



Advances in Molecular Toxicology: Chapter Five. Noncholinesterase Protein Targets of Organophosphorus Pesticides

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The acute toxicity of organophosphorus (OP) nerve agents and pesticides is due to inhibition of acetylcholinesterase (AChE) activity in cholinergic nerve synapses. Delayed neuropathy involves OP binding to neuropathy target esterase (NTE) in neurons. AChE and NTE are serine hydrolases. In vitro studies have demonstrated that OP toxicants bind not only to serine hydrolases but also to proteins that have no active site serine, for example, Tyr 411 of human albumin and Lys 296 of mouse transferrin. Mice treated with low doses of chlorpyrifos have OP-modified tubulin in brain. Mass spectrometry has identified OP-modified albumin in the blood of humans self-poisoned by chlorpyrifos and dichlorvos. Albumin is weakly reactive with OP, but the presence of OP-modified albumin in humans suggests the existence of additional noncholinesterase OP targets. In conclusion, long-term adverse effects from OP exposure may involve OP modification of proteins that have no active site serine.

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