

## The treatment of acetaminophen poisoning.

[Annu Rev Pharmacol Toxicol. 1983;23:87-101](#)

[Prescott LF](#), [Critchley JA](#).

Acetaminophen has become a very popular over-the-counter analgesic in some countries and as a result it is used increasingly as an agent for self-poisoning. Without treatment only a minority of patients develop severe liver damage and 1 to 2% die in hepatic failure. Until Mitchell and his colleagues discovered the biochemical mechanisms of toxicity in 1973 there was no effective treatment. They showed that the metabolic activation of acetaminophen resulted in the formation of a reactive arylating intermediate, and that hepatic reduced glutathione played an essential protective role by preferential conjugation and inactivation of the metabolite. Early treatment with sulphhydryl compounds and glutathione precursors has been dramatically effective in preventing liver damage, renal failure, and death following acetaminophen overdosage. It seems likely that these agents act primarily by stimulating glutathione synthesis. Inhibition of the metabolic activation of acetaminophen is another potential therapeutic approach that has not yet been put to the test clinically. The clinical management of acetaminophen poisoning has been transformed and it is particularly gratifying to have effective treatment based on a well established biochemical mechanism of toxicity. It is likely that effective treatment will be developed for toxicity caused through similar mechanisms by other agents.

