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Brain development and aging: Overlapping and unique patterns of change

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ABSTRACT

Early-life development is characterized by dramatic changes, impacting lifespan function more than changes in any other period. Developmental origins of neurocognitive late-life functions are acknowledged, but detailed longitudinal magnetic resonance imaging studies of brain maturation and direct comparisons with aging are lacking. To these aims, a novel method was used to measure longitudinal volume changes in development (n = 85, 8–22 years) and aging (n = 142, 60–91 years). Developmental reductions exceeded 1% annually in much of the cortex, more than double to that seen in aging, with a posterior-to-anterior gradient. Cortical reductions were greater than the subcortical during development, while the opposite held in aging. The pattern of lateral cortical changes was similar across development and aging, but the pronounced medial temporal reduction in aging was not precast in development. Converging patterns of change in adolescents and elderly, particularly in the medial prefrontal areas, suggest that late developed cortices are especially vulnerable to atrophy in aging. A key question in future research will be to disentangle the neurobiological underpinnings for the differences and the similarities between brain changes in development and aging.

Introduction

Despite increasing recognition of the importance of developmental processes for later neurocognitive functions (Deary et al., 2006; Kochunov et al., 2012), and neurodegenerative (Shaw et al., 2007) and neuropsychiatric (Gogtay et al., 2011; Paus et al., 2008) disorders, studies directly comparing brain changes in development and aging are lacking. Human brain development is notably protracted (Blakemore, 2012; Giedd and Rapoport, 2010; Jernigan et al., 2011), and much of the potential and many of the vulnerabilities of the brain depend on the first two decades of life (Toga et al., 2006). It is even suggested that common mechanisms may be implicated in brain maturation in childhood and degenerative changes in aging (Nikolaev et al., 2009; Wines-Samuelson and Shen, 2005), reflected in potentially more age-related atrophy in regions characterized by higher degree of plasticity during development (Mesulam, 2000; Toga et al., 2006). Still, testing of similarities and differences between patterns of change in development and healthy aging has not been undertaken. Thus, the purpose of the present study was to characterize developmental trajectories across childhood and adolescence in both the cerebral cortex and a range of subcortical structures, test how the pattern of maturation changes across development, and directly compare this to the pattern of atrophy in a sample of elderly.

Developmental magnetic resonance imaging (MRI) studies show that while the first years of life are characterized by gray matter (GM) increases (Gilmore et al., 2012; Knickmeyer et al., 2008), older children and adolescents show cortical GM decreases in most regions, increasing white matter (WM) volumes and heterogeneous changes in subcortical structures (Brain Development Cooperative Group, 2012; Brown et al., 2012; Giedd et al., 1996a, 1999; Lenroot et al., 2007; Muftuler et al., 2011; Østby et al., 2009; Sowell et al., 2004; Tamnes et al., 2010; Westlye et al., 2010b). Longitudinal studies with wide age-ranges are needed, however, as they allow modulation of differences in change-patterns across age, for instance enabling testing of the posterior–anterior theory of cortical maturation, which suggests that

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[1] Some of the data used in the preparation of this article were obtained from the Alzheimer's Disease Neuroimaging Initiative (ADNI) database (adni.loni.ucla.edu). As such, the investigators within the ADNI contributed to the design and implementation of ADNI and/or provided data, but did not participate in the analysis or writing of this report. A complete listing of ADNI investigators can be found at: adni.loni.ucla.edu.

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higher-order association cortices mature relatively late (Gogtay et al., 2004; Shaw et al., 2008). Moreover, simultaneous measurements of cortical and subcortical structures yield a unique possibility for characterizing the pattern of variation in developmental trajectories across anatomical areas (Østby et al., 2009). In a benchmark study, Shaw et al. (2008) found differing levels of complexity of developmental trajectories across the cortex and that this pattern aligned with the cortical types depicted in established cytoarchitectonic maps. Subcortical changes were not investigated, however. Furthermore, studies combining samples of children and elderly are necessary to test how developmental trajectories align with the pattern of atrophy in aging. Similar to development, a heterogeneous pattern of atrophy is seen in healthy aging, with the frontal and temporal regions showing the largest changes (Fjell and Walhovd, 2010; Lemaitre et al., 2012).

Here, we present analyses of longitudinal MRI data obtained from 85 children and adolescents (8–22 years), and compare developmental cortical and subcortical changes with atrophy in 142 healthy elderly participants (60–91 years) (Fjell et al., 2009a). The aims of the current study were to 1) characterize developmental changes in cortical and subcortical structures, 2) test how the pattern of maturation changes across development, and 3) directly contrast changes in development and aging. An accurate description of healthy brain development and detailed knowledge of individual differences in developmental trajectories are paramount to understanding the foundations of cognitive development, neurodevelopmental disorders, and later lifespan changes. We used a novel unbiased method to quantify volumetric change (Holland and Dale, 2011) that has been proven to be highly sensitive to even subtle changes over short time periods in elderly participants (Holland et al., 2012; Murphy et al., 2010), but which has never before been used in developing samples. Furthermore, this was combined with image segmentation and parcellation to obtain change estimates in a large number of cortical and subcortical regions.

