, without initial growth during the first years of the tested age range. The implications of the findings are discussed below. The current analyses and results are based on cross-sectional data, and need to be interpreted with this in mind.

Organizing Principles of Maturation: Expansion Across Primates

Comparative studies have revealed that cortical SA expands much more markedly than CT. For example, a 1000-fold increase in SA between mouse and human is accompanied by only a 2-fold increase in CT (Rakic 1995, 2009). SA has also increased much more than CT during hominid evolution (Van Essen and Dierker 2007), thus, a closer relationship between

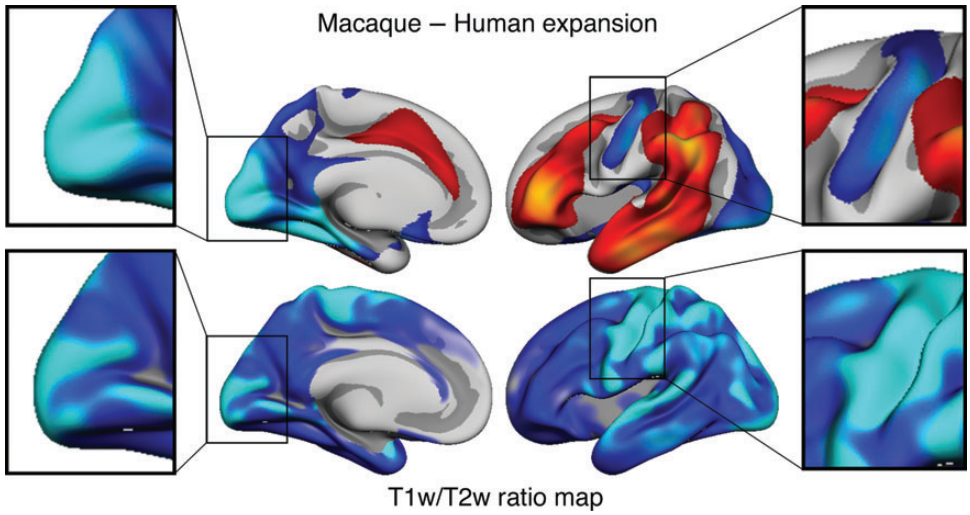

Figure 6. Similarities of human–macaque surface area expansion (top) and intracortical myelin content expressed by T_1w/T_2w ratio maps (bottom).

SA development and interspecific expansion across primates than CT and expansion was envisioned. Previously, Hill et al. (2010) pointed to similarities in regional expansion patterns of SA when comparing expansion maps from infants to adult humans and expansion maps from macaque to humans. In infant years, the human cerebral cortex undergoes marked expansion, more than doubling its SA between birth and 2 years of age (Li et al. 2013; Lyall et al. 2014). Interestingly, while CT is reported to reach 97% of adult values at age 2, the corresponding number for SA is 69% (Li et al. 2013; Lyall et al. 2014). After the age of 10–15 years, however, CT continues to decrease, while SA tends to stabilize (Raznahan, Shaw, et al. 2011). This makes CT a very relevant measure of the continuous cortical development through childhood and adolescence.

In the present data, the correlations between interspecific expansion and CT were comparable in magnitude with the correlations between interspecific expansion and SA. Thus, although CT and SA appear to be shaped by independent genetic factors (Rakic 1988; Panizzon et al. 2009) and neurobiological events (Rakic et al. 2009), and may even be negatively correlated in adults (Hogstrom et al. 2013), contrary to what could have been envisioned, they still seem to be related to the same rough index of regional growth in brain size.

The correlations between interspecific expansion and rate of human cortical development did not depend on whether interspecific expansion was computed between macaque and human brains, or across a wider range of primate brains, indicating that rather than representing human-specific adaptations, the interspecific expansion—development overlap may to a large degree be explained by allometric scaling laws (Rilling 2014). This pattern appears when analyzing the cortex on the gross level of cortical arealization, but that is not to say that the human brain is merely an allometrically scaled variant of the macaque brain, as several lines of evidence show that cortical adaptions have occurred during evolution (Clowry et al. 2010; Geschwind and Rakic 2013), related to gene expression (Enard et al. 2002; Dorus et al. 2004; Vallender et al. 2008; Lambert et al. 2011; Bufill et al. 2013; Sassa 2013), microstructural properties (Chen et al. 2013), and network organization (Buckner and Krienen 2013).

