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NEUROCHEMICAL, BEHAVIOURAL AND MOLECULAR LEVEL STUDIES OF METHYLPHENDIATE IN RATS

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Methylphenidate (MPD) is widely prescribed drug for the treatment of attention deficit hyperactivity disorder (ADHD). Despite its therapeutic importance, there is growing evidence that patients treated with MPD develop an addiction to their therapy. The drug is also used as a cognitive enhancer to improve academic performances. It is therefore important to monitor abuse potential of clinically useful doses of MPD and molecular mechanism associated with its cognition enhancing and reinforcing effect. The present study is designed to study abuse potential, if any, of clinically relevant doses of MPD. The levels of dopamine (DA), dihydroxphenyl acetic acid (DOPAC), homovanillic acid (HVA), 5-hydroxytryptamine (5-HT), 5-hydroxyindole acetic acid (5-HIAA), and noradrenaline-hydrochloride (NA-HCl) are monitored in the hippocampus and caudate. In view of role of 5HT-1A receptor in cognition as well as addiction, the expression of 5-HT1A receptors in the prefrontal cortex and nucleus accumbens is monitored in rats repeatedly treated with MPD. We report that lower doses (0.5 and 2.5 mg/kg) of MPD enhance learning acquisition and memory retention in a dose dependent manner in Morris water-maze test. Higher dose (5 mg/kg) of MPD however impairs these. The drug administered repeatedly at dose of 2.5 mg/kg is reinforcing in conditioned place preference paradigm. Sensitization like effect produced transient and are not consistently shown. Result shows an increase in 5-HT metabolism in the hippocampus as well as caudate. Effects of DA metabolism are not consistent. HVA levels are decrease markedly in hippocampus but are increases in the caudate. The expression of 5HT-1A receptor attenuated markedly in the nucleus accumbens, but no effect on 5HT-1A receptor occurs in the prefrontal cortex. The results strengthen our previous studies of a role of 5HT-1A receptors in addiction. The findings may be of use in improving therapeutics in ADHD and developing non addictive cognitive enhancers.

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