DFP, dimefox, schradan, TEPP, and parathion had been reported; all of these compounds have characteristic acute mammalian toxicity and two of these are important nerve gases. The introduction of low mammalian toxicity OP compounds, such as malathion, coupled with the problem of pest resistance to organochlorines, led to the production of OP compounds as the dominant class of insecticides for the next 30 years. The environmental toxicology of OP has garnered significant research interest among academic, industrial, and government research scientists. The primary focus of the text is the biochemical and toxicology of insecticidal organophosphorus compounds. The book was developed from an Agrochemicals Division, American Chemical Society symposium (bearing the same title), held in Boston, MA, in April 1990.

The text is divided into five sections: Introduction, Metabolic Fate, Toxic Effects—Noncholinergic Biochemical, Toxic Effects—Organismal, and Summary and Conclusions. The introductory material is a general and simplified overview of the broad scope of subject matter on OP compounds and their toxicology. This section includes historical material on OP chemistry; OP chemistry (classification, major synthetic routes, and chemical reactions); acetylcholinesterase OP biochemistry; secondary toxicity not related to acetylcholinesterase inhibition; and the metabolism of OP compounds by cytochrome P$_{450}$, glutathione transferases, and esterases. The second text section, Metabolic Fate, contains seven chapters describing the metabolism of OP compounds. The first four chapters cover the roles of flavin-containing monoxygenases, glutathione, phosphodiester hydrolases and carboxylesterase enyzmology in OP toxicological biochemistry. The remaining chapters describe the hepatic disposition of OP insecticides, the role of target-site activation of phosphorothionates, and transdermal transport and metabolism. The third section, Toxic Effects—Noncholinergic Biochemical, provides an overview of recent developments in OP toxicology that describe noncholinergic effects such as teratogenicity, delayed neuropathy, and immunotoxicity. Acute OP toxicity may be classified into muscarinic (parasympathetic), nicotinic (sympathetic and motor), and CNS symptoms; the type class is determined by the cellular site of action. The first two papers discuss the direct effect of OP compounds by inhibition of ligand binding to muscarinic acetylcholine receptors and secondary action on muscarinic receptors that are coupled to other effectors to mediate second messenger systems. Accounts of chronic, low-dosage, OP-induced tolerance has been reported; the third chapter in this section discusses several biochemical mechanisms for OP tolerance. The last paper in this section describes the interaction of OP compounds with esterases; these afford improved methods for predicting the OP neurotoxic potential. The fourth section, Toxic Effects—Organismal, contains five papers that give detailed analysis of OP compound toxicity by structure type with associated effects, a rodent model of OP-induced delayed neuropathy (OPIDN), immunotoxicity, teratogenic, and neurobehavioral effects (human and animal studies). The Summary and Conclusions section provides a brief overview of the OP research to date and clarification of the complex biochemical mechanisms of OP neurotoxicity.