It is suggested that the structures formed by neurons which are born relatively late in development also grow disproportionately larger as absolute brain size increases (Finlay et al. 2001). This provides a potential mechanism to account for the regional overlap between human cortical development and between-primate cortical expansion (Rosa and Tweedale 2005). It is possible that miniscule changes or modulations of genes regulating the length and speed of the first phase of cell division, and the timing of onset and length of the second phase, are responsible for the patterns we observed for SA and CT, respectively, in accordance with the radial unit hypothesis of cortical development (Rakic 1995; Rakic et al. 2009). For instance, previous developmental studies have demonstrated a relationship between prenatal variables such as birth weight and cortical expansion and cognitive function years later (Fjell et al. 2012; Raznahan et al. 2012; Walhovd et al. 2012), and Petanjek and Kostović (2012) suggested a connection between evolutionary changes operating on the mechanisms regulating brain development and early, prenatal influences, causing long-term effects on cortical expansion.

We hypothesized that regions with high cortical expansion between macaque and human would display more protracted developmental trajectories for SA and to some extent CT. For CT, the high-expanding areas showed a possibly more protracted trajectory, but the absolute effect across the age range was larger in the low-expanding regions. For SA, the absolute effect was larger in the high-expanding regions, but the trajectories were quite similar. The estimated rate of change in SA correlated more strongly with differential cortical expansion between macaque and human at young age than later, as could be expected from the rapid reduction in estimated annual expansion rate from 4 to 15 years. Interestingly, the same pattern of reduced relationships with higher age was also seen for CT, where substantial negative age effects were seen through the entire age range.

While monotonous CT reductions were seen across the age range, the processes driving the changes are probably not similar in young and in older age. With increasing age, the cortical changes observed would be less related to developmental processes, and relatively more affected by aging-related processes. The pattern of early maturation of the low-expanding

ROI, and the relatively late, protracted maturation of the high-expanding areas, corresponding quite well with sensory–motor regions and higher order association areas, respectively, resonates well with a hierarchical sequence of maturation of the neocortex (Yakovlev and Lecours 1967; Rakic et al. 1994; Guillery 2005; Bourne and Rosa 2006; Burman et al. 2007). Still, it may be that the fundamental principles for regionalization of brain growth are more strongly hard-coded in early development than in later, as the variance in CT attributed to genetic factors has been shown to decrease with age (Schmitt et al. 2014).

Trajectories of CT, SA, and Volume

In addition to investigating the principles governing regional differences in estimated change, we were interested in changes in the age effects across the age range per se. SA followed a nonlinear and nonmonotonous trajectory with positive age effects until about 12 years, before remaining stable for the rest of the studied age span, in accordance with previous studies (Raznahan, Shaw, et al. 2011; Brown and Jernigan 2012; Shaw et al. 2012; Wierenga et al. 2014). The early SA increases affected CV relatively more than did the CT reductions, which resulted in a net CV estimated increase until ~10 years of age. At this time, the positive age effect on SA subsided to such a degree that it was offset by the continuing negative CT effects. When estimated SA growth subsided around age 12, CV was mostly affected by CT, thus following a mostly linear negative trajectory for the rest of the age range.

Interestingly, CT was found to have a monotonic negative relationship with age across the whole cortex. Both the global cortical, ROI-based and vertex-wise analyses showed monotonous CT decrease. Even with a start age of 4.1 years, and reasonably high sampling density at the lower end of the age range, no indication of a positive age-thickness relationship was seen in any region. This is different from previously reported CT increases until 8–10 years (Gogtay et al. 2004; Shaw et al. 2008; Raznahan, Shaw, et al. 2011), but in concordance with several recent studies (Mutlu et al. 2013; Nguyen et al. 2013; Mills et al. 2014; Wierenga et al. 2014), although many of these did not sample individuals aged below 6–7 years, thus potentially missing an initial peak of CT. The results are also in coherence with the large multicenter Pediatric Imaging, Neurocognition, and Genetics (PING) study (Brown and Jernigan 2012; Brown et al. 2012), and recent infant imaging studies indicating that thickness might reach peak levels already at 2 years while SA is relatively less developed at that age (Li et al. 2013; Lyall et al. 2014). Studies of marmoset monkey, where CT peaks very early after birth, also supports the possibility that CT in humans peaks earlier than we are able to detect in the present study (Missler, Eins, et al. 1993).

