

PRELIMINARY COMMUNICATION

INHIBITION OF ARACHIDONIC ACID CASCADE ATTENUATES THE INDUCTION OF c-FOS PROTEINS BY DOI, 5-HT_{2A/2C} RECEPTOR AGONIST, IN THE RAT CORTEX

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Inhibition of arachidonic acid cascade attenuates the induction of c-Fos proteins by DOI, 5-HT_{2A/2C} receptor agonist, in the rat cortex. M. MAĆKOWIAK, A. CZYRAK, K. WĘDZONY. *Pol. J. Pharmacol.*, 2002, 54, 73–76.

Previous immunohistochemical studies have shown that c-Fos proteins induced by DOI, a 5-HT_{2A/2C} agonist, are present in the population of cortical neurons, which are devoid of 5-HT_{2A} receptors. A mechanism of the induction of c-Fos proteins expression by DOI is still unclear. However, the involvement of the 5-HT_{2A} and AMPA, but not 5-HT_{2C} receptors in this process has been reported. In the present study, we investigated whether arachidonic acid, a retrograde messenger, is involved in the above mechanism of c-Fos induction. Phospholipase A₂ pathway, which leads to the subsequent generation of arachidonic acid and its metabolites, is known to be coupled to 5-HT_{2A} receptor activation. The inhibition of arachidonic acid cascade both at the level of phospholipase A₂ (by dexamethasone, 1.5 mg/kg) or at the level of cyclooxygenases that catalyze arachidonic acid biotransformation (by indomethacin, 3 mg/kg), decreased the number of c-Fos immunopositive cells after induction by DOI (8 mg/kg). Our results suggest that arachidonic acid cascade may be involved in the induction of c-Fos proteins by DOI in the rat parietal cortex.

Key words: DOI, c-Fos proteins, phospholipase A₂, arachidonic acid, dexamethasone, indomethacin

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