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P tritium-labeled 'serotonin' /Amersham-Searle/ and 'LSD' /NEN/
Z specific binding to synaptic 'receptor' cf. tritium-labeled serotonin uptake by synaptosome intact cf. 'anthelmintic' 'pyrrole' intracerebral kainic-acid /Sigma-Chem./ induced 'brain' caudate-nucleus lesion 'lab.animal' model of chron.'chorea' rat /X/ /XXVI/ /XXXII/

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Loss of striatal serotonin synaptic receptor binding induced by kainic acid lesions: correlations with Huntington's Disease.

The effects of kainic acid (KA) on striatal serotonin (5-HT) synaptic receptor binding were studied.

Methods

2 µg KA (Sigma-Chem.) was injected into the rat caudate nucleus. After decapitation, performed 10 days later, the brains were removed and dissected. The lesioned striata and contralateral non-lesioned striata were analyzed for 5-HT uptake, using Krebs-Ringer-Tris medium. (^{3}H)5-HT (Amersham Searle) was added. Non-specific (^{3}H)-5-HT uptake was estimated using 10 µM Lilly 110140.

5-HT synaptic receptor binding was determined by homogenizing the frozen striata in Tris-hydrochloride buffer. Aliquots of homogenate were incubated with (^{3}H)-LSD (New England Nuclear) or (^{3}H)-5-HT and binding determined.

Results

Specific binding of (^{3}H)-LSD and (^{3}H)-5-HT to membrane preparations from KA-lesioned caudate nuclei was reduced 60-70% below values for contralateral un-lesioned caudate nuclei. Binding of both ligands was inhibited approximately the same percentate in lesioned and non-lesioned tissue by unlabeled LSD, 5-HT and 2-bromo-LSD. The integrity of presynaptic 5-HT terminals determined by synaptosomal high-affinity (^{3}H)-5-HT uptake was not changed by the lesions.

Conclusion

Since the decreases in (^{3}H)-LSD and (^{3}H)-5-HT binding to KA-lesioned caudate nuclei were similar in magnitude to those decreases found in neuronally depleted Huntington's disease caudate nuclei, this strengthened the usefulness of striatial KA lesions as an animal model for Huntington's disease.

1 Tab. 14 Ref. NA100/GM/WS

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