The reasons for the discrepant findings are not obvious. CT reductions during childhood may reflect use-dependent synaptic pruning (Bourgeois and Rakic 1993; Missler, Wolff, et al. 1993; Bourgeois et al. 1994), maturation of white matter causing proliferation of myelin into the neuropil (Sowell et al. 2003), cortical stretching (Seldon 2005), or a combination of the above. In macaque, net synaptogenesis occurs only in a period spanning the 2 last months of pregnancy through the 2 first postnatal months. After this period, a decrease in synaptic density continues in most regions until puberty, by then the synaptic density decrease accelerates (Rakic et al. 1986; Bourgeois and Rakic 1993; Bourgeois et al. 1994). In humans, synaptogenesis occurs from at least 6 months after gestation until 15 months after birth, before reduction in synaptic density becomes the net results of the synaptogenic and pruning processes (Huttenlocher and Dabholkar 1997). The timing of synaptogenic processes, however, shows regional heterogeneity across cortical layers (Huttenlocher 1990; Petanjek et al. 2008), and across regions, with sensory and motor areas reaching peak synaptic density at age 3–4 months, while prefrontal cortex reaches its peak not until 15 months. Consequently, there is little reason to expect an increase in CT attributed to synaptogenic processes alone in the age range under study, as we can expect net synaptic density loss in this age range. Use-dependent synaptic elimination, or pruning, can therefore not be ruled out as one of the probable explanations for the monotonous CT reductions we observed. However, there is large individual variation in cortical changes that learning experiences and other epigenetic factors might influence. Dendritic length, for example, has been shown to be related to educational level (Jacobs et al. 1993), but it may not always be the case that changes in dendritic length follows changes in spine density, and the measures may even be oppositely related (Kolb et al. 2008).

Another probable factor affecting CT during development is maturation of white matter underlying the neuropil of the neocortex. The time-course of myelination follows that of synaptogenesis and elimination, with late and protracted myelination of prefrontal areas (Yakovlev and Lecours 1967; Sowell et al. 2004). The proliferation of myelin into the neuropil can possibly affect how the surface model of the boundary between GM and WM is reconstructed, pushing the boundary outward in older participants with more progressed myelination. The present finding that the regions with little cortical expansion between macaque and human, showed the largest early CT reductions, and also had the highest intracortical myelin content (see Fig. 6), lends support to the possibility that white matter maturation can be one of several factors driving the cortical thinning seen. Thus, highly myelinated cortical regions show large negative age effects on CT early in development, which level off with increasing age, and are generally low-expanding between a smaller macaque and larger human brains.

A third possible explanation of the CT reductions needs to be considered in concert with the SA increases. While we did not examine WM volume in the present study, previous results in a comparable sample, although not extending below 8 years of age, showed extensive WM volume growth from 8 to 30 years (Tamnes et al. 2013). Expansion of WM could possibly mechanically affect the structure of the cortical layer by exerting pressure tangential to it. This model of brain development suggested by Seldon (2005), posits that pressure on the cortical layer exerted by increased myelination may stretch it, like inflating a balloon, thereby simultaneously increasing SA and decreasing CT, which would explain the observed inverse relationship between local arealization and CT in adults (Hogstrom et al. 2013).

None of these possible mechanisms give strong indications about what to expect in terms of early increase versus decrease of CT, but all are consistent with the observed pattern of monotonous CT reductions with simultaneous SA increases. A point of discrepancy between studies that give a hint that the conflicting CT trajectories reported may partly be due to differences in analysis methods is the case of sex differences in CT.

For instance, consistent with Raznahan et al. (2011), we find no sex differences in CT trajectories. In the Raznahan et al. (2011) study, however, boys were reported to have larger mean CT than girls, while no such sex difference exists in our data. In previous studies, we did not find significant sex differences in absolute CT by use of FreeSurfer, even when analyzing MRI scans from 1143 participants from a total of 7 different independent samples (Fjell et al. 2009; Tamnes et al. 2010). To the degree that sex differences in CT tend to be reported with some algorithms (CIVET) and not others (FREESURFER), this may reflect a methodological difference in how thickness is calculated, that could carry over to estimations of developmental trajectories independently of any sex effects per se. Different approaches may all reflect meaningful neurobiological properties of the cortex, with some papers using FreeSurfer seemingly yielding results that differ more between CT and SA, and hence also CV, compared with papers using CIVET, reporting more similar trajectories across metrics. This could also reflect differences in sample composition or recruitment procedures, or image parameters and quality, for example, related to MRI contrast parameters or in-scanner head movement. Such issues could be resolved by studies directly testing the effects of different CT estimation procedures on developmental trajectories based on the same set of data.

Conclusions

In conclusion, we found that differential cortical expansion across primates with different brain sizes was related to regional maturation of both SA and CT. This relationship seemed to adhere to allometric scaling laws that govern the expansion of the primate brain (Chaplin et al. 2013). Both metrics also tended to be most closely related to interspecific expansion in early years, with correlations steadily dropping at higher ages. Of further interest, CT followed a monotonic negative trajectory from 4 to 30 years, with no indication of growth at any age within this range, while SA displayed a more complex trajectory with areal expansion in the years from 4 to ~12, with relatively little further development. We believe that the "evo–devo" approach is fruitful in shedding light on the processes driving cortical development, both ontogenetic and phylogenetic, and we welcome future endeavors that include chimpanzees and other great apes in similar comparisons.

Supplementary Material

Supplementary Material can be found at http://www.cercor.oxfordjournals.org/.

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Notes

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