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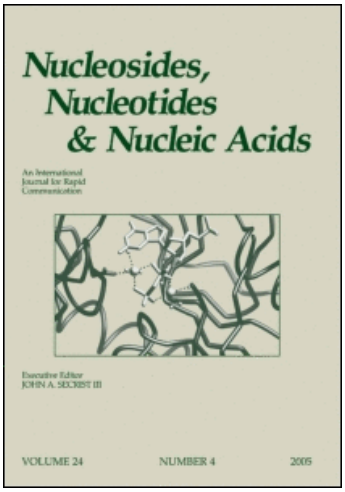

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Unilateral Entorhinal Cortex Lesion - An Animal Model For Cognitive Impairment in Human Disease: Effects on Adenosine Receptors and Second Messengers

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UNILATERAL ENTORHINAL CORTEX LESION - AN ANIMAL MODEL FOR COGNITIVE IMPAIRMENT IN HUMAN DISEASE: EFFECTS ON ADENOSINE RECEPTORS AND SECOND MESSENGERS

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Abstract: Entorhinal cortex pathology has been demonstrated in several neuropsychiatric diseases. Decreased binding to adenosine A1 receptors and adenylate cyclase in the dentate gyrus after entorhinal cortex lesion indicates impaired adenosinergic neuromodulation in these diseases.

Pathological changes in the entorhinal cortex have been demonstrated in several brain disorders with cognitive impairment as schizophrenia, Alzheimer`s disease and Parkinson`s disease (e.g. [1]). Adenosine is a major inhibitory neuromodulator of entorhinal cortex neurons providing 85% of the synaptic input to the the outer two thirds of the molecular layer of the dentate gyrus [2,3]. Adenosine receptor antagonists caffeine and theophylline are consumed as "cognitive enhancers" [4].

The right entorhinal cortex of five male Wistar rats was removed surgically [5]. On the fourth postoperative day the five operated and five control animals were sacrificed and 20 micron cryostat sections were obtained. Binding of (^{3}H)cyclohexyladenosine, (^{3}H)5`-N-ethylcarboxamidoadenosine,

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(^{3}H)nitrobenzylthioinosine, (^{3}H)forskolin, (^{3}H)phorboldibutyrate ester and (^{3}H)nitrendipine to slide-mounted sections was performed [6-9]. Quantitative analysis was done using (^{3}H)microscales and an image processing system.

(^{3}H)cyclohexyladenosine and (^{3}H)forskolin binding to adenosine A1 receptors and adenylate cyclase were reduced by 40 and 25 percent resp. in the molecular layer of the right dentate gyrus. Binding of ligands to adenosine non-A1 receptors, adenosine uptake sites, protein kinase C and L-type calcium channels appeared not affected.

In human neuropsychiatric diseases with cognitive impairment and entorhinal cortex pathology adenosinergic neuromodulation in hippocampus may be compromised.

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