EV0711

The relationship between responsiveness to social and monetary rewards and ADHD symptoms

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Introduction Alterations in reward processing are frequently reported in ADHD. One important factor that affects reward processing is the quality of reward, as social and monetary, rewards are processed by different neural networks. However, effect of reward type on reward processing in ADHD was not extensively studied.

Aims We aimed to explore the effect of reward type (i.e., social or monetary) on different phases of reward processing and also to test the hypothesis that ADHD symptoms may be associated with a problem in processing of social rewards.

Methods We recorded event-related potentials (ERPs) during a spatial attention paradigm in which cues heralded availability and type of the upcoming reward and feedbacks informed about the reward earned. Thirty-nine (19 males and 20 females) healthy individuals (age range: 19–27) participated in the study. ADHD symptoms were measured using ADHD self-report scale (ASRS).

Results The feedback related potentials, namely feedback related negativity (FRN), P200 and P300 amplitudes, were larger for social rewards compared to monetary rewards (Fig. 1). There was a consistent negative correlation between the hyperactivity subscale of ASRS and almost all feedback related ERPs. ERP amplitudes after social rewards were smaller for individuals with more hyperactivity.

Conclusions Our findings suggest that hypo responsiveness to social rewards may be associated with hyperactivity. However, the results have to be confirmed with clinical populations.

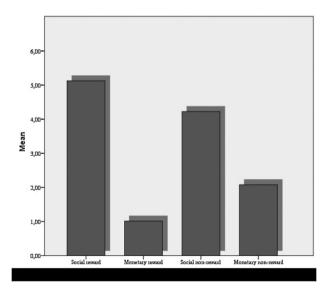


Fig. 1 FRN amplitudes.

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The differential effect of event rate on pupil dilation patterns suggests effort dysregulation problems in ADHD

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Introduction The state regulation model postulates that ADHD performance difficulties result from failures to regulate activation states in response to changing environmental conditions – producing poor performance under sub-optimal conditions. Behavioral and electrophysiological studies involving the manipulation of event rate (ER) lend support to this idea.

Aim In this preliminary study, we extended this investigation by comparing pupil dilation, an established marker of cognitive effort allocation, in individuals with ADHD, and controls, in response to varying ERs on a simple cognitive task.

Methods Nineteen children with ADHD (age range: 8–14 years) and 21 controls (age range: 10–16 years) completed a target detection task under three different ERs (1300, 4000, and 8000 msec). Pupil dilation was monitored using an eye-tracker.

Results Our results show that for controls, pupil dilation to targets varied as a function of ER according to a "U" function – with fast and slow ERs inducing greater phasic dilation than the moderate ER. However, for children with ADHD the relationship was linear with dilation increasing as ER decreased.

Conclusions The results provide the first pupillary evidence suggestive of effort allocation dysregulation in ADHD especially under fast event rate conditions. Future studies should explore interventions to overcome effort allocation problems.

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Interrelationships between cortisol, cognition and dementia: A review of the literature and new own findings

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Introduction Cortisol exerts effects on the brain via two different receptors, producing complex and sometimes opposite effects on the brain structures involved with the different cognitive functions. Objective To scrutinize the interrelationships between cortisol, cognition and dementia.

Methods Review of the literature and new own findings.

Animal and clinical studies showed an association between increased cortisol and poorer overall cognitive performance, declarative memory, language, processing speed, executive functioning, spatial memory, as well as social memory. High cortisol may exhibit neurotoxic effects on the hippocampus, and exacerbate oxidative injury and amyloid β peptide toxicity. Increased CSF cortisol levels have been found in subjects with dementia and Mild Cognitive Impairment (MCI) due to Alzheimer's disease (AD) compared to control subjects with normal cognition. In MCI due to AD, high CSF cortisol may also predict a more rapid cognitive decline. Higher cortisol levels have been also observed in delirium. Increased cortisol levels interact with inflammatory mediators, neurotransmitters, and growth factors, and may mediate the effects of depression, stressful life events, and personality traits, sleep disturbances, and cardiovascular risk factor on cognitive performance and cognitive decline.