Platelet serotonin transporters in schizophrenic patients

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Many hypotheses have been proposed for neurochemical imbalance in schizophrenia. One of these is that this disease is caused by serotonin dysfunction. Our previous study showed that drug-free schizophrenic patients had a significant increase in Bmax of 5-HT₂A receptors and declined to normal level after treatment with different neuroleptic drugs (Govitrapong et al. 2000). In order to examine the mechanism of the change of 5-HT₂A receptor in schizophrenia, the serotonin transporters on human platelets of 27 control subjects and 41 schizophrenic patients were determined in this study by using [³H]-imipramine and fluoxetine. The data showed that the maximum number (Bmax) of platelet serotonin transporter for schizophrenic patients without neuroleptic therapy was significantly higher than normal controls. The Bmax values for [³H]-imipramine binding to platelets of schizophrenic patients on phenothiazine butyrophenone, thioxanthene and serotonin–dopamine antagonist (SDA) therapies were significantly lower than those obtained from the drug–free group, but were comparable to control values. The effect of various medication periods on platelet serotonin transporter was also examined, and it was found that after 1–4 months, 4–12 months and more than 1 year of neuroleptic treatments, the Bmax values were significantly decreased when compared with values in the drug–free group. Significant clinical improvements occurred in all types of neuroleptic–treated groups and for all different treatment durations in this study. The mechanisms of how neuroleptics achieve their therapeutic effects still need to be further evaluated.

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