Cerebral Cortex, 2019; 1–19

doi: 10.1093/cercor/bhz053
Original Article

ORIGINAL ARTICLE

Maturational Changes in Human Dorsal and Ventral Visual Networks

Kristina T. R. Ciesielski[1,2], Moriah E. Stern[2], Adele Diamond[3], Sheraz Khan[1,4], Evelina A. Busa[1], Timothy E. Goldsmith[2], Andre van der Kouwe[1], Bruce Fischl[1,4] and Bruce R. Rosen[1,4]

[1]Department of Radiology, MGH/MIT/HMS A. A. Martinos Center for Biomedical Imaging, Massachusetts General Hospital, Harvard Medical School, Charlestown MA 02129, USA, [2]Pediatric Neuroscience Laboratory, Department of Psychology, Psychology Clinical Neuroscience Center, University of New Mexico, Logan Hall, Albuquerque NM 87131, USA, [3]Department of Psychiatry, University of British Columbia, Vancouver BC V6T2A1, Canada and [4]Harvard-Massachusetts Institute of Technology, Division of Health Sciences and Technology, Cambridge, MA 02139, USA

Address correspondence to: Kristina T. Rewin Ciesielski. Email: KCIESIELSKI@mgh.harvard.edu

Abstract

Developmental neuroimaging studies report the emergence of increasingly diverse cognitive functions as closely entangled with a rise-fall modulation of cortical thickness (CTh), structural cortical and white-matter connectivity, and a time-course for the experience-dependent selective elimination of the overproduced synapses. We examine which of two visual processing networks, the dorsal (DVN; prefrontal, parietal nodes) or ventral (VVN; frontal-temporal, fusiform nodes) matures first, thus leading the neuro-cognitive developmental trajectory. Three age-dependent measures are reported: (i) the CTh at network nodes; (ii) the matrix of intra-network structural connectivity (*edges*); and (iii) the proficiency in network-related neuropsychological tests. Typically developing children (age ~6 years), adolescents (~11 years), and adults (~21 years) were tested using multiple-acquisition structural T1-weighted magnetic resonance imaging (MRI) and neuropsychology. MRI images reconstructed into a gray/white/pial matter boundary model were used for CTh evaluation. No significant group differences in CTh and in the matrix of edges were found for DVN (except for the left prefrontal), but a significantly thicker cortex in children for VVN with reduced prefrontal ventral-fusiform connectivity and with an abundance of connections in adolescents. The higher performance in children on tests related to DVN corroborates the age-dependent MRI structural connectivity findings. The current findings are consistent with an earlier maturational course of DVN.

Key words: brain maturation, cortical thickness, dorsal and ventral visual networks, network connectivity, neuropsychological proficiency

Introduction

Our brains display a similar overall anatomical architecture, yet our cognitive behavior is distinctly diverse. The major process underlying such diversity, apart from genetic determination, is the experience-dependent neuronal connectivity emerging from selective elimination of the initially over-generated synapses (Changeux and Danchin 1976; Huttenlocher 1979; Rakic et al. 1986). An important insight into the puzzle of diversity may, thus, come from examining the developmental dynamics of cortical thinning and structural cortical connectivity associated with two long-range visual processing networks, Dorsal and Ventral (DVN and VVN; Maunsell and Van Essen

1983; Mishkin et al. 1983; Haxby et al. 1991; Goodale and Milner 1992; Corbetta and Shulman 2002; Fox et al. 2006). DVN and VVN constitute a model of major significance for understanding the neuronal diversity in typical cognitive development and etiology of neurodevelopmental disorders (Atkinson 2000; Atkinson and Braddick 2003; Iliescu and Dannemiller 2008; Johnson et al. 2008; Klaver et al. 2011), yet, the essential question as to which network may develop earlier and, thus, lead the developmental neuro-behavioral trajectory, remains unsolved.

The current study relies on a compelling body of knowledge suggesting that a consistent organizational principle drives maturation of the human brain cortex. Accordingly, two regions of the cortex that are functionally related may also display strong structural covariance (Seeley et al. 2009; Zielinski et al. 2010; Zhou et al. 2012; Alexander-Bloch et al. 2013), such as in cortical thickness (CTh) or in volume of white-matter pathways. For example, studies of brain integration using diffusion tensor or spectra imaging (DTI, DSI) of white-matter pathways and functional resting state (RS)-fMRI demonstrate high correlational convergence (Bullmore and Sporns 2009; Hagmann et al. 2010). In agreement, in the present study we define a complex "network", as a set of "cortical nodes" with well-determined functional anatomy, correlated time-courses ("network edges") in task-induced activation or in the rate of developmental anatomical transformations (Bullmore and Sporns 2009). Complex networks form gradually through a process of modularity or clustering of nodes into high-density intra-network connections, but decreasing non-directional correspondence with inter-network modules (Girvan and Newman 2002; Bullmore and Sporns 2009). Here, we acquired data on cortical structural connectivity displayed by correlations of age-dependent thinning in CTh between intra-network nodes. As noted above, cortical nodes that are connected will demonstrate a significant pair-wise correlative "relationship" (a significant network edge) of progressive changes in CTh. Since changes reported as prominent during developmentally sensitive time windows of early childhood and adolescence parallel progress in cognitive proficiency (Sporns et al. 2004; Casey et al. 2005; Bassett and Bullmore 2006; Bressler et al. 2008; Shaw et al. 2008; Blakemore et al. 2010; Raznahan et al. 2011; Walhovd et al. 2016), we expected that in children cognitive skills specific to the earlier-maturing network will be closer to adult performance.

The development of CTh in humans is marked by nonlinearity with a density of the cortex displaying a rise-fall pattern specific to a brain region and age of a child, with elevated CTh in early childhood and its gradual decrease into later adolescence (Giedd et al. 1999; Sowell et al. 2004; Gogtay et al. 2008). The pattern of maturational thinning follows a developmental sequence, with the primary somato-sensory and visual sensory regions maturing first, and with the higher-order association prefrontal cortices showing a protracted course (Sowell et al. 2001; Gogtay et al. 2004; O'Donnell et al. 2005; Shaw et al. 2008). In this approach, an investigation of connectivity between functional nodes of a particular network require a-priori knowledge about their functional and anatomical significance (Bressler and Tognoli 2006).

The functional anatomy of cortical nodes in DVN and VVN is well researched. Earlier lesion studies in monkeys led to the definition of two anatomically and functionally separate cortical streams both emerging in the primary visual cortex (Mishkin et al. 1983). Earlier reports related DVN to the posterior-inferior parietal and the dorsal superior frontal (SF)/premotor cortex, and VVN to "the occipitotemporal cortex, the fusiform area and the ventrolateral/medial prefrontal cortex". Multiple clinical and functional imaging studies associated nodes of DVN to visual-spatial localization, visually guided "tool" manipulation, visual working memory and attentional eye movement control (Jones and Powell 1970; Mesulam et al. 1977; Bachevalier and Mishkin 1986; Desimone and Ungerleider 1989; Van Essen et al. 1992; Friedman and Goldman-Rakic 1994; Courtney et al. 1998; Culham and Kanwisher 2001; Rizzolatti and Matelli 2003; Chen et al. 2008, 2017; Milner and Goodale 2008). A discrete stream of fibers, providing a specific cortical–cortical connectivity between the posterior and anterior nodes of the DVN includes the occipital-frontal fasciculus and superior longitudinal fasciculus II (Pandya and Seltzer 1982; Yeterian and Pandya 1993). In contrast, the cortical nodes of VVN have been associated with identification of objects via fine detail and color feature extraction, and with categorical semantic object labeling (Kuypers et al. 1965; Jones and Powell 1970; Van Essen and Maunsell 1983; Van Essen et al. 1992; Milner and Goodale 1995; Gerlach et al. 2000; Martin et al. 2000; Smith and Jonides 2000; Chen et al. 2008). The major white-matter fibers connecting VVN nodes include the inferior longitudinal fasciculus and external capsule (Chavis and Pandya 1976; Yeterian and Pandya 1995).

Diffusion tensor imaging (DTI) tractography studies, measuring the thickness, density and directionality of white-matter pathways connecting cortical hubs (Tuch et al. 2003; Gong et al. 2009; Loenneker et al. 2011) in combination with RS-fMRI showed that in long-range networks the more dense and thicker the connecting white-matter pathway, the stronger the structural and functional connectivity between the cortical nodes (Paus et al. 2001; Salat et al. 2009; Hagmann et al. 2010). Several DTI studies suggested that white-matter trajectories relevant to DVN may attain volumetric maturity later than VVN (Klingberg 2006; Mabbott et al. 2006). Studies of perception of faces in newborns were originally interpreted as supporting the early readiness of VVN, but are now in dispute (Johnson and deeHaan 2015). In contrast, regional MRI cortical morphometry studies have found that DVN cortical nodes mature earlier than ventral prefrontal and temporal regions (Sowell et al. 1999, 2001; Grill-Spector et al. 2008; Shaw et al. 2006, 2008). Further multidisciplinary insight about the rules governing maturation of both networks, their connectivity and governance of performance is important.

A primary basis for structural cortical–cortical brain connectivity emerges around the 8th week of gestation when dynamic proliferation and migration of neural cells is followed by emerging axonal processes, myelination, cell dendritic arborization and, consequently, by formation of the first neural circuits within sensory and motor roots (Yakovlev and Lecours 1967). The current general view is that major structural pathways and cortical hubs are predetermined (Rakic 2009). They emerge in early childhood, around age 2, similar to adults, but continue to strengthen till adulthood (Supekar et al. 2009). The nodes and efficiency of networks increases across "development" through refinement of a regional increase in thickness of myelination and diameter of axons (Löbel et al. 2009), with structural white-matter and functional connectivity development significantly correlated across ages (Hagmann et al. 2008). At the age of 5–6, the head circumference stabilizes and permits a reliable acquisition of MRI signals across all age groups (Caviness et al. 1996; Giedd et al. 1999; Sowell et al. 2004). Moreover, till about age 8 children increase in volume of CTh and from that time point the regional thinning of the cortex and increasing strength of between-nodes connectivity becomes a normative developmental trend (Giedd et al. 1999; Sowell et al. 2004; Shaw et al. 2008). The maturation of the brain

continues into later adolescence/adulthood with a trend of mostly regional cortical thinning (Sowell et al. 2004). Structural maturation studies showed a positive correlative interrelationship between structural cortical transformations and rising functional connectivity (Hyde et al. 2009; Seeley et al. 2009; Hagmann et al. 2010). Little corroboration, however, has been provided by neuropsychological data about the developmental brain–behavior relationship. We examined the age-dependent changes in CTh and structural connectivity within DVN and VVN in typically developing children (C), age ~6, in early adolescents (D) age ~11, and in adults (A) age ~21. Based on prior developmental, molecular, and neuroimaging data depicting formation of cortical–cortical circuits, we expected large group-age differences between youngest group of children and adolescents.

Maturational changes in gray matter density, volume and in CTh have been reported to differ between males and females. Greater volumes of gray matter in the frontal brain of adolescent males as compared with females were demonstrated in earlier studies (Reiss et al. 1996; Giedd et al. 1999). These are accompanied by more recent findings of delayed thinning of CTh and delayed neuronal coupling in frontal-polar cortical regions of boys as compared with girls, age 9–22 (Raznahan et al. 2010, 2011). The latter study relates CTh morphometry findings to sex differences in cognitive and behavioral profiles of adolescent children, offering a very attractive translational model. A recent (large *n*) study (Gennatas et al. 2017), however, shows a modest effect of sex on CTh, distributed across the cortical mantle with some higher CT in males over the area of the insula, frontal and occipital areas till adolescence (15 years). Then the pattern reverses, with females having a somewhat thicker cortex. These findings emphasize also that the reported lower gray matter volume in females coexist with increasing gray matter density, thus CTh morphometry measurements may benefit from histological validation. In another study on mean CTh in the frontal-polar brain of subjects age 8–20, sex did not show any significant differences (O'Donnell et al. 2005). The authors consider that these findings are limited because of a relatively small (18 males vs. 17 females) group sample. Consistently, a stereologic cortical morphometry study in males and females (age 12–24) found no sex differences in CTh (Rabinowicz et al. 2009). Thus, current findings on age-related sex differences in cortical anatomy are variable. The consistency may significantly depend on the number of available MRI scans and on the employed methodology of measurements. If we were to examine age-dependent sex differences, we would need a larger sample size. However, considering all of the above, in order to control the influence of sex, if any, we balance the distribution of males and females across our age groups.

The principal aim of this study is to examine which of two fundamental brain networks, DVN and VVN, displays a more advanced maturational state in early ontogeny, and thus assumes a governance in the development of cognitive proficiency. Towards this goal, three sets of age-dependent measurements are undertaken: (i) changes of CTh transformations using MRI signals in the specific DVN and VVN cortical nodes; (ii) changes in organization of the matrix of "intra-network" and "inter-network" connectivity based on the coherence of age-dependent CTh reduction; and (iii) proficiency of performance on neuropsychological tests associated with DVN and VVN in children as compared with adults. Our predictions that maturational thinning of the cortex is attained earlier in nodes of DVN rather than VVN is based on prior CTh morphometry reports (e.g., Gogtay et al. 2004; Sowell et al. 2004), on the high evolutionary significance of cognitive functions related to DVN and on our earlier fMRI and magnetoencephalography (MEG) studies with children (Ciesielski et al. 2006, 2010). We expect that the structural connectivity matrix in children inferred from correlations between cortical thinning in network nodes would show a stronger topology and edges for DVN, and would more closely resemble an adult pattern. This prediction is in line with converging evidence of protracted maturation in the prefrontal and temporal regions of VVN that govern verbal categorical labeling and facial cognitive interpretation based on detailed extraction of texture and color (review, Grill-Spector et al. 2008). Accordingly, we expect that performance in children on neuropsychological tests associated with DVN will mature earlier than on tests associated with VVN, and may show more similarity to adult performance. Thus, we predict that the pattern of CTh in DVN nodes will be similar in children and adults, the structural connectivity in children will be stronger in DVN than in VVN and children's performance proficiency will be closer to adults on visual-spatial-construction tests representing DVN. These predictions may all corroborate an earlier developmental course of DVN.

