Microstructure abnormalities in children with attention deficit hyperactivity disorder combined and inattentive subtypes reveals by diffusion tensor imaging

Xiaoxia Du[1], Du Lei[1], Jun Ma[2], Guohua Shen[1], and Gengying Li[1]

[1]*Shanghai Key Laboratory of Magnetic Resonance, Department of Physics, East China Normal University, Shanghai, China, People's Republic of,* [2]*Department of Developmental and Behavioral Pediatrics of Shanghai Children's Medical Center, XinHua Hospital affiliated to Shanghai Jiao Tong University School of Medicine, Shanghai Key Laborat, Shanghai, China, People's Republic of*

Introduction Inattention and impulsivity are the most prominent clinical features of attention deficit hyperactivity disorder (ADHD). Abnormalities of white matter have been found in ADHD patients by diffusion tensor imaging (DTI) study, but results were inconsistent [1-4]. It is still unclear whether different ADHD sub-types have similar microstructure abnormalities in the brain. The purpose of this study was to explore the differences of brain microstructure between children with ADHD predominantly inattentive (ADHD-I) and combined (ADHD-C) sub-types by DTI, which detects changes in microstructure based on properties of diffusion.

Materials and Methods In this work, we evaluated structural changes in the brains of children with ADHD using DTI. Three groups consisting of 30 ADHD-C, 44 ADHD-I and 43 healthy children were scanned. The diffusion parameters of fractional anisotropy (FA) and mean diffusivity (MD) were subjected to whole-brain, voxel-wise group comparisons using statistical parametric mapping (SPM). For all analyses, the statistical maps were thresholded at $p < 0.001$ (uncorrected). Moreover, an extent threshold of 10 contiguous voxels was applied to exclude small clusters that emerged by chance.

Results When compared to healthy subjects, FA values in ADHD-C patients were significantly reduced in temporal lobe/sub-gyral, frontal lobe/sub-gyral and fusiform gyrus (see figure 1B); ADHD-I group had lower FA than controls in sub-lobar/extra-nuclear and fusiform gyrus (see figure 1C). There were also some differences of brain areas between ADHD-C and ADHD-I: comparing to ADHD-I subjects, FA values in ADHD-C patients were significantly reduced in sub-lobar, inferior frontal gyrus, middle temporal gyrus and thalamus (see figure 1A).
When compared to healthy subjects, MD values in ADHD-C patients were significantly reduced in frontal lobe/precentral gyrus, superior frontal gyrus, medial frontal gyrus, parietal lobe/precuneus, inferior parietal lobule and inferior frontal gyrus (see figure 2B). And ADHD-I group had lower MD than controls in temporal lobe/hippocampus, limbic lobe/hippocampal gyrus and middle temporal gyrus (see figure 2C). There were many differences of brain areas between ADHD-C and ADHD-I, including parietal lobe, frontal lobe and occipital lobe (see figure 2A).

Discussion Although both ADHD-C and ADHD-I showed abnormality in FA and MD compared to the healthy controls, there were obvious differences of microstructure between ADHD-C and ADHD-I. Previous studies reported that the patients with ADHD showed FA difference in frontal lobe [1-4] occipital lobe [1], temporal lobe [3] and parietal lobe [1] compared to healthy subjects. The previous results were inconsistent [1-4], which may be due to the reason that ADHD patients were composed of different subtype and age in different researches.

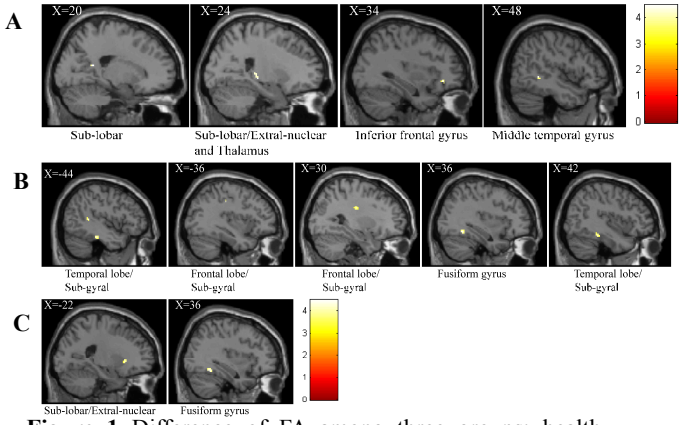

Figure 1 Difference of FA among three groups: healthy group, ADHD-C and ADHD-I.
A: ADHD-C compared to ADHD-I;
B: ADHD-C compared to healthy group;
C: ADHD-I compared to healthy group.

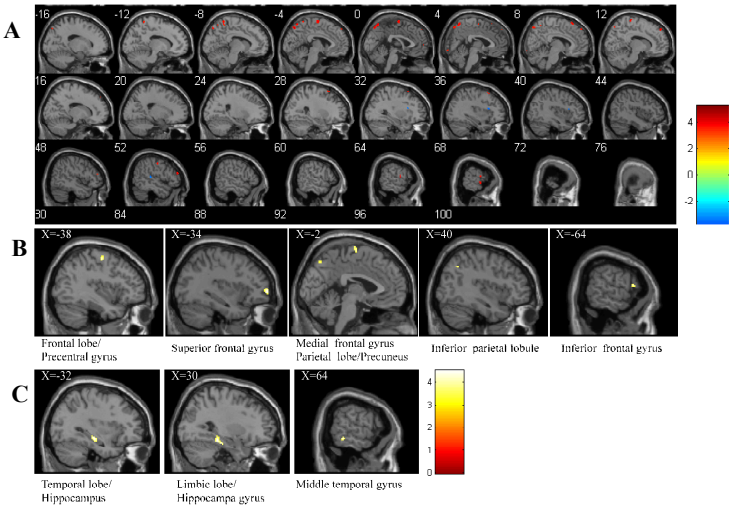

Figure 2 Difference of MD among three groups: healthy group, ADHD-C and ADHD-I.
A: ADHD-C compared to ADHD-I;
B: ADHD-C compared to healthy group;
C: ADHD-I compared to healthy group

References

1. Ashtari M, Kumra S, et al. (2005) Biol Psychiatry 57: 448-455.
2. Davenport ND, Karatekin C, et al. (2010) Psychiatry Res 181: 193-198.
3. Konrad A, Dielentheis TF, et al. (2010) Eur J Neurosci 31: 912-919.
4. Li Q, Sun J, et al. (2010) Neuro Endocrinol Lett 31: 747-753.