In search of the mode of action of antidepressants: 5-HTP/tyrosine mixtures in depression.

van Praag HM.

Abstract
For a long time, antidepressants have been considered to act via enhancement of central MA-ergic activity (due to reuptake or MAO inhibition). An alternative hypothesis holds that their action is based on down-regulation of MA-ergic activity (due to decrease in density or sensitivity of certain receptor populations). In this chapter I have discussed the likelihood of both hypotheses and have reached the conclusion that the first one is the more plausible. I have discussed the following arguments: The 5-HT precursor 5-HTP, which is transformed to 5-HT in the brain, has antidepressant properties. There are indications that the same holds true for tyrosine, a CA precursor transformed in the brain to DA and NE. I found evidence that the 5-HTP effects in depression are potentiated by tyrosine. Since activation rather than suppression of MA-ergic activity seems to be linked to antidepressant activity, it seems likely that the signs of decreased MA metabolism that has been demonstrated in certain types of depression are the expression of a primary metabolic deficit rather than a phenomenon secondary to receptor hyper-sensitivity. Further clinical studies of 5-HT/CA precursor combinations in depression are justified.

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