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A paper published in the current issue of Psychotherapy and Psychosomatics provides new findings on the role of psychotherapy in regulating serotonin receptors.

This study was part of a larger project comparing psychotherapy and selective serotonin reuptake inhibitor (SSRI) drug treatment in major depressive disorder (MDD).

Patients with MDD were randomized to receive either fluoxetine 20-40 mg/day or brief psychodynamic psychotherapy for 4 months. Brain serotonin 5-HT1A receptors were measured before and after treatment with positron emission tomography and the radioligand [carbonyl-11C] WAY-100635.

Of all the patients in the study, 23 participated in the positron emission tomography part of the study: 8 from the psychotherapy group and 15 from the fluoxetine group. Clinical evaluations included (in addition to the main outcome measures HAM-D and Beck Depression Inventory) Social and Occupational Functioning Assessment Scale (SOFAS) and Social Adjustment Scale-Self-Report (SAS-SR) and Brief Symptom Inventory.

In both groups, the SOFAS scores increased in a similar way. In the whole group, increase in 5-HT1A receptor BPND was positively correlated with increase in SOFAS scores after treatment in the orbitofrontal cortex, suggesting that those who had the highest improvements in social and occupational functioning had the largest increases in 5-HT1A receptor BPND.

Further analyses indicated that this association was driven by patients receiving psychotherapy. In this group, increase in 5-HT1A receptor BPND was positively correlated with an increase in SOFAS scores after treatment in the orbitofrontal cortex, ventral anterior cingulate cortex , medial prefrontal cortex , and parietal cortex and lateral temporal cortex. Such correlations were not seen in the fluoxetine group.

This is the first study to show that the increase in the density of the 5-HT1A receptors after psychotherapy is strongly associated with the increase in social and occupational functioning. Thus, among depressed subjects, 5-HT1A may be a marker of social functioning, not of the severity of depression symptoms.

While both treatments improved SOFAS, only psychotherapy was associated with increase in 5-HT1A density. The reason for this may be that the serotonergic neurotransmission is enhanced by SSRI treatment in a different way than by psychotherapy.

Our findings suggest that SSRI medication, although leading to decreased symptoms and increased functioning in the short run, nevertheless is associated with an incomplete recovery of the serotonin system after treatment. This could be related to higher relapse risk.

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