Material and methods

Participants

The sample of children and adolescents was drawn from the longitudinal project *Neurocognitive Development* (Østby et al., 2009; Tamnes et al., 2010), University of Oslo. The study was approved by the Regional Ethical Committee of South Norway. Typically developing children and adolescents aged 8–19 years were recruited through newspaper ads and local schools. Written informed consent was obtained from all participants older than 12 years of age and from a parent for participants under 16 years of age. Oral informed consent was given by participants under 12 years of age. Parents and participants aged 16 years or older were at both time-points screened with separate standardized health interviews to ascertain eligibility. Participants were required to be right handed, fluent Norwegian speakers, have normal or corrected to normal vision and hearing, not have a history of injury or disease known to affect central nervous system (CNS) function, including neurological or psychiatric illness or serious head trauma, not be under psychiatric treatment, not use psychoactive drugs known to affect CNS functioning, not have had complicated or premature birth, and not have MRI contraindications. Additionally, all scans were evaluated by a neuroradiologist at both time-points and required to be deemed free of significant injuries or conditions.

At time-point 1 (tp1), 111 participants satisfied these criteria and had adequate processed and quality checked MRI data. Eighteen participants did not want to or were unable to participate at time-point 2 (tp2), two were not located, three had dental braces and three were excluded due to neurological or psychiatric conditions. Thus, at tp2, 85 participants (38 females) underwent a second MRI scan. The mean age at tp1 for this final sample was 13.7 years (SD = 3.4, range = 8.2–19.4) and the mean IQ, as assessed by the Wechsler Abbreviated Scale of Intelligence (WASI) (Wechsler, 1999), was 109.0 (SD = 11.4, range = 82–141). The mean age at tp2 was 16.3 years (SD = 3.4, range = 10.8–21.9) and the mean IQ was 112.5 (SD = 10.5, range = 87–136). The mean interval between the two scanning sessions was 2.6 years (SD = 0.2, range = 2.4–3.2). The length of the interval was not related to age ($r = -.03$, $p = .772$) and not different for girls and boys ($t = 0.42$, $p = .675$).

The sample of healthy elderly was originally obtained from the Alzheimer's Disease Neuroimaging Initiative (ADNI) database (adni.loni.ucla.edu), and has previously been reported by Fjell et al. (2009a), but has not previously been used as in the current study. The total number of participants entering ADNI as healthy controls at baseline, and who attended the one year follow-up was 204. ADNI eligibility criteria are described at www.adni-info.org. Briefly, participants were 55–91 years of age, had an informant able to provide an independent evaluation of functioning, spoke either English or Spanish and had Mini-Mental State Examination (MMSE) (Folstein et al., 1975) scores between 24 and 30 (inclusive) and a Clinical Dementia Rating (CDR) (Morris, 1993) of 0. Of these 204, only those with MR segmentation passing internal quality control for baseline, one year follow-up and processing of change were included, and we additionally excluded those that had worse CDR sum of boxes score at the time of follow-up. The final sample consisted of 142 participants (tp1 mean age = 75.6 years, range = 59.8–90.2 years, 68 females), which were followed for one year (tp2 mean age = 76.7 years, range = 60.8–91.3 years).

MRI acquisition

Imaging data on children and adolescents were collected using a 12 channel head coil on a 1.5 T Siemens Avanto scanner (Siemens Medical Solutions) at Rikshospitalet, Oslo University Hospital. The same scanner and sequences were used at both time-points. The pulse sequences used for morphometry analysis were two repeated 160 slice sagittal T1-weighted magnetization prepared rapid gradient echo (MPRAGE) sequences (time repetition (TR)/time echo (TE)/time to inversion (TI)/flip angle (FA) = 2400 ms/3.61 ms/1000 ms/8°) per participant per visit. To increase the signal-to-noise ratio (SNR) the two runs were averaged during pre-processing. The protocol also included a 176 slice sagittal 3D T2-weighted turbo spin-echo sequence (TR/TE = 3390/388 ms) and a 25 slice coronal FLAIR sequence (TR/TE = 7000–9000/109 ms) to aid the neuroradiological examination.

All ADNI scans used for comparison purposes in the present paper were from 1.5 T scanners. Data were collected across a variety of scanners with protocols and acquisition parameters standardized across platforms, as described in detail at adni.loni.ucla.edu/research/protocols/mri-protocols/mri-protocols-adni-1. A major effort has been devoted to evaluating and adjusting the sequences for morphometric analyses (Jack et al., 2008). For the sample included in the current study, raw DICOM MRI scans (including two T1-weighted volumes per case) were downloaded from the ADNI site. Consequently, while all the MRI data from the children and adolescents were acquired on the same scanner using the same sequence, different scanners were used across subjects in the healthy elderly sample. Although this constitutes a caveat and potential source of bias, previous studies have shown that brain morphometry can be reliably estimated across a number of image acquisition variables (Han et al., 2006; Jovicich et al., 2009), as well as consistent age-related differences in elderly participants across multiple samples (Fjell et al., 2009b; Walhovd et al., 2011).

MRI analysis

Image processing and analyses were performed at the Multimodal Imaging Laboratory, University of California, San Diego. The raw data were reviewed for quality, and automatically corrected for spatial distortion due to gradient nonlinearity (Jovicich et al., 2006) and B_1