Materials and Methods

Participants

Thirty-six healthy volunteers participated in this study: 12 children (C: mean age 6.3 years, standard deviation (SD): 4 months), 12 early adolescents (D: 10.9 years; SD: 7 months), and 12 adults (A: 21 year 6 month, SD: 18 month). The recruitment process was rigorous: gender was distributed equally within and among the groups; prenatal, perinatal, and early postnatal complications were screened out; central nervous system medications and recreational substances were exclusionary. Pre-test screening interviews included milestones of development, adaptive, social-academic functioning, and personal and family history of neurological and psychiatric disorders. Consequently, two children and one adult were discontinued from participation in testing and from MRI scanning. All remaining participants were evaluated using a battery of neuropsychological tests to determine their typical cognitive status. Neuropsychological performance was found within an average to high-average range, as compared with age norms, qualifying subjects from all three groups for participation in the MRI component of the study. Each subject participated in a relaxation session prior to MRI scanning to reduce motor movement and increase comfort. The study's protocol was approved by the Institutional Review Board for Human Research, Massachusetts General Hospital. Signed informed consent and assent forms were obtained accordingly from each adult and child participant, and parent/legal caretaker.

Neuropsychological Data Acquisition and Analysis

The design of the current study included a standard neuropsychological battery of tests (Lezak et al. 2012). The foremost goal was to secure sample uniformity as a representation of typically developing, normative subjects. To attain this goal, the assessment raw data were related to standard age and gender corrected-norms (Strauss et al. 2006). The criterion was: subjects who performed −1.3 SDs below the age-corrected norm on three or more related measures would be excluded from the study as displaying an atypical cognitive profile. The second goal was to select, prior to FreeSurfer analysis, several

tasks targeting functions associated with DVN or VVN and to compare between groups the profiles of performance proficiency.

The rationale for selecting effective measures of functions targeted by neuropsychological tasks to maximally dichotomize functional anatomy of dorsal and ventral pathways was guided by findings of functional deficits in lesion studies on primates and humans, and by current neuroimaging findings. Brain lesion studies in humans were seminal for clinical neuropsychology (Teuber & Mishkin 1954; Luria 1961; Milner 1963) revealing an association between a location of a brain lesion and deficits in task performance. Current progress in neuroimaging demonstrates, however, that a complex visual task involves multiple levels of functional complexity and is associated with a network of synchronously connected cortical nodes rather then a single cortical region (e.g., Ardila et al. 2015). This rationale led us to utilize the relationship between certain main functional components of tests and visual processing networks.

The chief function of tasks linked with DVN, such as **CFT-Copy**, CFT-Immediate Recall (**CFT-IR**; Rey 1941; Osterrieth 1944), and Block Design (**BD;** a subtest of Wechsler Scales; Wechsler, 1997) include complex visual perception, organization and planning, top-down inhibitory control of visual interference, non-verbal working memory and perceptual-motor coordination. These functions are associated with network connectivity between the parietal/occipital, prefrontal dorsal, and premotor cortical nodes (Fuster 1989; Milner and Goodale 1995; Somerville et al. 2010).

In contrast, the neuropsychological tasks linked to VVN such as Stroop Word-Color Interference Test (**STROOP-WC**; Stroop 1935; Alvarez and Emory 2006), FAS-Controlled Oral Word Fluency Test (**FAS-VF**, Benton et al. 1994), and Wisconsin Card Sorting Test with perseverative errors measurements (**WCST-PE**; Berg 1948; Grant and Berg 1948; Heaton and Heaton 1981) all require cognitive proficiency in identification of an object's detailed properties (color, shape), integrating these properties into a verbally-driven rule of working-memory and using it with flexible alternation for controlling stimulus-response. The critical functional components of tests linked to VVN are the ability for self-control of motivation and verbally-driven rules of performance and to control of impulsive perseverative responses. All these functions are engaging connectivity between the prefrontal ventral (PFV)/orbital cortex and the inferior regions (with broad contributions from the limbic system). Thus, the control of impulsive perseverative errors in WCST-PE will engage the frontal orbital (FO)/ventral and inferior temporal-occipital cortical network. The perseverative errors, frequent in prefrontal lesions, are reported to be highly prevalent in lesions of the ventral/orbitofrontal system, while other cognitive and intellectual functions, including categorical thinking may remain preserved (Freedman and Oscar-Berman 1986).

The orbitofrontal section of the ventral prefrontal cortex has been closely associated with deficits in a delayed alternation behavior (Goldman-Rakic 1987; Fuster 1989), where perseverative responses are of the essence, and task functional demands resemble WCST and Stroop Task. These tasks require following a stimulus-response rule where the subject must inhibit, on each trial, the previously rewarded response and make a new decision. The ventral prefrontal region including the orbitofrontal cortex extending towards the frontal-polar and ventral part of the dorsolateral subdivision were reported as playing an important role in a healthy subject's performance on delayed alternation tasks (Mishkin and Pribram 1956; Warren and Akert 1964; Numan 1978; Rosenkilde 1979; Stuss and Benson 1986). Following the above rationale, we present tasks representing functions associated with DVN and VVN in Table 1.

To attain our second goal of neuropsychological testing, the assessment of the level of proficiency across age groups in functions associated with DVN and with VVN, Kruskal–Wallis tests were performed on raw scores from the neuropsychological tests representing DVN and VVN. Multiple comparisons, using a Bonferroni correction with a family-wise error rate of alpha = 0.05, were used to compare each pair of subjects from three age groups (Table 1).

MRI Data Acquisition

High-resolution structural MR scans were performed at the MGH/MIT/HMS Athinoula A. Martinos Center for Biomedical Imaging, Massachusetts General Hospital. Siemens Sonata, 1.5 Tesla Siemens AG, Erlangen, Germany. Imaging for the morphometric analysis was done with a 3D inversion recovery with a fast-low flip angle gradient echo sequence (MP-RAGE scans), providing 128 sagittal slices, 1.33 mm slice-thickness, with TR between inversion pulses 2730 ms; TR/TE/flip angle/TI: 2730 ms/3.44 ms/7 degrees/1000 ms; acquisition matrix of 256·192·128; square FOV of 256 mm; NEX 1; and two MP-RAGEs, each 8 min 46 s. These acquisition parameters were empirically optimized to increase gray/white and gray/cerebrospinal fluid contrast. Obtaining a single image with high contrast-to-noise, required for each participant two separate MP-RAGE acquisitions (8 min 46 s each).

MRI Data Analysis

Cortical reconstruction and volumetric segmentation was performed with the FreeSurfer image analysis program (http://surfer.nmr.mgh.harvard.edu/; Dale et al. 1999; Fischl, Sereno, Dale 1999; Fischl et al. 2002, 2004, Reuter et al. 2012). In summary, the 3D structural scans were used to construct models of each individual cortical surface. Cross-subject statistics were generated in a cortical surface-based coordinate system (Dale and Sereno, 1993; Fischl et al. 1999a).

In consecutive stages Freesurfer processing included: (i) motion correction and averaging (Reuter et al. 2012) of two high-resolution volumetric T1-weighted images; (ii) removal of non-brain tissue using a hybrid watershed/surface deformation procedure; (iii) automated Talairach transformation; (iv) intensity normalization; (v) tessellation of the gray matter/white-matter boundary, automated topology correction; and (vi) surface deformation following intensity gradients to optimally localize the gray/white and gray/pial matter, the segmentation of the subcortical white matter and deep gray matter volumetric structures (Dale et al. 1999; Fischl, Sereno, Dale 1999). When the cortical models were completed, deformable procedures were performed for further data processing and analysis including surface inflation (Fischl, Sereno, Dale 1999), registration to a spherical atlas which is based on individual cortical folding patterns to match cortical geometry across subjects (Fischl et al. 1999a), parcellation of the cerebral cortex into units with respect to gyral and sulcal structure (Desikan et al. 2006), and creation of maps of curvature and sulcal depth. Both intensity and continuity information from the entire 3D-MR volume was used in segmentation and deformation procedures to produce representations of CTh.

CTh was calculated as the closest distance from the gray/white boundary to the gray/CSF boundary. The distance

between those surfaces was measured at each point across the cortical mantle. We compare mean CTh for selected nodes representative of DVN and VVN. We use the mean thickness over preselected regions of interest (ROI), similarly to Dickerson et al. (2008) to estimate the CTh in mm^2 in the following ROIs for DVN: inferior posterior parietal cortex, left and right (IPP-L, IPP-R), SF dorsal cortex left and right (SF-L, SF-R), precuneus/posterior medial cortex left and right (PCN-L, PCN-R) and for VVN: fusiform gyrus/inferior temporal-occipital cortex left and right (FG-L, FG-R), ventral prefrontal cortex left and right (VPF-L, VPF-R), frontal orbital cortex extending to medial left and right (FO-L and FO-R). Using the spherical morph from each subject that transforms that subject's cortical surface model to the average cortical surface template, these ROIs were mapped from the template of each individual subject and the mean cortical CTh within each ROI in each subject was measured. This generated, for the MRI data set for each subject, a mean ROI CTh measure. The ROI analysis avoids the problem of having to correct for large numbers of statistical tests, the so-called multiple comparison problem. The Freesurfer procedures for the measurement of CTh have been validated using manual measurements (Kuperberg et al. 2003; Salat et al. 2004). Freesurfer morphometric procedures have been demonstrated to show good test–retest reliability across scanners and field strengths (Han et al. 2006; Reuter et al. 2012).

Before the FreeSurfer measurements were submitted to analysis two sets of evidence were examined, developmental and empirical validation, providing reassurance that the measurements of CTh are reliable and valid in our population of children age 6 and 11 (middle childhood). The developmental evidence provided confidence that the age-related gray/white tissue contrast will not negatively influence tissue segmentation in our youngest 6 year-old youngsters since age-related contrasts in gray/white matter after the age of three are no longer noticeable (Barkovich 2000). In addition, the dimensions of the brain, cortical architecture of sulci, gyri, and gray/white-matter contrast are reaching values comparable to adults around the age of 4–5 (Reiss et al. 1996; Barkovich 2000, 2005; Nolte 2008; Bray et al. 2015). Although the gray matter thickness was reported to be highest at the age of 4 (Pfefferbaum et al. 1994), after the age of 5 no significant age-changes in total cerebral volume were noted (Reiss et al. 1996). This is in sharp contrast to data reported from infants (age 0–2) where the MRI challenges are significant for brain morphometry including low gray/white-matter contrast to noise ratios, significantly smaller size of the cerebrum and high motion artifacts (Barkovich 2005; Prastawa et al. 2005; Shi et al. 2010).

The empirical validation of FreeSurfer is in line with the above developmental data. The validation study using FreeSurfer on a large sample of children age 4–11 demonstrated good validity of segmentation of gray matter and sub-cortical white matter in children around 5 years of age (Ghosh et al. 2010) (Important, Freesurfer includes motion correction of T1-weighted images and removal of non-brain tissue using a hybrid watershed/surface deformation procedure that improves the brain anatomical readability; Ségonne et al. 2004). Investigation of age bias in Talairach and spherical registration of brain coordinates between ages 4 and 11 found no bias of age down to 4 years 9 months (Ghosh et al. 2010). Since the youngest children were 6 years old, and thus far above the critical age of 5, the results of Talairach and other spherical registration techniques is expected to demonstrate comparable accuracy in spherical registration between adults and children. An automatic Talairach transformation was previously validated in pediatric and adult populations (Burgund et al. 2002) with a consistent outcome.

Analysis of Age-Group Differences between CTh in Nodes of DVN and VVN

The prime question was whether the thinning of the CTh in DVN and VVN showed a pattern determined by age. To compare CTh estimates across subjects in each age group, the cortical surface models were aligned using a high-resolution surface-based averaging technique that aligns cortical folding patterns. Each reconstructed brain was morphed to an average spherical surface representation that optimally aligned sulcal and gyral features across subjects, while minimizing metric distortion (Fischl et al. 1999a). To remove noise-induced variations in measurements, a surface-based Gaussian blurring kernel with a SD of 7 mm was applied. Mean CTh and variance of the mean were calculated at each location and mapped to the common space. Statistical CTh maps from selected brain regions were averaged across all A using high-resolution surface-based averaging techniques and compared with the CTh from C and D (Fischl et al. 2004). Statistically significant thickness difference maps were generated using *t*-tests for between samples, that is, at each vertex using a random effect model across both cortical hemispheres.

Figure 1 displays well-defined functionally and anatomically DVN and VVN cortical regions that were submitted to measurement. The morphometric cortical maps by Desikan et al. (2006) were used as a prototype for identifying the regions. For DVN, means of CTh and variance were calculated for the bilateral inferior parietal region (**IPP-L, IPP-R**), bilateral premotor cortex including "dorsal superior frontal" region (**SF-L, SF-R**) and "bilateral precuneus in medial plane" (**PCN-L, PCN-R**). For VVN representative nodes included: the "fusiform gyrus" (**FG-L, FG-R**) within the bilateral Inferior temporal/occipital region, the "prefrontal ventral cortex" (**PFV-L, PFV-R**), and the "bilateral frontal orbital cortex" (**FO-L, FO-R**). Tukey–Kramer comparison was used for post hoc calculations (after one-way ANOVA with alpha set at 0.05) to examine between-group differences in CTh (Group data in Supplementary Materials: Table S1C).

Analysis of Age-group Differences in Within-Network Connectivity

Is the matrix of connectivity between nodes of the network, as defined by coherent age-dependent changes in CTh, different in DVN and VVN? Two nodes within-network were considered to express structural connectivity when they displayed statistically significant correlations of CTh transformations in a particular age-group (He et al. 2008). The statistical correspondence between two nodes was determined by computing "The Pearson" correlation coefficient across all nodes of DVN and VVN. A symmetrical connectivity matrix was estimated for six cortical nodes in DVN and six cortical nodes in VVN for each subject within each age-group. The level of edge significance for the one-tailed test was set at $P = 0.05$. For a clear demonstration of the CTh-transformation-based connectivity "a graph representation" was developed for DVN and VVN using MATLAB (see the Results).

The connectivity results represented in Pearson's *r* correlation index (1CSM) are not corrected here by the traditional

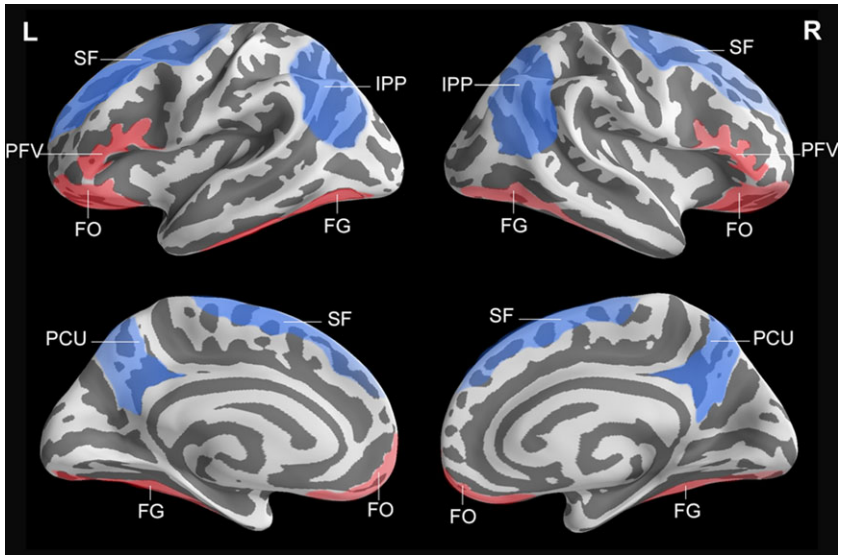

Figure 1. Cortical regions of interest (ROI) representing DVN and VVN in the lateral and medial plane of the brain. The cortical regions used for cortical thickness measurements for the dorsal visual network **(DVN): SF** (superior frontal cortex), **IPP** (inferior posterior parietal), **PCN** (precuneus/medial inferior parietal); ventral visual cortex **(VVN):** PFV (ventral prefrontal), **FG** (fusiform gyrus/inferior temporal), **OF** (orbital frontal/medial). Identification of ROIs based on Desikan et al. 2006.

