Impulse Control Disorders in Parkinson Disease

Dysregulated dopamine may not be the sole cause of impulse control problems in patients with Parkinson disease.

Replacing diminished dopamine is standard treatment for Parkinson's disease (PD), since loss of dopamine-producing substantia nigra neurons largely causes the degenerative neurological condition. But what happens when dopamine levels get too high?

A subset of those people with PD who are taking dopamine replacement therapies can develop impulse control disorders. This is not surprising, since the dopaminergic system helps to control decision-making and the sense of being rewarded for a behavior. Dysregulated dopamine may therefore underlie impulse control problems. These disorders are characterized by a lack of emotional and behavioral self-control, and categories according to the DSM-5 can include pyromania, kleptomania, and intermittent explosive rage attacks. Pathological gambling and hypersexuality can also occur in people with PD.

According to a recent study out of London, UK, specific structural brain differences might underlie the tendency for impulse control difficulties in people with PD taking medications that target the dopamine system.

The researchers, led by Clelia Pellicano of the Division of Brain Sciences, Neurology Imaging Unit, Imperial College London, London, UK, studied 18 PD patients with impulse control disorders, 18 people with PD but no impulse control disorder, and 24 age and sex-matched healthy control participants without either condition.

Pellicano and colleagues measured cortical thickness and subcortical nuclei volume using magnetic resonance imaging (MRI) and an “automated surface-based analysis package” called FreeSurfer. The scientists found statistically significant differences in MRI measurements between all three groups. Specifically, the nucleus accumbens of all individuals with PD, both with and without an impulse control disorder, had a loss of volume. People with PD and an impulse control disorder also had apparent volume loss in several other brain regions, including the caudate, hippocampus, and amygdala, when compared to the control subjects.

The researchers also noted statistically significant increased cortical thickness in several regions of the brains of people with PD and impulse control disorders when compared to people with PD and no impulse control problems. The increased regions included rostral anterior cingulate cortex and frontal pole as well as medial prefrontal regions.

The authors noted that the brain changes could either be traits already present that predisposed these individuals to developing impulse control disorders, or might alternatively have developed due to the use of the dopamine-stimulating medications. In their research report they noted, “We speculate that these findings reflect either a pre-existing neural trait vulnerability to impulsivity or the expression of a maladaptive synaptic plasticity under non-physiological dopaminergic stimulation.”

Future studies may help to shed light on whether or not the brain structural changes occur after dopamine replacement therapy, by measuring these brain differences prior to initiating PD medication. The study could help in treatment decisions about which therapy or what dose to use in treating people with PD, although avoiding dopamine-replacement medication for PD may be difficult or impossible since it is standard therapy. The research may help neurologists to predict who is likely to develop an impulse control disorder in response to PD medication.

**Key Points**

- Impulse control disorders can occur in people with PD in response to medications that increase dopamine.
- Statistically significant differences were measured in several regions of the brains of people with PD and impulse control disorders when compared to people with PD and no impulse control problems.
- Brain differences in people with PD and impulse control may occur either before or after dopamine treatment.
replacement medication.

References:


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