

NEUROCHEMICAL AND PHARMACOLOGICAL ASPECTS OF COCAINE-INDUCED SEIZURES

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Seizures associated with cocaine intoxication are serious clinical problem requiring immediate and adequate treatment, however their mechanism has not been fully elucidated. In contrast to early views, which convulsive properties of cocaine ascribed predominantly to the effect of this drug on voltage-dependent sodium channels, recent reports put much emphasis on the interaction of cocaine with GABAergic and glutamatergic systems. Accordingly, pharmacological studies demonstrated that cocaine-induced seizures were efficiently inhibited by GABA-A receptor agonists and NMDA receptor antagonists, whereas sodium and calcium channel blockers were ineffective. An involvement of serotonin 5-HT₂, dopamine and sigma receptors in cocaine-induced seizures has also been proposed. Furthermore, adaptive changes in various neuronal systems following cocaine-induced seizures has been vigorously investigated. Some of those changes, such as expression of immediate early genes and increase in neuropeptide biosynthesis may play a compensatory anticonvulsive role, however, other alterations e.g. up-regulation of NMDA receptors may increase susceptibility to seizures. This short review summarises recent advances in basic research on some neurochemical and pharmacological aspects of cocaine-induced seizures.

Key words: cocaine-induced seizures, GABA receptors, NMDA receptors, glutamatergic system