Bonferroni correction. The significance levels based on the standard Bonferroni formula (0.05:180 = 0.0003) are not attainable in our small *n*. Thus, following Cohen (1992) we use the original values of ***r*** statistics (product–moment correlation coefficient *r*, df = *n*–2) as a representation of effect sizes (ES) for the found correlations (large = 0.50 and higher; medium = 0.30; small = 0.10, Cohen 1992). Large and medium ES predict that with a larger ***n*** sample the statistically significant *P* value corrected for multiple comparisons will be attainable with high probability. In the current study, we accepted for statistical analysis only those coefficients that displayed medium (0.30) and large (0.50) **ES**.

Graphical Representation of Within-Network and Between-Network Structural Connectivity

To display structural connectivity on the cortical mantle FsAverage cortical measurements from the Freesurfer algorithm were used (Fischl et al. 1999). FsAverage Cortical surface measures along with the cerebellum were plotted using Matlab custom code and "a graph representation" was developed for DVN and VVN, whereby the significant edges represented undirected connections between each single node. The gravity center of nodes was estimated by computing the Euclidean mean of the vertices in each ROI.

On the graph presented in the Result section below (Fig. 4), blue nodes represent the DVN, red represent the VVN. The connections between DVN nodes are shown in blue color edges, connections between VVN nodes in red color edges, and connections between DVN and VVN in green edges. The size of a circle represents strength of the node, that is, sum of weights of connections to the node, also known as strength of a node in graph theory (Rubinov and Sporns 2010). The connection strength (edge) was represented as thickness of the connecting line between the nodes: the thinnest line represents correlation significance at $P < 0.05$, uncorrected, and the thickest line represents $P < 0.001$, both are supported by large ES ($r_p = 0.50$ or higher; J. Cohen 1992).

Results

Age Effects on Neuropsychological Performance on Tasks Associated with DVN and VVN

Table 1 compares individual raw scores from C, D, and A's performance on tasks associated with DVN and VVN. The statistics revealed a lower speed and performance accuracy in C and D as compared with A on all tasks associated with VVN. Thus, C and D, as compared with A, showed significantly more perseverative errors in WCST-PE, less correct responses in Stroop-WC Test and lower fluency of words in FAS-VF. C and D did not perform significantly differently on FAS-VF test. When applying task specific to DVN and relying on visual-spatial-motor perception and organization (e.g., BD subtest of Wechsler Scales), both **C** and **D** performed with a high and similar proficiency, and with a level of skill and speed comparable to adults. However, in visual-spatial memory tasks, such as CFT-Copy and CFT-IR, where the demand for top-down control of interference in the memory domain is high, C were least proficient than A, whereas D already performed with competence comparable to A. In conclusion, in children and adolescents the proficiency of performance on tasks associated with specific functions of DVN appears closer to the adult level of performance than on tasks associated with VVN.

Age Effects on Difference Maps of Cortical Thickness

The primary question raised was whether the cortex in dorsal and ventral visual streams mature at different rates. Following previous evidence on age-dependent cortical thinning in later childhood and adolescence (Sowell et al. 2004) and considering a variable interrelationship between functional and structural brain development (Goldman-Rakic 1988; Ciesielski et al. 2006; Seeley et al. 2009), we expected that in our pediatric populations, C and D, the network that matures early may display a CTh pattern not significantly different from adults. Figure 2 displays statistically significant CTh-difference maps for age-group contrasts: Figure 2*A* is contrasting CTh in young adults (A)–children (C), and Figure 2*B* is contrasting CTh in young

Table 1 Age-dependent performance skills on neuropsychological tests related to DVN and VVN

	Kruskal–Wallis		Multiple comparisons		
Tests	χ^2	P	C vs. D	D vs. A	C vs. A
DVN					
CFT-Copy	18.77	0.001	n.s.	n.s.	*
CFT-IR	14.49	0.001	n.s.	n.s.	*
BD	11.35	0.003	n.s.	n.s.	n.s.
VVN					
WCST-PE	16.71	0.001	*	*	*
FAS-VF	21.56	0.001	n.s.	*	*
STROOP-WC	18.81	0.001	n.s.	*	*

Multiple comparisons, using a Bonferroni correction with family-wise error rate of alpha = 0.05, were used to compare each pair of subjects from all age groups. *Statistically significant group contrasts. **DVN** –> Dorsal Visual Network; **VVN** –> Ventral Visual Network; **C** –> children (6 years old), **D** –> Adolescents (~10–11 years old), **A** –> Adults (~21 years old); a Neuropsychological test associated with **DVN:** B–D (Block Design Subtest from Wechsler Intelligence Scales), and with **VVN:** FAS-VF (FAS-Benton Controlled Verbal Fluency Test).

adults (A)–adolescents (D). There are no significant age-group differences in nodes of DVN with one exception a significantly thicker cortex in C in the left dorsal SF cortex extending to the supplementary motor area. In contrast, for VVN most of the cortical nodes show a significantly thicker cortex in C than in A and in VPF for D than A, while contrast in CTh in C versus D (not included here) was not significantly reflected in the maps.

Group-differences in CTh were calculated using post hoc Tukey–Kramer All Pair Test (after one-way ANOVA, alpha set at 0.05; Fig. 3). The values for DVN showed no statistically significant differences between C and D or between C and A (P between 0.1 and 0.9). There is one exception in DVN pattern, when CTh in the left SF node is statistically significantly thicker in C as compared with A (C: 2.71; A: 2.58 mm, $P = 0.04$). The measurements for CTh in nodes of VVN (Supplementary Table S1C) display significant differences for C versus A (ranging between $P = 0.05$ and $P = 0.00001$), and for D versus A in bilateral VPF, FG, FO; ($P = 0.05$–0.0001). FO in the left hemisphere is the only VVN region in which CTh did not reach statistical significance for D versus A. The differences in CTh in C versus D are not significantly different for any of the nodes in VVN, corroborating the view about the protracted maturation of these regions. Visual examination of the within age-group distribution of mean values for CTh in female and male brains did not show any trend of sex-related differences. The female and male measurements did not significantly differ within groups and in relation to regions of the cortex.

Structural Connectivity: a Correlation of Age-Dependent Cortical Thinning Between-Network Nodes

We asked whether DVN and VVN, defined in prior DTI and DSI studies on white-matter connectivity, will also demonstrate high correlation in time-courses of maturational cortical thinning, and therefore display structural intra-network connectivity. We define "structural network connectivity" as coherent "cortical thickness transformations between two nodes of a functional network". Since developmental neuroimaging studies showed that function and structure are interconnected, in that an increased similar functional experience engages trophic processes in the network which may promote a specific anatomical plasticity (Lerch et al. 2006; He et al. 2017), we further ask if there is an age-dependent difference in the maturation of structural connectivity in DVN and VVN. Since DVN is innately involved with early childhood sensory-motor development, and has been suggested to be more fundamental to cognitive development in health and developmental psychopathology (Braddick et al. 2003; Atkinson and Braddick 2011) one may hypothesize that DVN will display earlier a more mature pattern of cortical connectivity as compared with VVN.

A "graph representation" of within-network and between-network structural connectivity is displayed in Figure 4. A matrix of Pearson correlation coefficients of CTh transformations between nodes of each network presents the within-network structural connectivity for DVN (in blue), VVN (in red), and for between networks DVN and VVN (in green). The

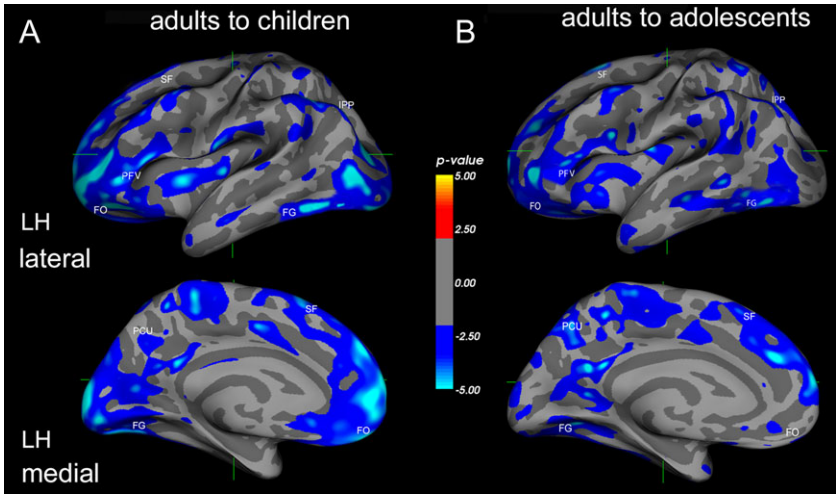

Figure 2. Statistical CTh-difference maps based on FreeSurfer analysis of MRI. (A) is contrasting CTh in young adults (A) with children (C), and (B) is contrasting CTh in young adults (A) with adolescents (D). The maps are overlaid on the lateral and medial surface of the left-brain hemisphere with average folding patterns of sulci (dark gray) and gyri (light gray) derived by using the surface-based morphing procedure (Dale et al. 1999; Fischl et al. 1999b). The color scale presents a range of statistically significant changes in – log10(p) · sign(c): we accepted for interpretation the contrast represented by light blue color that corresponds to high statistical significance (corrected) of thicker cortex in C: 2 and 5 on the scale correspond, respectively, to $P = 0.01$ and $P = 0.00001$; (note the significantly thicker cortex in C for VVN in FG, PFV, and FO nodes; for DVN in the left SF).

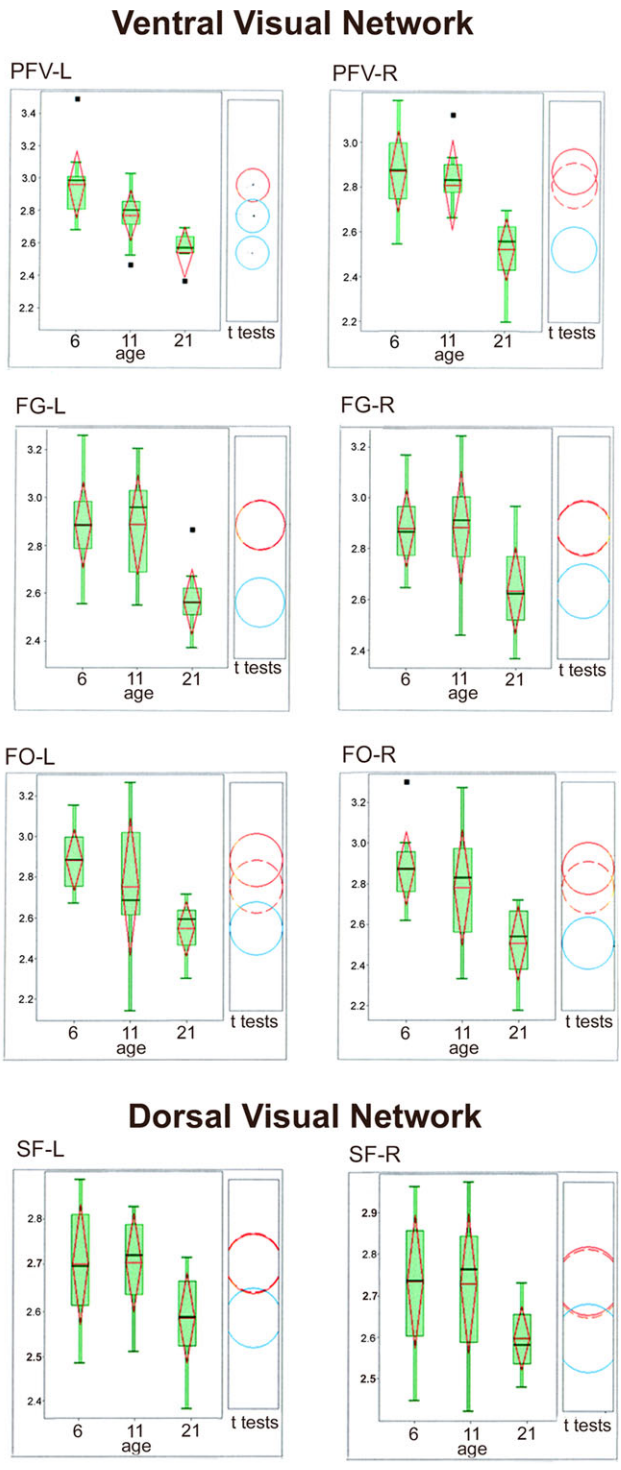

Figure 3. Age-dependent contrasts in CTh using box-plots and Tukey–Kramer all pair test. The box-plots demonstrate typical statistical parameters as quartiles, interquartile distance, median, and the real existing values smaller or bigger than 1.5 times the interquartile distance (in green); the mean and the corresponding standard deviation (red); the outliers (black). The Tukey–Kramer comparison (after one-way ANOVA, $P = 0.05$) is visualized using circles in red for the non-significant and circles in blue for the significant values. The center of each circle is aligned to the mean in the corresponding box-plots. The radius of the circles reflects the group variance, the larger the circle the bigger the variance. Tukey–Kramer all pairs comparison results demonstrate statistically significant differences in VVN for A versus C; A versus D in FG-L, FG-R, FO-L, FO-R, VPF-R. The statistically significant differences in the area VPF-L relate to A versus C, A versus D, and D versus C. In DVN, the node in SF-L is the only region with significant differences in CTh for C versus A. No significant age-dependent cortical thinning was found in other nodes of DVN.

structural connectivity between nodes of DVN and VVN are represented in green edges. The size of a circle represents strength of the node, that is, sum of weights of connections to the node. The connection strength is represented in thickness of the connecting edges between the nodes. The thinnest edge represents correlation significance at $P < 0.05$, uncorrected, and thickest edge represents $P < 0.0001$. The edges are accompanied by large ES, 0.50.

