

Abstract

Obsessive-compulsive disorder (OCD) is a common, debilitating psychiatric illness characterized by recurrent, unwanted thoughts (obsessions) and conscious, ritualized acts (compulsions), usually attributed to attempts to deal with anxiety generated by the obsessions. Medications that are strong serotonin reuptake inhibitors and specific behavioral therapies that use the principles of exposure and response prevention (deconditioning) are effective in reducing the symptoms of OCD in many patients. Although the cause is uncertain, recently many investigators have postulated a role for a corticostriato-thalamic brain system in the mediation of OCD symptoms.

Background: We sought to determine in a new patient sample whether symptomatic improvement in obsessive-compulsive disorder treated with behavior modification is accompanied by significant changes in glucose metabolic rates in the caudate nucleus, measured with positron emission tomography, as seen in a previous study. Second, by combining samples from this and the previous study, we also examined whether there were pathologic correlational relationships among brain activity in the orbital cortex, caudate nucleus, and thalamus that obtained before behavioral treatment of obsessive-compulsive disorder, but that decreased significantly with symptom improvement.

Methods: Nine patients with obsessive-compulsive disorder were studied with positron emission tomography before and after 10 weeks of structured exposure and response prevention behavioral and cognitive treatment. Results were analyzed both alone and combined with those from nine similar subjects from the previous study.

Results: Behavior Therapy responders had significant ($p < .05$) bilateral decreases in caudate glucose metabolic rates that were greater than those seen in poor responders to treatment. Before treatment, there were significant correlations of brain activity between the orbital gyri and the head of the caudate nucleus and the orbital gyri and the thalamus on the right. These correlations decreased significantly after effective treatment.

Conclusions: These results replicate and extend previous findings of changes in caudate nucleus function with behavior therapy for obsessive-compulsive disorder. A prefrontal cortico-striato-thalamic brain system is implicated in mediation of symptoms of obsessive-compulsive disorder.

For more information on this study you are referred to the original journal article. The journal may be found in the periodical or reference department of major medical, university, or large public libraries. The reference librarian can be of assistance. The complete article citation follows:

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