Poster | Disorders of Nervous Systems and Treatment

[2P]Neurodevelopmental Disorders

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*Videos are available throughout the meeting period.

[2P-229]In vivo imaging of dopamine D1 receptor and activated microglia in attention-deficit/hyperactivity disorder: A positron emission tomography study.

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Since the current medical treatment for attention-deficit/hyperactivity disorder (ADHD) is often accompanied by insufficient tolerability, novel therapeutic targets and mechanisms have been expected to be unraveled. As alterations in both the dopamine D1 receptor (D1R) and microglial activation have been implicated in the pathophysiology of ADHD, we investigated the contributions of the D1R and activated microglia and their interactions to the clinical symptoms and cognitive deficits in ADHD individuals using positron emission tomography (PET). Twenty-four psychotropic-naive ADHD individuals and 24 age-matched typically developing (TD) subjects underwent PET measurements with [11C]SCH23390 for the D1R and [11C](R)PK11195 for activated microglia as well as assessments of clinical symptoms and cognitive functions. The ADHD individuals showed decreased D1R in the anterior cingulate cortex (ACC) and increased activated microglia in the dorsolateral prefrontal cortex (DLPFC) and orbitofrontal cortex (OFC) compared with the TD subjects. The decreased D1R in the ACC was associated with severe hyperactivity in the participants with ADHD. Microglial activation in the DLPFC were associated with deficits in processing speed and attentional ability, and that in the OFC was correlated with lower processing speed in the ADHD individuals. Furthermore, positive correlations between the D1R and activated microglia in both the DLPFC and the OFC were found to be significantly specific to the ADHD group and not to the TD group. The current findings suggest that microglial activation and the D1R reduction as well as their aberrant interactions underpin the neurophysiological mechanism of ADHD and indicate these biomolecular changes as a novel therapeutic target.