The strongest Pearson correlation coefficients for developmental thinning of cortex were found between left–right homologous regions in both networks, ranging from $r = 0.60$, $P = 0.002$ to highest $r = 0.87$, $P = 0.0003$ (Table 2). In many cases, these values are considerably lower, particularly in D, as compared with correlations for homogenous within-network nodes. In C, green edges are mostly non-significant (correlations $P = 0.075$) but in A they are significant ($P = 0.008$).

Age-Group Effects on Structural Connectivity for DVN and VVN

The graph matrix of correlated CTh changes in nodes of the two examined networks displayed by Figure 4 strongly implied age-dependent differences in structural connectivity for DVN and VVN, raising a question about their statistical validity. The mean values of Pearson's correlation coefficients for the selected cortical edges of DVN and VVN for subjects in each age-group were averaged and submitted to one-way ANOVA. For DVN the effect of age-group was not significant [$F = 2.08$, df = 2, $p = 0.138$; means: r_p: C = 0.439(0.27), D = 0.615(0.19), A = 0.583 (0.25)]. The effect of age-group was significant for VVN ($F = 6.23$, df = 2, $P = 0.004$). Post hoc Tukey–Kramer All Pair Test (alpha set at 0.05) showed statistically significant increase in correlational properties of edges in D versus C (mean difference: 0.235; $P_{Tukey} = 0.003$), suggesting that a significant change in structural connectivity within VVN may be spurting between the age of 6 and 11. No other age-dependent effects reached statistical significance, although the connectivity changes in D versus A and A versus C showed a clear trend to age-group effects, that may lead to a significant effect in a larger sample (D vs. A: $P_{Tukey} = 0.157$). It is important to note, that the mean changes in correlations between the nodes of VVN in C versus VVN in A are not statistically significant. This needs to be considered in context of statistically significant increases of the VVN structural connectivity in D. The increase in D may be transient before the connectivity is reduced in the third decade of life in adults and functionally strategized.

The most prominent effect of age-dependent changes was apparent for the between-network, DVN versus VVN structural connectivity ($F = 12.80$, df = 2, $P = 0.001$). Post hoc Tukey–Kramer All Pair Test (alpha set at 0.05) showed statistically significant increase in density of between-network structural connectivity in C versus D (mean difference: 0.231; $P_{Tukey} = 0.001$), and in C versus A (mean difference: 0.152; $P_{Tukey} = 0.004$). Figure 5 is illustrating the above statistics with clear differences in mean values for structural connectivity within-network (DVN in blue, VVN in red) and between networks (DVN–VVN in green).

The age-dependent effects in structural connectivity between nodes of DVN (in blue) were statistically not significant. In VVN (in red), the increase in structural connectivity

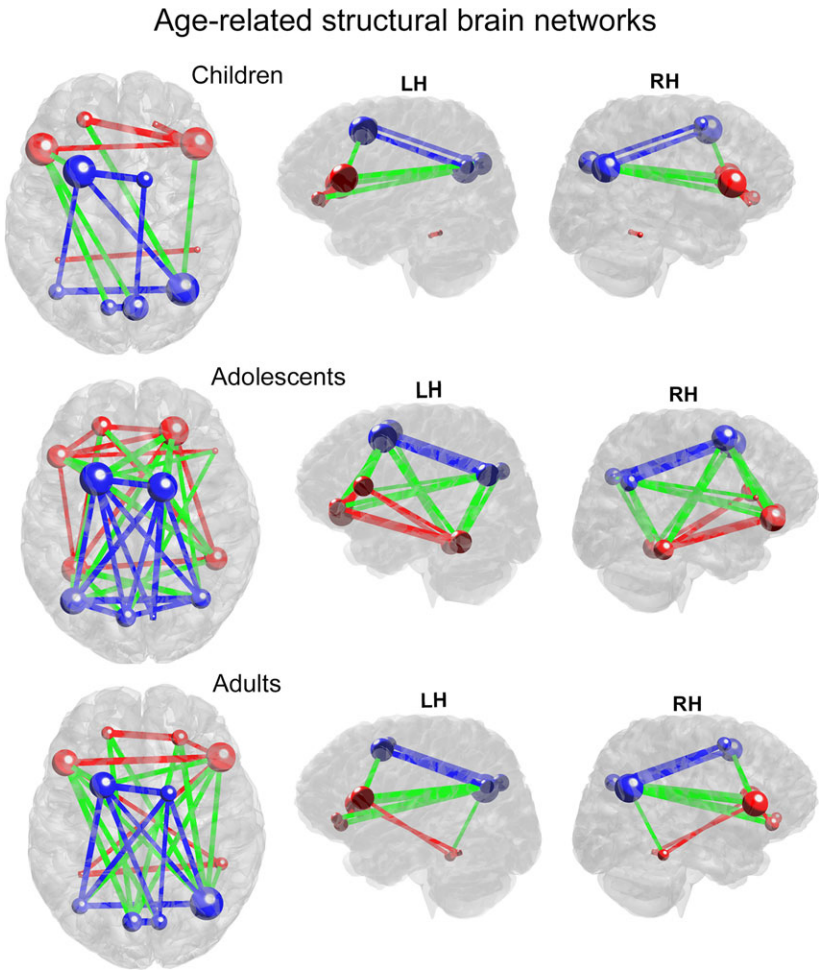

Figure 4. Age-dependent changes in structural connectivity within DVN (blue) and VVN (red) on anatomically informed diagram of network-specific cortical nodes. Between-network statistically significant edges are in green.

Table 2 Structural connectivity between homologous cortical nodes of DVN and VVN

Networks	DVN			VVN		
Group	IPP	SF	PCN	PFV	FG	FO
Children						
Pearson's *r*	0.863	0.764	0.766	0.565	0.567	0.471
P	0.0003	0.040	0.004	0.057	0.054	0.122
Adolescents						
Pearson's *r*	0.831	0.784	0.401	0.568	0.646	0.668
P	0.008	0.002	0.197	0.054	0.023	0.018
Adults						
Pearson's *r*	0.747	0.868	0.887	0.914	0.781	0.957
P	0.005	0.003	0.038	0.013	0. 003	0.001

P values in bold.

between C and D was statistically significant ($P = 0.004$). Although the age-related contrast in structural connectivity between C and D, and C and A did not reach the statistical significance (pt = 0.157 and 0.233, respectively) both were supported by strong ES. The between-networks structural

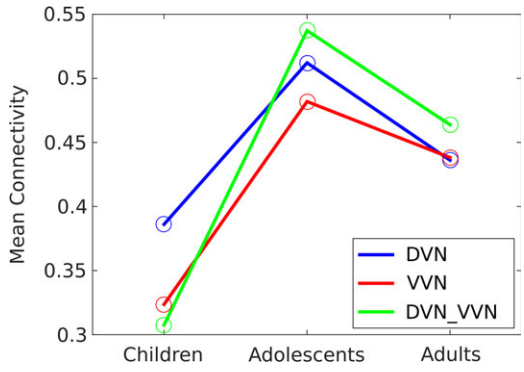

Figure 5. Mean within-network and between-betwork structural connectivity. The age-dependent effects in structural connectivity between nodes of DVN (in blue) were statistically not significant. In VVN (in red), the increase in structural connectivity between C and D was statistically significant ($P = 0.004$). Although the age-related contrast in structural connectivity between C and D, and C and A did not reach the statistical significance (pt = 0.157 and 0.233, respectively) both were supported by strong effect sizes. The between-networks structural connectivity (DVN–VVN in green) is an progressively firming property of the visual neuro-cognitive system reflected in significant statistical contrasts between C and D ($P = 0.001$), and C and A ($P = 0.004$).

connectivity (DVN–VVN in green) is an progressively firming property of the visual neuro-cognitive system reflected in significant statistical contrasts between C and D ($P = 0.001$), and C and A ($P = 0.004$).

Within-Network Homologous Structural Connectivity: DVN and VVN

An important characteristic of a network is its within-network structural modularity. The strong within-network structural connectivity would certify the network's cohesion, and the tightening of the CTh correlative connectivity within a network may become a possible marker of the network maturation, as discussed in earlier studies (Lerch et al. 2006; Alexander-Bloch et al. 2010). We will discuss the correlative values for CTh measurements within-network for homologous "lateralized regions", and relate them to correlative values representing other within-network and between-network connectivity. One may expect that the firm structural integrity of DVN be expressed in particularly high correlations within the functionally homologous nodes of DVN, as linking the structural with functional modularity (He et al. 2008).

To explore the within-network tightness for DVN and VVN, a set of Pearson correlations was calculated for the homologous (left–right) regions of the brain (Fig. 4), as particularly sensitive to reflecting the structural-functional network integrity. The numerical values for edges connecting homologous nodes within DVN and VVN is shown in Table 2. Values of Pearson correlation coefficient "r_p" are all around 0.60, consistent with and higher for long-range homologous nodes of DVN (SF-L, SF-R, with corresponding "P" values between 0.03 in children to 0.004 in adults, all with large ES). Both in A (SF-L to SF-R, $r = 0.82$, $P = 0.0003$; large ES) and in C the connectivity displayed between "homologs areas" was strong (SF-L vs. SF-R, $r_p = 0.764$; $P = 0.004$; IPP-L vs. IPP-R, $r_p = 0.863$, $P = 0.0003$; PCN-L vs. PCN-R, $r_p = 0.766$, $P = 0.003$). In D, the number of edges increased within DVN, demonstrating denser structural connectivity than in C. In D, SF-L is significantly correlated with SF-R ($r_p = 0.784$, $P = 0.002$), IPP-L with IPP-R ($r_p = 0.831$, $P = 0.008$), but the r_p for PCN-L to PCN-R did not reach the significance ($P = 0.197$). Following Cohen's principle (1992), all considered above Pearson correlations coefficients are supported by large ES. In VVN, the homologous nodes reached high correlative measures: in C "the structural connectivity", as demonstrated by statistically significant correlation of CTh transformations in homologous areas (Table 2), showed P values only approaching significance in DVN: FG-L versus FG-R ($r_p = 0.567$, $P = 0.054$); in PFV-L versus PFV-R ($r_p = 0.563$, $P = 0.057$); and for FO-L versus FO-R the edge remains non-significant ($r_p = 0.471$, $P = 0.122$).

DVN: In **C**, age-dependent correlative changes in nodes of long-range networks are strong between SF-L and IPP-R ($r_p = 0.578$, $P = 0.049$, LES), and between SF-L versus IPP-L ($r_p = 0.592$, $P = 0.042$), all supported by large ES (J. Cohen 1992). Connectivity between SF-R and PCN-R remains significant ($r_p = 0.580$, $P = 0.048$, large ES), but between SF-L and PCN-L ($P = 0.19$) and between SF-L and PCN-R ($P = 0.087$) is not. In **D**, the number of edges increased within DVN, showing denser structural connectivity than in C. In A, the number of significant edges is pruned down as compared with D (e.g., SF-R does not significantly connect to PCN-L, $P = 0.113$ and approaching only significance for SF-R and PCN-R, $P = 0.059$), and the number of within-network correlations is reduced. However, the connectivity between nodes that are significantly correlated is stronger than in C and D (e.g., SF-L to SF-R, $r = 0.82$, $P = 0.0003$; SF-R to IPP-L and SF-L to IPP-R, $r = 0.82$. $P = 0.007$, and $r = 0.670$, $P = 0.004$). In summary, in early childhood the structural connectivity within DVN, as assessed by correlations of maturational cortical thinning, shows well-formed and strong connections between homologous nodes, but weaker long-range connections along the SF–IPP and PFV-FG axis. In contrast, the abundant edges in D, are reduced in power. Those that remain, are markedly increasing in power, such as connectivity between SF-R and IPP-L ($r = 0.82$, $P = 0.007$, large ES), and SF-L and IPP-R ($r = 0.670$, $P = 0.004$, with large ES).

VVN: In **C**, we found a strong edge between FO-L and PFV-R ($r_p = 0.765$, $P = 0.004$) and FO-R with PFV-R ($r_p = 0.704$, $P = 0.011$). CTh transformations in PFV-L versus FG-R ($P = 0.318$) and PFV-R versus FG-L, ($P = 0.250$) were not significantly correlated, suggesting an ongoing maturation. The pattern of connectivity in the adolescent group (D) deriving from the maturational CTh changes was considerably different from that in **C.** An abundance of connections between nodes of the network, but with relatively less strong edges was found in D (e.g., PFV-L with FG-L, $r = 0.56$, $P = 0.057$). Furthermore, some of the edges displayed in D, disappear in A (e.g., FO-L vs. FG-L and FO-R vs. FG-R) and do not display statistically significant connectivity. In contrast, the edges which become significant for prefrontal ventral cortex, PFV-L with FG-L ($r_p = 0.539$, $P = 0.045$) and PFV-R versus FG-L ($r_p = 572$, $P = 0.05$) become significant. Summarizing, in C the pattern of VVN connectivity is bare. Some of the long-range connections are missing. There is no evidence of structural connectivity between bilateral PFV and the fusiform gyrus cortex (FG-L or FG-R). An unexpected, strong short-range connectivity is displayed between nodes of the frontal orbital cortex (FO-L and FO-R) and prefrontal ventral (PFV-R) area. In contrast, early adolescence is marked with unrefined abundant connectivity and multiple edges between nodes of VVN. In A, the pattern of structural connectivity in long-range networks becomes more selective with reduced number of edges, but increased strength of connectivity (PFV-L to FG-L; PFV-L to FG-R).

Discussion

The present study raises a three-factorial question about cortical maturation within DVN and VVN: whether the pattern of age-dependent transformations in CTh, the within-network structural connectivity and the proficiency in network-related cognitive functions vary between DVN and VVN, and if yes, which of the two networks matures first. Three significant findings are reported: (i) age-dependent differences in the pattern of MRI CTh point to an earlier maturational course of DVN; (ii) age-dependent differences in the pattern of within-network connectivity show stronger edges (in particular homologous) within DVN than within VVN, with a chaotic abundance of connections in adolescents across both networks; (iii) high and similar to adults cognitive proficiency in children and adolescents on visual-spatial perceptual and working memory tasks associated with the frontal-parietal dorsal visual processing network, but significantly lower proficiency on visual semantic categorization tasks targeting control of impulsive responses associated with the ventral visual network.

