Neuroscience in Psychiatry

EPP586

Appetite-regulating hormones, executive functions, and eating attitudes in adults with ADHD: A case-control study

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Introduction: Although the literature suggests a strong association between attention deficit hyperactivity disorder (ADHD) and obesity, the underlying mechanisms remain unclear. Eating attitudes and appetite-regulating hormones (ARH) are considered to play a role in this relationship. Recent studies have shown that ARH may be linked to executive functions, and dysregulation of these hormones may help explain the connection between ADHD and obesity.
Objectives: We aimed to investigate the levels of ARH, executive functions, eating attitudes, and ADHD symptoms in adults with ADHD compared to healthy controls.
Methods: The study included 44 drug-naive non-obese adults with ADHD who had no comorbid psychiatric diagnoses and 44 healthy controls matched for age, gender, education, and body mass index (BMI). All participants were diagnostically assessed using the Structured Clinical Interview for DSM-5 Disorders-Clinician Version. Also, participants completed the Adult ADHD Self-Report Scale, the Mind Excessively Wandering Scale, the Hospital Anxiety and Depression Scale, and the Eating Attitude Test (EAT). A battery of neuropsychological tests—Stroop Test (ST), Cancellation Test, Serial Digit Learning Test (SDLT), Wisconsin Card Sorting Test (WCST), and Judgment of Line Orientation Test (JLOT)—was administered. The serum samples obtained from fasting blood, after centrifugation were stored at -80°C until the time of analysis, at which point ARH levels (insulin, leptin, neuropeptide Y, orexine A, ghrelin, adiponectin) were measured using the ELISA method. The study was approved by Selçuk University Local Ethics Committee with the decision numbered 2023/328.
Results: Adults with ADHD exhibited worse ADHD symptoms, disordered eating attitudes, more severe anxiety and depression, and reduced executive functioning compared to healthy controls. Although ADHD groups showed more disordered eating attitudes compared to healthy controls, there was no significant difference in ARH levels between the two groups; however, these hormone levels were associated with specific parameters from ST, SDLT, WCST, and JLOT. Linear regression analyses to identify factors associated with each ARH separately revealed significant F values, except for ghrelin, which explained a unique variance ranging from 23.5% to 36%. These results indicated that visuospatial ability was associated with each ARH levels, even after controlling for age, gender, years of education, body mass index, severity of disordered eating attitudes, and the absence of an ADHD diagnosis.
Conclusions: Our findings suggest that dysregulation of ARH may associate cognitive processes related to executive functioning independent of disordered eating attitudes, BMI, and ADHD diagnosis. However, these hormones may be mediating factors in relation between ADHD and obesity, and to figure out this relation, longitudinal clinical studies with larger samples are needed.

Disclosure of Interest: None Declared

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Balancing Dopamine: Managing Parkinson's Disease and Psychiatric Symptoms through Agonist-Antagonist Pharmacotherapy

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Introduction: Parkinson's disease (PD) is characterized by basal ganglia dopamine depletion, leading to motor symptoms. Psychiatric symptoms, like psychosis, are associated with dopamine overactivity in the mesolimbic and mesocortical system. PD patients sometimes require both motor and psychiatric treatment. However, the pharmacologic treatments for each have opposing dopaminergic effects. Understanding the balance between dopamine antagonists to treat psychosis while increasing dopamine agonists (DAs) to treat PD is a difficult clinical task.
Objectives: We aim to explore how dopamine agonists and antagonists affect the responsiveness of dopamine receptors in various brain regions.
Methods: We conducted a comprehensive literature review on pharmacological management of patients with PD and concurrent psychiatric symptoms. Special attention was given to balancing dopamine agonists (e.g., Carbidopa-Levodopa) for PD and dopamine antagonists (e.g., Quetiapine) for psychiatric symptoms.
Results: While dopamine agonists and antagonists appear counterintuitive when used concurrently, their efficacy is contingent on target brain regions. DAs (Carbidopa-Levodopa) are most beneficial for increasing dopamine deficits in the striatum and nigrostriatal pathway, where voluntary movement is controlled. In PD, this pathway is primarily affected, and DAs are used to target the striatum's high concentration of D1 and D2 sub-receptors. Gold-standard DAs mainly target D1 and D2 receptors.
Dopamine antagonists mitigate excess dopamine activity in the mesolimbic and mesocortical systems, which affect reward, memory, and executive functioning. These systems possess a high concentration of D3 and D4 sub-receptors. Typical antipsychotics have strong D2 receptor affinity, making them counterintuitive to DA motor treatment. Atypical antipsychotics have partial D2 affinity, but also readily bind D3 and D4. Some atypicals minimize D1/D2 affinity to allow DAs to be more effective. Quetiapine is currently used for psychosis in PD, but the drug's MOA is not fully understood. Cariprazine binds D3 well but does cause extrapyramidal effects with its D2 affinity. Clozapine strongly binds D4 but can have severe adverse effects. Additionally, pramipexole and ropinirole have strong D3 and D4 affinity. Ultimately, the key lies in symptom control: achieving optimal motor function while controlling psychiatric manifestations.
Conclusions: PD management with concurrent psychiatric disease requires diligent pharmacologic balance of countering dopamine treatments. Dopamine agonists and dopamine antagonists do have opposed pharmacodynamics, but clinicians must understand that certain pharmacologic agents do not all target the same brain area nor dopamine sub-receptor. Clinicians must use this pharmacologic knowledge to carefully balance these therapies by adapting to the individual patient's symptomatology and treatment response.

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