



EFFECTS OF SYNTHETIC PYRETHROID, DECAMETHRIN,
ON NEUROTRANSMITTER SYSTEMS OF CORPUS STRIATUM OF THE RAT

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ABSTRACT

Pyrethrum extract and synthetic pyrethroids are popular household insecticides; they are thought to have relatively low human and animal toxicity. Decamethrin, a synthetic pyrethroid, reportedly produced abnormal motor activities, stereotyped behaviors and convulsion. Recently, the corpus striatum has been shown to be the center of abnormal discharges that spread to other parts of the brain following administration of decamethrin; reduction of as much as half of the normal content of acetylcholine in the brain was also reported. The results summarized here represent the findings in our attempt to study the effects of decamethrin on various neurotransmitter systems in the corpus striatum.

Rats treated with 50 mg/kg decamethrin intraperitoneally developed various symptoms as reported by other investigators. Peak action of decamethrin was 1.5-2 hr after injection. The animals were sacrificed at this time, the corpus striatum was removed for further studies.

Striatum dopamine content remained within normal range; but metabolites of dopamine (HVA and DOPAC) were elevated by decamethrin administration. Decamethrin thus appeared to increase the turn-over rate of dopamine; the increase in release of dopamine may indicate accentuated activity of the dopamine system.

Behavioral and electrophysiological studies also indicated that decamethrin enhanced dopaminergic activity in the brain. Thus, stereotyped behaviors typical to elevated dopaminergic discharge were observed, which were blocked by specific dopamine antagonists, haloperidol and chlorpromazine. Extracellular microelectrode recording from single neurons in the substantia nigra pars compacta, the dopaminergic neurons, revealed marked increase of neuronal discharge following decamethrin administration. However, binding study showed that this insecticide had no affinity toward the dopamine receptor.

There was also significant reduction in striatal muscarinic cholinceptor. Radio-receptor assay of the membrane fraction obtained from homogenate of the corpus striatum showed clearly that the quinuclidinyl benzilate binding site was reduced by decamethrin treatment without alteration in ligand binding affinity.

It was observed that symptoms of decamethrin toxicity do collectively resemble those of Huntington's chorea. Reduction in cholinceptor density was also reported in this disease. In view that the etiology of Huntington's chorea is far from being clear in sufficient details, decamethrin may serve as a valuable tool in producing animal model of this choreiform disease for further investigation.