Age-Group Differences in MRI CTh Favor Early Maturation of DVN

The non-linear developmental pattern of CTh thinning in DVN and VVN displayed here (see Fig. 3A), is consistent with earlier cortical morphometry findings presenting a sequence of thinning extending from the primary visual cortex to associative prefrontal regions (Caviness et al. 1996; Huttenlocher and Dabholkar 1997; Giedd et al. 1999, 2009; McAlonan et al. 2005;

Brickman et al. 2006; Shaw et al. 2008; Westlye et al. 2010). Specifically, the ventral prefrontal and ventral temporal cortex display significantly protracted transformations between childhood and young adulthood, in line with prolonged maturation in other ventrally located networks that play a critical role in cognition, such as the frontal-insular network (Uddin et al. 2011). The cortical nodes of VVN display globally an intense process of thinning and increased connectivity between the age of 6 and 11, from sparse edges in children through dynamic non-selective structural "over-connectivity" in adolescents, and later stabilization in adults. Endocrine data suggest that dynamic hormonal changes in adolescence may contribute to over-production of synapses, with enhanced left hemisphere coupling in males (Fair et al. 2007, 2009; Kolb 2009; Supekar et al. 2009).

The exact processes that underlie developmental cortical thinning are not clear, and one possible hypothesis is that the transformations in CTh and the consequential functional diversity may be sculptured by variable processes of structural fine-tuning, such as developmental apoptosis, synaptic pruning, proportional reduction of gray tissue that is paralleled with an increase in myelination and neurotransmitter/hormonal concentration (Goldman-Rakic et al. 2000; Barbas 2015). This leads to a reduction of density in neuronal cellular bodies which foster better communication between individual neurocells, and effectively between networks. In accordance with the principle that "ontogeny recapitulates phylogeny" the prefrontal and temporal cortex, are the latest to mature in our data consistent with reports from other laboratories (Diamond 2002; Bunge and Wright 2007).

Recent studies report that dendritic spine density in childhood exceeds values in adults by two- to three times, and the dynamic process of elimination begins in late childhood, and yet the elimination of synapses continues through the third decade of life when the cortico-cortical circuitry reaches maturity (Petanjek et al. 2011). This functional plasticity reflecting reorganization of neural circuitry, including synaptic elimination, is consistent with numerous EEG and fMRI reports suggesting its essential role for acquisition of higher cognitive functions such as mental flexibility, working memory and affective control (Casey et al. 2008; Feinberg and Campbell 2010; Webster et al. 2011). Thus, the prefrontal cortex related to higher cognitive functions undergoes protracted remodeling during adolescence at both functional and anatomical levels that are concomitant with increases in cortical-sub-cortical connectivity and functional integrity. Among the processes contributing to development of the prefrontal cortex excitatory/inhibitory control, the GABAergic system is of main interest as it undergoes extensive changes during adolescence at the level of protein expression and modulation by neurotransmitters (Caballero and Tseng 2016). Gamma aminobutyric acid (GABA) has been shown to be one of the earliest neurotransmitters present in the developing brain. GABA can depolarize cortical progenitor cells and, thus, may provide the main excitatory drive for the immature cortical network and play a central role in regulating cortical development (Letinic et al. 2002). GABA interneurons regulate many steps of neurogenesis in the brain including neuronal proliferation, migration, differentiation, formation of early neural networks and the experience-dependent tuning of new circuits. Thus, the GABAergic system may be one of the major contributors to the developmental thinning of the cortex that we report here.

Our current findings are generally in line with the maturational rules that have been reported for white-matter pathways, however, although a strong functional correlation has been reported between regions that are densely structurally interconnected, such as the cortical dorsal fronto-parietal network (Wright et al. 1999; Lerch et al. 2006), studies on structural changes in white matter suggest that it is the sharing functional engagement that dictates structural maturational coupling and may drive structural covariance. Loenneker et al. (2011) reported on differential courses of white-matter pathways for DVN and VVN. They found that corpus callosum fiber bundles feeding to ventral networks increased in volume by a factor of 2–3 between childhood (~6 years) and young adulthood (~27 years), while the factor for white matter changes in the dorsal network increased only 1.5–2 times. Thus, age-dependent increases in fractional anisotropy and decreases in radial diffusivity were found in both DVN and VVN, but the authors interpret changes in DVN white-matter pathways as prolonged. However, although the volume of the VVN pathway appears to reach an adult-like volume of fibers at the age of 7, the authors point out that in children bundles of fibers are sent into lingual visual areas that may have prolonged pruning till adulthood in line with experience-related plasticity. Similarly, the fibers running into the fusiform and parahippocampal gyri are not yet established in children, which is consistent with our data showing late maturation of the cortex in nodes of VVN. A comprehensive review of studies on development of visual networks suggested an earlier maturation of the cortical volume in DVN in contrast to an earlier maturation of the white-matter pathways volume in VVN (Klaver et al. 2011).

Our findings are consistent with studies across-species employing diverse techniques (Distler et al. 1996). For example, the development of dorsal and ventral visual pathways was investigated using a local cerebral glucose utilization (LCGU) technique in rhesus monkeys (*Macaca mulata*) age 2–9 days, 1–6 month, and 3–4 years. Visual stimulation consisted of a high-contrast black–white geometrical pattern rotating 30° counterclockwise around the animal (Bachevalier et al. 1991). Optical densities related to regions of the autoradiographs were measured with a photoscan P-100 densitometer on a computer-based image processing system. Measures of autoradiographs were obtained at 1 mm intervals. The comparison of LCGU results in dorsal and ventral visual pathways suggested that in rhesus monkeys the dorsal visual processing stream develops earlier than ventral.

Age-Group Differences in Within-Network Connectivity: Strong Edges for DVN Homologous Nodes

Current CTh findings favor a view of earlier cortical maturation of the DVN module that is consistent with multimodality studies pointing to earlier functional and structural development of DVN (Kovács et al. 1999; Atkinson 2000; Kovács 2000; Atkinson and Braddick 2003; Johnson and Munakata 2005; Ciesielski et al. 2006; Alexander-Bloch et al. 2010). One might expect, therefore, that DVN will also reveal high inter-regional correlations between the homologous left and right hemisphere nodes. Such a pattern will be consistent with a direct link between the structural and functional individual modularity of the network, as suggested by prior studies (He et al. 2008). The present study finds significant homologous edges for both networks with statistically stronger edges for DVN than VVN. Thus, an initial formation of homologous connectivity across the corpus callosum may be a general principle of the brain networks formation. In our data, the lead in functional development of this connectivity is assumed by DVN. This is consistent with

developmental studies on cortical morphometry and neurobehavior (O'Donnell et al. 2005; Chen et al. 2008) and functional neuroimaging (Adleman et al. 2002).

Complementary to structural maturational changes in white-matter connectivity are fMRI reports of positive correlations between increasing RS functional connectivity (Greicius et al. 2003; Sporns, Tononi and Kötter 2005) and long-range connectivity maturing later (Lebel et al. 2008). RS-fMRI studies in older children report a similar general pattern of nodes and connectivity as in adults. However, cortical nodes associated with higher-order cognitive networks such as DVN and VVN, may not be integrated yet into a cohesive system (Damoiseaux et al. 2006; Damoiseaux and Greicius 2009). Our current CTh data is consistent with the latter as the thinning in the SF node is, in contrast to the posterior parietal, prolonged till adolescence in DVN, although generally DVN develops earlier than VVN.

Lerch et al. (2006) reported that the thickness of the cortex in areas subserving related functional specialization increases in structural similarity to each other with age. The present study shows that the frontal-parietal connectivity of DVN is lower in children than in adults, and yet children are quite proficient in tasks demanding visual-spatial skills. A possible explanation is that the broad sub-cortical contribution of the cerebellum and caudate nuclei through the parietal and premotor cortices (Yeterian and Pandya 1995; Clower et al. 2005) may compensate for the immature frontal-parietal connectivity and thus, secure performance on complex tasks. The functional and structural networks variability becomes more evident in pathophysiological studies (Atkinson 2000; Ciesielski et al. 2006, 2004) and by deductive mathematics (Friston and Price 2011).

The quantitative changes in the age-dependent matrix of edge density that we observed in the current study are consistent with the report by Khundrakpam et al. (2012). These authors show an increase in the number of connector hubs from low in early childhood (age 4.8–8.4 years), to an extensive distribution of hubs in late childhood/early adolescence (age 8.5–14.7), and again reduction of hubs in late adolescence (till 18 years of age). Data from our study and from other laboratories also show qualitative similarities in distribution of connectivity nodes, marked by an age-dependent shift from short-range FO (anterior cingulate in the Khundrakpam et al. study) to long-range connectivity between associative areas of the prefrontal ventral to posterior-inferior parietal cortex and precuneus. The unexpected finding of adolescent "over-connectivity" in both DVN and VVN awaits a large study investigation. Our data showing high-density chaotic connectivity in adolescents and poorly developed frontal-parietal axis in children are consistent with low efficiency of the top-down inhibitory control system, often discussed in adolescent literature as responsible for erratic behavior and the search for sensations (Davidson et al. 2006; Shaw et al. 2008; Cohen et al. 2010; Raznahan et al. 2010; Van Leijenhorst et al. 2010).

Compelling supportive evidence for earlier maturation of DVN came from our recent MEG studies on developmental connectivity among nodes of DVN and VVN as reflected in RS-MEG alpha oscillatory synchronization (Ciesielski et al. 2014). We acquired resting state functional connectivity (fcMEG) measures from 12 healthy male participants age 6–12 and 12 adults age 19–28 during a 6 min fixation on a hair-lined cross. The phase lag index (PLI) was then calculated (Stam and Reijneveld 2007). PLI is a reliable estimate of phase alpha synchronization, as a measure of connectivity between ROIs of DVN and VVN. PLI is also a measure of asymmetry of distribution of phase differences between two time-coupled oscillating ROIs. 16 ROIs specific to DVN and 16 ROIs specific to VVN were parceled and transformed into an MNI atlas for group analysis (Van Dijk et al. 2010). Increased PLIs of fcMEG alpha synchronization indicate a significantly stronger functional connectivity between coupled oscillators in the DVN as compared with VVN, in children. The PLI values for DVN present a similar pattern of connectivity in children and adults. In VVN the PLIs are significantly lower in children than in adults suggesting an incomplete development of network connectivity. Summarizing, our findings on maturational cortical thinning and correlation of these cortical changes suggest an earlier maturational course for DVN. A question of major importance remains whether cortical DVN leadership is reflected in cognitive and behavioral proficiency.

Developmental Pattern of Cognitive Performance associated with DVN and VVN

Thinning of the cortex as a maturational process has been reported to parallel increases in functional diversity and proficiency. For instance, responsiveness develops earlier within the primary sensory cortex that displays cortical thinning first. The associative frontal regions of the cortex, linked to tasks with complex mental flexibility, remain in the active process of thinning until young adulthood (Sowell et al. 2001; Gogtay et al. 2004; Toga et al. 2006; Giedd et al. 2008). Our neuropsychological measures provide a corresponding illustration: children are slower and less accurate than adults on almost all tests, including visually mediated working memory such as ROCFT-Recall and Wisconsin Card Sorting Test-PE, with two exceptions – they performed on a comparable level during copying of the complex ROCFT figure and on Blocks Design. Adolescents, however, who are closer to adults in thickness of the cortical mantle, demonstrate more comparable abilities to adults in performance on tests challenging visual-spatial cognitive abilities and working memory, such as the Wisconsin Card Sorting Test-PE and ROCFT- Recall. Furthermore, in Verbal Fluency and Stroop Word-Color Interference tests, relying on the late-maturing ventral prefrontal and temporal regions (PFV, FG) both children and adolescents continue to be significantly underperforming relative to adults.

The relationship between functional brain connectivity and cognitive proficiency was examined in prior studies using RS and task-related fMRI connectivity paradigms. The findings suggested higher cognitive proficiency with stronger functional connectivity, as reported for the dorsal frontal-parietal network and visual-spatial functions (Gilbert and Wu, 2013). More recent studies tracking RS functional brain connectivity with source-based MEG neuronal oscillations showed a positive covariance between increased integrity of the frontal-parietal network and children's proficiency in visual-spatial working memory tasks (Barnes et al. 2016). It is also the case in our study, where a statistically significantly stronger structural connectivity in DVN as compared with VVN in children is associated with better performance on tasks of visual-spatial perception and memory, functions specific to DVN. Our current CTh data are consistent with our earlier RS-MEG study (Ciesielski et al. 2014) and studies from other Laboratories (Boersma et al. 2011) showing stronger structural connectivity between nodes of DVN than VVN, with corresponding higher proficiency on visual-spatial tasks. One must, however, recognize that the DVN–VVN dichotomy could only be a matter of degree as both pathways may share certain neuroanatomic connectivity and neurotransmitter activation (Oscar-Berman et al. 1991; Zachariou et al. 2015). Illustrative here are

studies on recognition of faces, frequently considered to be a strong marker of inferior occipital/temporal activation within VVN. Recent fMRI and TMS studies on processing of faces clearly demonstrate activation of the inferior temporal/occipital gyrus of VVN, but also activation of parietal/occipital and the right dorsal frontal cortex, components of the visual-spatial DVN (Zhen et al. 2013; Zachariou et al. 2016).

As presented above, the developmental CTh properties of the DVN module reflect tight structural cohesiveness within each age group, although the bilateral prefrontal (SF) cortex in children has not yet reached maturation. One consequence of this prefrontal immaturity is a delay in formation of inter-regional connectivity along the long-range prefrontal-parietal axis (see Fig. 4 and section below), crucial for attentional expertise (Corbetta and Shulman 2002; Bressler et al. 2008; Gregoriou et al. 2009). Our data suggest, therefore, that neither, DVN or VVN, have reached complete maturity in children or adolescents, and yet both pediatric groups demonstrate a considerable expertise in tasks governed by these networks in adults. We suggest that the involvement of other age-specific sub-cortical compensatory circuits, cerebellum and caudate nuclei that have a robust functional connectivity with the inferior parietal cortex, may need to be considered (Ciesielski et al. 2006). Findings from recent studies in children on increase of GABA concentration with age and reduction in concentration of glutamate, the excitatory neurotransmitter in the striatum (Ghisleni et al. 2015) is consistent with such a hypothesis. Alternatively, the concept of signatures of network maturation may need to be reconsidered.

The principles that determine interrelationship between changes in CTh and connectivity within the long-range networks, such as DVN and VVN, and behavior have been a focus of neuroimaging studies. There are still many unanswered questions. In her recent cortical structural model for brain connectivity Barbas (2015) suggests that systematic structural variation across cortical areas could be considered as a core organizing principle for variability of connections and functions. This model emphasizes differences in the timing of development of different cortical areas as a likely mechanism for the emergence of systematic cortical structural variation that leads to a broad diversity of multiple pathway connections and neural computations, to differential recruitment of areas for flexible behavior, to variable behavioral dysfunctions and psychiatric disorders (Herbert et al. 2004). Systematic differences in the number of cortical layers, in neuronal spine density and dendritic complexity across brain areas have been reported by molecular studies (Allman and McGuinness 1988; Dombrowski et al. 2001; Elston et al. 2009; Lebel et al. 2008; Kaas 2008, Collins et al. 2010). The central principle is that the changes in laminar structure are not random but systematic and determined by a specific developmental time principle, yet, early prenatal and perinatal influences may lead to highly predictable variance (Walhovd et al. 2016). Our results provide support for this model, suggesting that CTh and structural brain connectivity rely on the systematic structural variation of the cortical mantle that is both genetically-determined and experience-dependent (Rakic, et al. 1996; Rakic 2009).

Methodological Considerations

The size of our sample is relatively small, but since the age distribution within each age-group is narrow (~1 year), the number of participants from our study is comparable to large developmental studies where testing sizable samples of participants permits only a small size *n* representing a particular age window. The internal validity of our data is, therefore, high but limited to the narrow age window that we tested. The high validity of data was also secured by rigorous selection of participating typically developing children using clinical interviews and neuropsychological assessment of each participant by the same clinician. The high control of the participant's motion during acquisition of two high resolution sets of MRI images was achieved by training each child in a relaxation session prior to scanning. Moreover, since the youngest children-participants were age 6 and older, the age-related gray/white brain tissue contrasts did not influence brain tissue segmentation or registration of brain coordinates during the FreeSurfer measurements of CTh that deserve to be considered as valid and reliable. The converging findings, CTh morphometry, intra-network connectivity, and neuropsychological performance contribute to the validity of our conclusion on the earlier maturation of the DVN. Thus, small-sample studies, with high internal integrity, may provide good validity data. Saying this, the current findings need to be submitted to scrutiny of replication on larger **n** samples across broader age groups before the early development of DVN could be considered as a predictive developmental marker and guidance for preventive efforts.

Concluding Remarks

The study reports three sets of converging evidence supportive of the earlier maturation of DVN as compared with VVN: (i) MRI CTh showed earlier developmental thinning of the cortical mantle in nodes of DVN than VVN; (ii) Connectivity between nodes within DVN in children was not significantly different from that in adults (except for dorsal SF), with edges between the homologous nodes stronger in DVN than VVN; (iii) Cognitive performance on tests associated with DVN (visual-spatial perception and memory) was comparable across age groups, in contrast to significant age-group differences on tasks associated with VVN. An important characteristic of the present connectivity data in both networks is the inverted U pattern of age-dependent connectivity, reflecting a low number of edges in children, sharp rise in early adolescence and reduction of edges in adults. This observation urges more detailed studies on quantitative and qualitative changes in adolescent brain connectivity, that may elucidate the neurobiology of unique sensitivity, increased activation of cortical-striatal networks and frequency of impulsive and scantily controlled behavior in early adolescence (Somerville and Casey 2010). Thus, current MRI cortical morphometry findings suggest that DVN may be leading the trajectory of visual networks development in healthy children, although, the functional and structural development of DVN neither in children nor early adolescents achieved the level of maturity comparable to young adults. Indeed, young 21 years old adults may not represent the "maturity plateau" in visual networks development, since white-matter pathways, cortical connectivity and cognitive control may undergo age-dependent improvement till almost the age of 30 (Tamnes et al. 2010; Petanjek et al. 2011; Amlien et al. 2016). It was shown that during an emotionally challenging task the brain of 21-year-olds shows activity resembling adolescents more than adults (Cohen et al. 2010). The present data demonstrate a dynamic window of changes in functional DVN connectivity between childhood and adolescence, thus, providing support to the needs for early intervention and enrichment of translational programs at this critical time of brain plasticity.

Supplementary Material

Supplementary material is available at *Cerebral Cortex* online.

Funding

Supported in part by National Institute for Biomedical Imaging and Bioengineering (P41EB015896) and by Nancy Lurie Marks Family Foundation, Boston.

Notes

We gratefully acknowledge important contributions by Dr Galina Ivanova (Leipzig University, Germany), Dr Elisheva H. Levin, Dr Davood Toffigi (UNM), Dr David Salad, and Dr Douglas Greve (MGH) at different stages of this project. Sincere appreciation to our most helpful Reviewers. *Conflict of Interest*: None declared.

References

Adleman NE, Menon V, Blasey CM, White CD, Warsofsky IS, Glover GH, Reiss AL. 2002. A developmental fMRI study of the Stroop color-word task. Neuroimage. 16:61–75.

Alexander-Bloch A, Gogtay N, Meunier D, Birnm R, Clasen L, Lalonde F, Lenroot R, Giedd J, Bullmore ET. 2010. Disrupted modularity and local connectivity of brain functional networks in childhood-onset schizophrenia. Front Syst Neurosci. 4:147–167.

Alexander-Bloch A, Raznahan A, Bullmore E, Giedd J. 2013. The convergence of maturational change and structural covariance in human cortical networks. J Neurosci. 33(7): 2889–2899.

Allman J, McGuinness E 1988. Visual cortex in primates. See Steklis, Erwin 1988, pp. 279–326.

Alvarez JA, Emory E. 2006. Executive function and the frontal lobes: a meta-analytic review. Neuropsychol Rev. 16:17–42.

Amlien IK, Fjell MA, Tamnes CK, Grydeland H, Krogsrud SK, Chaplin TA, Rosa MGP, Walhovd KB. 2016. Organizing principles of human cortical development-thickness and area from 4–30 years: insights from comparative primate neuroanatomy. Cereb Cortex. 26(1):257–267.

Ardila A, Bernal B, Rosselli M. 2015. Lannguage and visual perception associations: meta-analytic connectivity modeling of broadmann area 37. Behav Neurol. 2015:565871.

Atkinson J. 2000. The developing visual brain. Oxford: Oxford University Press.

Atkinson J, Braddick O. 2003. Neurobiological models of normal and abnormal visual development. In: de Haan M, Johnson E, editors. The Cognitive Neuroscience of Development. Hove, East Sussex: Psychology Press. p. 43–71.

Atkinson J, Braddick O. 2011. From genes to brain development to phenotypic behavior: "dorsal-stream vulnerability" in relation to spatial cognition, attention, and planning of actions in Williams syndrome (WS) and other developmental disorders. Prog Brain Res. 189:261–283.

Bachevalier J, Hagger C, Mishkin M. 1991. Functional maturation of the occipitotemporal pathway in infant rhesus monkeys. In: Lassen NA, Ingvar DH, TRaichle ME, Friberg L, editors. Alfred Benzon Symposium 31. Brain Work and Mental Activity. Coopenhagen: Munksgaard. p. 231–240.

Bachevalier J, Mishkin M. 1986. Visual recognition impairment follows ventromedial but not dorsolateral prefrontal lesions in monkeys. Behav Brain Res. 20:249–261.

Barbas H. 2015. General cortical and special prefrontal connections: principles from structure to function. Annu Rev Neurosci. 38:269–289.

Barkovich AJ. 2000. Concepts of myelin and myelination in neuroradiology. Am J Neuroradiol. 21(6):1099–1109.

Barkovich AJ. 2005. Magnetic resonance techniques in the assessment of myelin and myelination. J Inherit Metab Dis. 28(3):311–343.

Barnes JJ, Nobre AC, Woolrich MW, Baker K, Astle DE. 2016. Training working memory in childhood enhances coupling between frontoparietal control network and task-related regions. J Neurosci. 36(34):9001–9011.

Bassett DS, Bullmore E. 2006. Small-world brain networks. Neuroscientist. 12:512–523.

Benton AL, Hamsher K de S, Sivan AB. 1994. Mutilingual aphasia examination. Psychological Assessment Manual. 3rd ed. PAR, Lutz, Florida.

Berg EA. 1948. A simple objective treatment for measuring flexibility in thinking. J Gen Psychologie. 39:15–22.

Blakemore SJ, Burnett S, Dahl RE. 2010. The role of puberty in the developing adolescent brain. Hum Brain Mapp. 31: 926–933.

Boersma M, Smit DJ, de Bie HM, Van Baa GC, Boomsma DI, de Geus EJ, Delemarre-van de Waal HA, Stam CJ. 2011. Network analysis of resting state EEG in the developing young brain: structure comes with maturation. Hum Brain Mapp. 32: 413–425.

Braddick O, Atkinson J, Wattam-Bell J. 2003. Normal and anomolous development of visual motion processing: motion coherence and "dorsal stream vulnerability". Neuropsychologia. 41: 1769–1784.

Bray S, Krongold M, Cooper C, Lebel C. 2015. Synergistic effects of age on patterns of white and gray matter volume across childhood and adolescence. eNeuro. 2(4):e0003–e0015.

Bressler SL, Tang W, Sylvester CM, Shulman GL, Corbetta M. 2008. Top-down control of human visual cortex by frontal and parietal cortex in anticipatory visual spatial attention. J Neurosci. 28(40):10056–10061.

Bressler S, Tognoli E. 2006. Operational principles of neurocognitive networks. Int J Psychophysiol. 2:139–148.

Brickman AM, Zimmerman ME, Paul RH, Grieve SM, Tate DF, Cohen RA, Williams LM, Clark CR, Gordon E. 2006. Regional white matter and neuropsychological functioning across the adult lifespan. Biol Psychiatry. 5:444–453.

Bullmore E, Sporns O. 2009. Complex brain networks: graph theoretical analysis of structural and functional systems. Nat Rev Neurosci. 3:186–198.

Bunge SA, Wright SB. 2007. Neurodevelopmental changes in working memory and cognitive control. Curr Opin Neurobiol. 17:243–250.

Burgund ED, Kang HC, Kelly JE, Buckner RL, Snyder AZ, Petersen SE, Schlaggar BL. 2002. The feasibility of a common stereotactic space for children and adults in fMRI studies of development. Neuroimage. 17(1):184–200.

Caballero A, Tseng KY. 2016. GABAergic function as a limiting factor for prefrontal maturation during adolescence. Trends Neurosci. 39(7):441–448.

Casey BJ, Galvan A, Hare TA. 2005. Changes in cerebral functional organization during cognitive development. Curr. Opin Neurobiol. 15:239–244.

Casey BJ, Jones RM, Hare TA. 2008. The adolescent brain. Ann N Y Acad Sci. 1124:111–126.

Caviness VS Jr, Kennedy DN, Richelme C, Rademacher J, Filipek PA. 1996. The human brain age 7–11 years: a volumetric

analysis based on magnetic resonance images. Cereb Cortex. 5:726–736.

Changeux JP, Danchin A. 1976. Selective stabilization of developing synapses as a mechanism for the specification of neuronal networks. Nature. 264:705–712.

Chavis DA, Pandya DN. 1976. Further observations on corticofrontal connections in the rhesus monkey. Brain Res. 3: 369–386.

Chen ZJ, He Y, Rosa-Neto P, Germann J, Evans AC. 2008. Revealing modular architecture of human brain structural networks by using cortical thickness from MRI. Cereb Cortex. 18:2374–2381.

Chen J, Snow JC, Culham JC, Goodale MA. 2017. What role does "Elongation" Play in "Tool-Specific" activation and connectivity in the dorsal and ventral visual streams? Cereb Cortex. 28(4):1117–1131.

Ciesielski KT, Ahlfors SP, Bedrick EJ, Kerwin AA, Hämäläinen MS. 2010. Top-down control of MEG alpha-band activity in children performing categorical N-Back task. Neuropsychologia. 48:3573–3579.

Ciesielski KT, Harris RJ, Cofer LF. 2004. Posterior brain ERP patterns related to the Go/No-Go task in children. Psychophysiology. 41: 882–892.

Ciesielski KT, Lesnik PG, Savoy RL, Grant EP, Ahlfors SP. 2006. Developmental neural networks in children performing a categorical N-Back task. Neuroimage. 33:980–990.

Ciesielski KR, Rosen BR, Kenet T, VanDijk KRA, Khan S, Stufflebeam S, Hamalainen MS Developmental connectivity among nodes of dorsal and ventral visual networks as displayed in resting state MEG alpha oscillatory synchronization. Fourth Biennial International Conference on Brain Connectivity, Boston, September 2014.

Clower DM, Dum RP, Strick PL. 2005. Basal ganglia and cerebellar inputs to "AIP". Cereb Cortex. 15:913–920.

Cohen J. 1992. A power primer. Quantitative methods in psychology. Psychol Bull. 112(1):155–159.

Cohen JR, Asarnow RF, Sabb FW, Bilder RM, Bookheimer SY, Knowlton BJ, Poldrack RA. 2010. A unique adolescent response to reward prediction errors. Nat Neurosci. 13:669–671.

Collins CE, Airey DC, Young NA, Leitch DB, Kaas JH. 2010. Neuron densities vary across and within cortical areas in primates. Proc Natl Acad Sci USA. 107:15927–15932.

Corbetta M, Shulman GL. 2002. Control of goal-directed and stimulus-driven attention in the brain. Nat Rev Neurosci. 3 (3):201–215.

Courtney SM, Petit L, Maisog JM, Ungerleider LG, Haxby JV. 1998. An area specialized for spatial working memory in human frontal cortex. Science. 279:1347–1351.

Culham JC, Kanwisher NG. 2001. Neuroimaging of human parietal cortex. Curr Opin Neurobiol. 11:157–163.

Dale AM, Sereno MI. 1993. Improved localization of cortical activity by combining EEG and MEG with MRI cortical surface reconstruction: a linear approach. J Cogn Neurosci. 5: 162–176.

Dale AM, Fischl B, Sereno MI. 1999. Cortical surface-based analysis I. Segmentation and surface reconstruction. Neuroimage. 9:179–194.

Damoiseaux JS, Greicius MD. 2009. Greater than the sum of its parts: a review of studies combining structural connectivity and resting-state functional connectivity. Brain Struct Funct. 213:525–533.

Damoiseaux JS, Rombouts SA, Barkhof F, Scheltens P, Stam CJ, Smith SM, Beckmann CF. 2006. Consistent resting-state networks across healthy subjects. Proc Natl Acad Sci USA. 103:13848–13853.

Davidson MC, Amso D, Anderson LC, Diamond A. 2006. Development of cognitive control and executive functions from 4 to 13 years: evidence from manipulations of memory, inhibition and task switching. Neuropsychologia. 44:2037–2078.

Desikan RS, Segonne F, Fischl B, Quinn BT, Dickerson BC, Blacker D, Buckner RL, Dale AM, Maguire RP, Hyman BT, et al. 2006. An automated labeling system for subdividing the human cerebral cortex on MRI scans into gyral based regions of interest. Neuroimage. 31:968–980.

Desimone R, Ungerleider LG. 1989. Neural mechanisms of visual processing in monkeys. Handbook of Neuropsychology. 2: 267–299.

Diamond A. 2002. Normal development of prefrontal cortex from birth to adulthood: cognitive functions, anatomy, and biochemistry. In: Stuss DT, Knight RT, editors. Principles of frontal Lobe Function. London UK: Oxford University Press. p. 466–503.

Dickerson BC, Fernstermacher DH, Salat DH, Wolk DA, Maguire RP, Desican R, Pacheco J, Quinn BT, Van der Kouwe A, Greve DN, et al. 2008. Detection of cortical thickness correlates of cognitive performance reliability across MRI scan sessions, scanners, and field strengths. NeuroImage. 39:10–18.

Distler C, Bachevalier J, Kennedy C, Mishkin M, Ungerleider LG. 1996. Functional development of the corticocortical pathway for motion analysis in the Macaque monkey: a ^{14}C-2-deoxyglucose study. Cereb Cortex. 6:184–195.

Dombrowski SM, Hilgetag CC, Barbas H. 2001. Quantitative architecture distinguishes prefrontal cortical systems in the rhesus monkey. Cereb Cortex. 11:975–988.

Elston GN, Oga T, Fujita I. 2009. Spinogenesis and pruning scales across functional hierarchies. J Neurosci. 29:3271–3275.

Fair DA, Cohen AL, Power JD, Dosenbach NU, Church JA, Miezin FM, Schlaggar BL, Petersen SE. 2009. Functional brain networks develop from a "local to distributed" organization. PLoS Comput Biol. 5(5):1000381.

Fair DA, Dosenbach NU, Church JA, Cohen AL, Brahmbhatt S, Miezin FM, Barch DM, Raichle ME, Petersen SE, Schlaggar BL. 2007. Development of distinct control networks through segregation and integration. Proc Natl Acad Sci USA. 104(33):13507–13512.

Feinberg I, Campbell IG. 2010. Sleep EEG changes during adolescence: an index of a fundamental brain reorganization. Brain Cogn. 72:56–65.

Fischl B, Salat DH, Busa E, Albert M, Dieterich M, Haselgrove C, van der Kouwe A, Killiany R, Kennedy D, Klaveness S, et al. 2002. Whole brain segmentation: automated labeling of neuroanatomical structures in the human brain. Neuron. 33: 341–355.

Fischl B, Sereno MI, Dale AM. 1999. Cortical surface-based analysis. II: Inflation, flattening, and a surface-based coordinate system. Neuroimage. 9:195–207.

Fischl B, Sereno MI, Tootell RB, Dale AM. 1999a. High-resolution intersubject averaging and a coordinate system for the cortical surface. Hum Brain Mapp. 8:272–284.

Fischl B, van der Kouwe A, Destrieux C, Halgren E, Segonne F, Salat DH, Busa E, Seidman LJ, Goldstein J, Kennedy D, et al. 2004. Automatically parcellating the human cerebral cortex. Cereb Cortex. 14:11–22.

Fox MD, Corbetta M, Snyder AZ, Vincent JL, Raichle ME. 2006. Spontaneous neuronal activity distinguishes human dorsal and ventral attention systems. Proc Natl Acad Sci USA. 103: 10046–10051.

Freedman M, Oscar-Berman M. 1986. Bilateral frontal lobe disease and selective delayed-response deficits in humans. Behav Neurosci. 100:337–342.

Friedman HR, Goldman-Rakic PS. 1994. Coactivation of prefrontal cortex and inferior parietal cortex in working memory tasks revealed by 2DG functional mapping in the rhesus monkey. J Neurosci. 14:2775–2788.

Friston KJ, Price CJ. 2011. Modules and brain mapping. Cogn Neuropsychol. 33:1–10.

Fuster JM. 1989. The Prefrontal Cortex. New York: Raven Press.

Gennatas ED, Avants BB, Wolf DH, Sattertwaite TD, Ruparel K, Ciric R, Hakonarson H, Gur RE, Gur RC. 2017. Age-related effects and sex differences in gray matter density, volume, mass, and cortical thickness from childhood to young adulthood. J Neurosci. 37(20):5065–5073.

Gerlach C, Law I, Gade A, Paulson OB. 2000. Categorization and category effects in normal object recognition: a PET study. Neuropsychologia. 38:1693–1703.

Ghisleni C, Bollmann S, Poil S-S, Brandeis D, Martin E, Micjels L, O'Gorman R, Klaver P. 2015. Subcortical glutamate mediates the reduction of short-range functional connectivity with age in a developmental cohort. J Neurosci. 35(22):8433–8441.

Ghosh SS, Kakunoori S, Augustinack J, Nieto-Castanon A, Kovelman L, Gaab N, Christodoulou JA, Triantafyllou C, Gabrieli JD, Fischl B. 2010. Evaluating the validity of volume-based and surface-based brain image registration for developmental cognitive neuroscience studies in children 4 to 11 years of age. Neuroimage. 53(1):85–93.

Giedd JN, Blumenthal J, Jeffries NO, Castellanos FX, Liu H, Zijdenbos A, Paus T, Evans AC, Rapoport JL. 1999. Brain development during childhood and adolescence: a longitudinal MRI study. Nat Neurosci. 2:861–863.

Giedd JN, Lalonde FM, Celano MJ, White SL, Wallace GL, Lee NR, Lenroot RK. 2009. Anatomical brain magnetic resonance imaging of typically developing children and adolescents. J Am Acad Child Adolesc Psychiatry. 48:465–470.

Giedd JN, Lenroot RK, Shaw P, Lalonde F, Celano M, White S, Tossell J, Addington A, Gogtay N. 2008. Trajectories of anatomic brain development as a phenotype. Novartis Found Symp. 289:101–112. discussion 112-8, 193-5.

Gilbert CD, Wu Li. 2013. Top-down influences on visual processing. Nature Reviews Neurosci. 14:350–363.

Girvan M, Newman ME. 2002. Community structure in social and biological networks. Proc Natl Acad Sci USA. 99: 7821–7826.

Gogtay N, Giedd JN, Lusk L, Hayashi KM, Greenstein D, Vaituzis AC, Nugent TF, Herman DH, Clasen LS, Toga AW, et al. 2004. Dynamic mapping of human cortical development during childhood through early adulthood. Proc Natl Acad Sci USA. 101:8174–8179.

Gogtay N, Lu A, Leow AD, Klunder AD, Lee AD, Chavez A, Greenstein D, Giedd JN, Toga AW, Rapoport JL, et al. 2008. Three-dimensional brain growth abnormalities in childhood-onset schizophrenia visualized by using tensor-based morphometry. Proc Natl Acad Sci USA. 105(41):15979–15984.

Goldman-Rakic P. 1987. Circuitry of primate prefrontal cortex and regulation of behavior by representational memory. In: Plum F, editor. Handbook of Physiology: The Nervous System. Vol. 5. Bethesda: American Physiological Society. p. 373–417.

Goldman-Rakic PS. 1988. Topography of cognition: parallel distributed networks in primate association cortex. Annu Rev Neurosci. 11:137–156.

Goldman-Rakic PS, Muly EC 3rd, Williams GV. 2000. D(1) receptors in prefrontal cells and circuits. Brain Res Rev. 31:295–301.

Gong G, Rosa-Neto P, Carbonell F, Chen ZJ, He Y, Evans AC. 2009. Age- and gender-related differences in the cortical anatomical network. J Neurosci. 29:15684–15693.

Goodale MA, Milner AD. 1992. Separate visual pathways for perception and action. Trends Neurosci. 15:20–25.

Grant DA, Berg EA. 1948. A behavioral analysis of the degree of reinforcement and ease of shifting to new responses in a Weigl-type card sorting problem. J Exp Psychol. 38:404–411.

Gregoriou GG, Gotts SJ, Zhou H, Desimone R. 2009. High-frequency, long-range coupling between prefrontal and visual cortex during attention. Science. 324:1207–1210.

Greicius MD, Krasnow B, Boyett-Anderson JM, Eliez S, Schatzberg AF, Reiss AL, Menon V. 2003. Regional analysis of hippocampal activation during memory encoding and retrieval: fMRI study. Hippocampus. 13:164–174.

Grill-Spector K, Golarai G, Gabrieli J. 2008. Developmental neuroimaging of the human ventral visual cortex. Trends Cogn Sci. 12:152–162.

Hagmann P, Cammoun L, Gigandet X, Meuli R, Honey CJ, Wedeen VJ, Sporns O. 2008. Mapping the structural core of human cerebral cortex. PLoS Biol. 6:159–167.

Hagmann P, Sporns O, Madan N, Cammoun L, Pienaar R, Wedeen VJ, Meuli R, Thiran JP, Grant PE. 2010. White matter maturation reshapes structural connectivity in the late developing human brain. Proc Natl Acad Sci USA. 107: 19067–19072.

Han X, Jovicich J, Salat D, van der Kouwe A, Quinn B, Czanner S, Busa E, Pacheco J, Albert M, Killiany R, et al. 2006. Reliability of MRI-derived measurements of human cerebral cortical thickness: the effects of field strength, scanner upgrade and manufacturer. Neuroimage. 32(1):180–194.

Haxby JV, Grady CL, Horwitz B, Ungerleider LG, Mishkin M, Carson RE, Herscovitch P, Schapiro MB, Rapoport SI. 1991. Dissociation of object and spatial visual processing pathways in human extrastriate cortex. Proc Natl Acad Sci USA. 88:1621–1625.

He Y, Chen ZJ, Evans A. 2008. Small-world anatomical networks in the human brain revealed by cortical thickness from MRI. Cereb Cortex. 17:2407–2419.

He Z, Han D, Efimova O, Guijarro P, Yu Q, Oleksiak A, Jiang S, Anokhin K, Velichkovsky B, Grunewald S, et al. 2017. Comprehensive transcriptome analysis of neocortical layers in humans, and macaques. Nat Neurosci. 20(6):886–895.

Heaton SR, Heaton RK. 1981. Testing the impaired patient. In: Filskov SB, Ball TJ, editors. Handbook of Clinical Neuropsychology. New York: Wiley-Interscience. p. 526–544.

Herbert MR, Ziegler DA, Makris N, Filipek PA, Kemper TL, Normandin JJ, Sanders HA, Kennedy DN, Caviness VS Jr. 2004. Localization of white matter volume increase in autism and developmental language disorder. Ann Neurol. 55(4):530–540.

Huttenlocher PR. 1979. Synaptic density in human frontal cortex—developmental changes and effects of aging. Brain Res. 163:195–205.

Huttenlocher PR, Dabholkar AS. 1997. Regional differences in synaptogenesis in human cerebral cortex. J Comp Neurol. 387:167–178.

Hyde KL, Lerch J, Norton A, Forgeard M, Winner E, Evans AC, Schlaug G. 2009. Musical training shapes structural brain development. J Neurosci. 29:3019–3025.

Iliescu B, Dannemiller J. 2008. Brain-behavior relationships in early visual development. In: Nelson CA, Luciana M, editors. Handbook of Developmental Cognitive Neuroscience. Cambridge, MA: MIT Press. p. 127–146.

Johnson M, deeHaan M. 2015. Developmental Cognitive Neuroscience. 4th ed. Oxford, UK: Wiley-Blackwell..

Johnson MH, Mareschal D, Csibra G. 2008. The development and integration of the dorsal and ventral visual pathways in object processing. In: Nelson CA, Luciana M, editors. Handbook of Developmental Cognitive Neuroscience. 2nd ed. Developmental Cognitive Neuroscience. Cambridge, MA, US: MIT Press. p. 467–478.

Johnson MH, Munakata Y. 2005. Processes of change in brain and cognitive development. Trends Cogn Sci. 9:152–158.

Jones EG, Powell TP. 1970. An anatomical study of converging sensory pathways within the cerebral cortex of the monkey. Brain. 93:793–820.

Kaas JH. 2008. The evolution of the complex sensory and motor systems of the human brain. Brain Res Bull. 75:384–390.

Khundrakpam BS, Reid A, Brauer J, Carbonell F, Lewis J, Ameis S, Karama S, Lee J, Chen Z, Das S, et al. 2012. Developmental changes in organization of structural brain networks. Cereb Cortex. 23(9):2072–2085.

Klaver P, Marcar V, Ernst M. 2011. Neurodevelopment of the visual system in typically developing children. In: Braddick O, Atkinson J, Innocenti G, editors. Progress in Brain Research. Vol. 189. Amsterdam-St., Louis-New York: Elsevier B.V. p. 113–136.

Klingberg T. 2006. Development of a superior frontal-intraparietal network for visuo-spatial working memory. Neuropsychologia. 44:2171–2177.

Kolb B. 2009. Brain and behavioural plasticity in the developing brain: neuroscience and public policy. Paediatr Child Health. 14:651–652.

Kovács I. 2000. Human development of perceptual organization. Vision Res. 40:1301–1310.

Kovács I, Kozma P, Fehér A, Benedek G. 1999. Late maturation of visual spatial integration in humans. Proc Natl Acad Sci USA. 96:12204–12209.

Kuperberg GR, Broome MR, McGuire PK, David AS, Eddy M, Ozawa F, Goff D, West WC, Williams SC, van der Kouwe AJ, et al. 2003. Regionally localized thinning of the cerebral cortex in Schizophrenia. Arch Gen Psychiatry. 60:878–888.

Kuypers HG, Szwarcbart MK, Mishkin M, Rosvold HE. 1965. Occipitotemporal corticocortical connections in the rhesus monkey. Exp Neurol. 11:245–262.

Lebel C, Walker L, Leemans A, Phillips L, Beaulieu C. 2008. Microstructural maturation of the human brain from childhood to adulthood. Neuroimage. 40:1044–1055.

Lerch JP, Pruessner J, Zijdenbos AP, Collins DL, Teipel SJ, Hampel H, Evans AC. 2006. Automated cortical thickness measurements from MRI can accurately separate Alzheimer's patients from normal elderly controls. Neurobiol Aging. 29:23–30.

Letinic K, Zoncu R, Rakic P. 2002. Origin of GABAergic neurons in the human neicortex. Nature. 417(6889):645–649.

Lezak DM, Hovieson DB, Bigler ED, Tranel D. 2012. Neuropsychological Assessment.. 5th ed. New York: Oxford University Press.

Loenneker T, Klaver P, Bucher K, Lichtensteiger J, Imfeld A, Martin E. 2011. Microstructural development: organizational differences of the fiber architecture between children and adults in dorsal and ventral visual streams. Hum Brain Mapp. 32:935–946.

Luria AR. 1961. The role of Speech in Regulation of Normal and abnormal Behavior. London: The Pergamon Press.

Löbel U, Sedlacik J, Gullmar D, Kaiser WA, Reichenbach JR, Mentzel HJ. 2009. Diffusion tensor imaging: the normal evolution of ADC, RA, FA and eigenvalues studied in multiple anatomical regions of the brain. Neuroradiology. 51:253–263.

Mabbott DJ, Noseworthy M, Bouffet E, Laughlin S, Rockel C. 2006. White matter growth as a mechanism of cognitive development in children. Neuroimage. 33:936–946.

Martin A, Ungerleider LG, Haxby JV. 2000. Category specificity and the brain: the sensory motor model of semantic representations of objects. In: Gazzaniga MS, editor. The Cognitive neurosciences. 2nd. ed. Cambridge: MIT Press.

Maunsell JH, Van Essen DC. 1983. The connections of the middle temporal visual area (MT) and their relationship to a cortical hierarchy in the macaque monkey. J Neurosci. 3:2563–2586.

McAlonan GM, Cheung V, Cheung C, Suckling J, Lam GY, Tai KS, Yip L, Murphy DG, Chua SE. 2005. Mapping the brain in autism. A voxel-based MRI study of volumetric differences and intercorrelations in autism. Brain. 128:268–276.

Mesulam MM, Van Hoesen GW, Pandya DN, Geschwind N. 1977. Limbic and sensory connections of the inferior parietal lobule (area PG) in the rhesus monkey. Brain Res. 136:393–414.

Milner B. 1963. Effects of different brain lesions on card sorting: the role of the frontal lobes. Arch Neurol. 9:90–100.

Milner A, Goodale M 1995. The visual brain in action. Oxford Psychology Series, No. 27.

Milner AD, Goodale MA. 2008. Two visual systems re-viewed. Neuropsychologia. 46:774–785.

Mishkin M, Pribram KH. 1956. Analyses of the effects of frontal lesions in monkey: II Variations of delayed response. J Comp Physiol Psychol. 49:36–40.

Mishkin M, Ungerleider LG, Macko KA. 1983. Object vision and spatial vision: two cortical pathways. Trends Neurosci. 378: 408–411.

Nolte J. 2008. The Human Brain: An Introduction to its Functional Anatomy. 6th ed.. Mosby Elsevier.

Numan R. 1978. Cortical-limbic mechanisms and response control: a theoretical review. Physiol Psychol. 6:445–470.

Oscar-Berman M, McNamara P, Freedman M. 1991. Delayed-response tasks: parallels between experimental ablation studies and findings in patients with frontal lesions. In: Levin HS, Eisenberg HM, Benton AL, editors. Frontal Lobe Function and Dysfunction. NY, Oxford: Oxford University Press. p. 230–255. Chapter 12.

Osterrieth PA. 1944. Le test de copie d"une figure complexe. Arch de Psychologie. 30:206–356. (Corwin and Bylsma, 1993, Transl. Clinical Neuropsychologist).

O'Donnell S, Noseworthy MD, Levine B, Dennis M. 2005. Cortical thickness of the frontopolar area in typically developing children and adolescents. Neuroimage. 24:948–954.

Pandya DV, Seltzer B. 1982. Intrinsic connections and architectonics of posterior parietal cortex in the rhesus monkey. J Comp Neurol. 204:196–210.

Paus T, Collins DL, Evans AC, Leonard G, Pike B, Zijdenbos A. 2001. Maturation of white matter in the human brain: a review of magnetic resonance studies. Brain Res Bull. 54: 255–266.

Petanjek Z, Judas M, Simic G, Rasin MR, Uylings HBM, Rakic P, Kostovic I. 2011. Extraordinary neoteny of synaptic spines in human prefrontal cortex. Proc Natl Acad Sci USA. 108(32): 13281–13286.

Pfefferbaum A, Mathalon DH, Sullivan EV, Rawles JM, Zipursky RB, Lim KO. 1994. A quantitative magnetic resonance imaging study of changes in brain morphology from infancy to late adulthood. Arch Neurol. 51(9):874–887.

Prastawa M, Gilmore JH, Lin E, Gerig G. 2005. Automatic segmentation of MR images of the developing newborn brain. Med Image Anal. 9:457–466.

Rabinowicz T, Petetot JM, Khoury JC, De Courten-Myers GM. 2009. Neocortical maturation during adolescence change in neuronal soma dimension. Brain Cogn. 69:328–336.

Rakic P. 2009. Evolution of the neocortex: a perspective from developmental biology. Nat Rev Neurosci. 10:724–735.

Rakic P, Bourgeois JP, Eckenhoff MF, Zecevic N, Goldman-Rakic PS. 1986. Concurrent overproduction of synapses in diverse regions of the primate cerebraal cortex. Science. 232:232–235.

Rawley JB, Constantinidis C. 2009. Neural correlates of learning and working memory in the primate posterior parietal cortex. Neurobiol Learn Mem. 91(2):129–138. Special Issue: Parietal Cortex.

Raznahan A, Lee Y, Stidd R, Long R, Greenstein D, Clasen L, Addington A, Gogtay N, Rapoport JL, Giedd JN. 2010. Longitudinally mapping the influence of sex and androgen signaling on the dynamics of human cortical maturation in adolescence. Proc Natl Acad Sci USA. 107:16988–16993.

Raznahan A, Lerch JP, Lee N, Greenstein D, Wallace GL, Stockman M, Clasen L, Shaw PW, Giedd JN. 2011. Patterns of coordinated anatomical change in human cortical development: a longitudinal neuroimaging study of maturational coupling. Neuron. 72:873–884.

Reiss AL, Abrams MT, Singer HS, Ross JL, Dencla MB. 1996. Brain development, gender and IQ in children: a volumetric imaging study. Brain. 119:1763–1774.

Reuter M, Schmansky NJ, Rosas HD, Fischl B. 2012. Within-subject template estimation for unbiased longitudinal image analysis. Neuroimage. 61(4):1402–1418.

Rey A. 1941. L"examen psychologique ans les cas d"encephalopatie traumatique. Archives de Psychologie. 28:286–340. (Corwin and Bylsma, 1993 Transl.).

Rizzolatti G, Matelli M. 2003. Two different streams from the dorsal visual system: anatomy and functions. Exp Brain Res. 153:146–157.

Rosenkilde CE. 1979. Functional heterogeneity of the prefrontal cortex in the monkey. A review. Neural Biol. 25:301–345.

Rubinov M, Sporns O. 2010. Complex network measures of brain connectivity: uses and interpretations. Neuroimage. 52:1059–1069.

Salat DH, Buckner RL, Snyder AZ, Greve DN, Desikan RS, Busa E, Morris JC, Dale AM, Fischl B. 2004. Thinning of the cerebral cortex in aging. Cereb Cortex. 14:721–730.

Salat D, Lee S, van der Kouwe A, Greve DN, Fischl B, Rosas HD. 2009. Age-associated alterations in cortical gray white matter signal intensity and gray white matter contrast. Neuroimage. 48(1):21–28.

Seeley WW, Crawford RK, Zhou J, Miller BL, Greicius MD. 2009. Neurodegenerative diseases target large-scale brain networks. Neuron. 62:42–52.

Shaw P, Greenstein D, Lerch J, Clasen L, Lenroot R, Gogtay N, Evans A, Rapoport J, Giedd J. 2006. Intellectual ability and cortical development in children and adolescents. Nature. 440:676–679.

Shaw P, Kabani N, Lerch JP, Eckstrand K, Lenroot R, Gogtay N, Greenstein D, Liv Clasen L, Evans A, Rapoport JL, et al. 2008. Neurodevelopmental trajectories of the human cerebral cortex. J Neurosci. 28:3586–3594.

Shi F, Fan Y, Tang S, Gilmore JH, Lin W, Shen D. 2010. Neonatal brain image segmentation in longitudinal MRI studies. Neuroimage. 49:391–400.

Smith EE, Jonides J. 2000. The Cognitive Neuroscience of Categorization. The New Cognitive Neurosciences. Cambridge, MA: MIT Press. p. 1013–1022.

Somerville LH. 2016. Searching for signatures of brain maturity: what are we searching for? Neuron. 92(6):1164–1167.

Somerville LH, Casey BJ. 2010. Developmental neurobiology of cognitive control and motivational systems. Curr Opin Neurobiol. 20:236–241.

Somerville J, Tremont G, Stern RA. 2010. The Boston qualitative scorring system as a measure of executive functioning in Rey-Osterrieth complex figure performance. J Clin Exp Neuropsych. 22(5):613–621.

Sowell ER, Thompson PM, Holmes CJ, Jernigan TL, Toga AW. 1999. *In vivo* evidence for post-adolescent brain maturation in frontal and striatal regions. Nat Neurosci. 2:859–861.

Sowell ER, Thompson PM, Leonard CM, Welcome SE, Kan E, Toga AW. 2004. Longitudinal mapping of cortical thickness and brain growth in normal children. J Neurosci. 24:8223–8231.

Sowell ER, Thompson PM, Tessner KD, Toga AW. 2001. Mapping continued brain growth and gray matter density reduction in dorsal frontal cortex: inverse relationships during post-adolescent brain maturation. J Neurosci. 21: 8819–8829.

Sporns O, Chialvo DR, Kaiser M, Hilgetag CC. 2004. Organization, development and function of complex brain networks. Trends Cogn Sci. 8:418–425.

Sporns O, Tononi G, Kötter R. 2005. The human connectome: a structural description of the human brain. PLoS Comput Biol. 1(4):e42.

Stam CJ, Reijneveld JC. 2007. Graph theoretical analysis of complex networks in the brain. Nonlinear Biomed Phys. 1:3–12.

Strauss E, Sherman EM, Spreen O, Spreen O. 2006. A Compendium of Neuropsychological Tests: Administration, Norms, and Commentary. 3rd. ed. New York, NY. Oxford: Oxford University Press.

Stroop JR. 1935. Studies of interference in serial verbal reactions. J Exper Psychol. 18:643–662.

Stuss DT, Benson DF. 1986. The Frontal Lobes. New York: Raven Press.

Supekar K, Musen M, Menon V. 2009. Development of large-scale functional brain networks in children. PLoS Biol. 7:e1000157.

Ségonne F, Dale AM, Busa E, Glessner M, Salat D, Hahn HK, Fischl B. 2004. A hybrid approach to the skull stripping problem in MRI. Neuroimage. 22(3):1060–1075.

Tamnes CK, Ostby Y, Fjell AM, Westlye LT, Due-Tennessen P, Walhovd KB. 2010. Brain maturation in adolescence and young adulthood: regional age-related changes in cortical thickness and white matter volume and microstructure. Cereb Cortex. 20:534–548.

Teuber HL, Mishkin M. 1954. Judgement of visual and postural vertical after brain injury. J Psychol. 38:161–175.

Toga AW, Thompson PM, Sowell ER. 2006. Mapping brain maturation. Trends Neurosci. 29:148–159.

Tuch DS, Reese TG, Wiegel MR, Wedeen VJ. 2003. Diffusion MRI of complex neural architecture. Neuron. 40:885–895.

Uddin LQ, Supekar KS, Ryali S, Menon V. 2011. Dynamic reconfiguration of structural and functional connectivity across core neurocognitive brain networks with development. J Neurosci. 31(50):18578–18589.

Van Dijk KR, Hedden T, Venkataraman A, Evans KC, Lazar SW, Buckner RL. 2010. Intrinsic functional connectivity as a tool for human connectomics: theory, properties, and optimization. J Neurophysiol. 103:297–321.

Van Essen DC, Anderson CH, Felleman DJ. 1992. Information processing in the primate visual system: an integrated systems perspective. Science. 255:419–423.

Van Essen DC, Maunsell JHR. 1983. Hierarchical organization and functional streams in the visual cortex. Trends Neurosci. 6:370–375.

Van Leijenhorst L, Zanolie K, Van Meel CS, Westsenberg PM, Rombouts SA, Crone EA. 2010. What motivates the adolescents? Brain regions mediating reward sensitivity across adolescence. Cereb Cortex. 20:61–69.

Walhovd KB, Krogsrud SK, Amlien IK, Bartsch H, Bjørnerud A, Due-Tønnessen P, Grydeland H, Hagler DJ Jr, Håberg AK, Kremen WS, et al. 2016. Neurodevelopmental origins of lifespan changes in brain and cognition. Proc Natl Acad Sci USA. 113(33):9357–9362.

Warren JM, Akert K, editors. 1964. The Frontal Granular Cortex and Behavior. New York: McGraw-Hill.

Webster MJ, Elashoff M, Weickert CS. 2011. Molecular evidence that cortical synaptic growth predominates during the first decade of life in humans. Int J Dev Neurosci. 29: 225–236.

Wechsler D. 1997. Wechsler adult intelligence scale - III. San Antonio, TX: Psychological Corporation.

Westlye LT, Walhovd KB, Dale AM, Bjørnerud A, Due-Tønnessen P, Engvig A, Grydeland H, Tamnes CK, Østby Y, Fjell AM. 2010. Differentiating maturational and aging-related changes of the cerebral cortex by use of thickness and signal intensity. Neuroimage. 52:172–185.

Wright IC, Sharma T, Ellison ZR, McGuire PK, Friston KJ, Brammer MJ, Murray RM, Bullmore ET. 1999. Supra-regional brain systems and the neuropathology of schizophrenia. Cereb Cortex. 9:366–378.

Yakovlev OI, Lecours AR. 1967. The myelogenetic cycles of regional maturation of the brain. In: Minkowski A, editor. Regional Development of Brain in Early Life. Oxford: Blackwell Science Publications. p. 3–70.

Yeterian EH, Pandya DV. 1993. Striatal connections of the parietal association cortices in rhesus monkeys. J Comp Neurol. 332:175–197.

Yeterian EH, Pandya DV. 1995. Corticostriatal connections of extrastriate visual areas in rhesus monkeys. J Comp Neurol. 352:436–457.

Zachariou V, Nikas CV, Safiuliah ZN, Behrmann M, Klatzky R, Ungerleider LG. 2015. Common dorsal stream substrates for mapping of surface texture to object parts and visual spatial processing. J Cog Neurosci. 27(12):1–20.

Zachariou V, Nikas CV, Safiuliah ZN, Gotts SJ, Ungerleider LG. 2016. Spatial mechanisms within the dorsal visual pathway contribute to the configural processing of faces. Cereb Cortex. 27(8):4124–4138.

Zhen Z, Fang H, Liu J. 2013. The hierarchical brain network for face perception. PLoS One. 8(3):e59886.

Zhou J, Gennatas ED, Kramer JH, Miller BL, Seeley WW. 2012. Predicting regional neurodegeneration from the healthy brain functional connectome. Neuron. 73:1216–1227.

Zielinski BA, Gennatas ED, Zhou J, Seeley WW. 2010. Network-level structural covariance in the developing brain. Proc Natl Acad Sci USA. 107:18191–18196.