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top notch 1 second edition free pdf 14 top notch 1 workbook second edition Rat liver alcohol dehydrogenase and acetaldehyde dehydrogenase from quetiapine-treated rats: a possible mode of action of the neuroleptic. The activity of two hepatic enzymes, alcohol dehydrogenase (ADH) and acetaldehyde dehydrogenase (ALDH), has been studied in quetiapine-treated rats. When the enzyme activity was assayed in the presence of a non-selective inhibitor of NAD(P)(+)-dependent dehydrogenases, tyrosine, the activity of both enzymes was decreased in a dose-dependent manner. Measurements of the enzymatic activity with guanosine-5'-O-(3-thiotriphosphate) (GTP γ S) revealed that the K_m values were unaltered. These data suggest that inhibition of both enzymes by quetiapine is due to an allosteric alteration. ADH and ALDH were assayed in the presence of cycloheximide. The results indicated that protein synthesis is not necessary for enzyme

inhibition by the drug. When animals were treated with [3H]quetiapine and the drug was measured in the liver after bromine-DMSO extraction, the metabolism of the neuroleptic was significantly decreased. Taken together, the data indicate that quetiapine is able to specifically inhibit the two main metabolic enzymes of alcohol in the liver, ADH and ALDH. In an attempt to establish the role of the enzyme inhibition in the determination of the neurotoxic effects of the drug, the administration of ADH- and ALDH-inhibiting doses of quetiapine was performed to rats for 13 weeks. It was demonstrated that both doses decreased the activity of the enzymes in comparison with controls, although the inhibition was less marked with the higher dose. The results from the neurobehavioral testing (open-field test and evaluation of seizure intensity, onset and duration) demonstrated that both doses were able to induce significant changes in some of the parameters, although these changes were not directly correlated to the enzyme inhibition. The concomitant decrease in enzyme activity together with the other biochemical and behavioral parameters may explain the neurotoxicity observed in humans and in animal models. h t e r m o f - 7 0 7 , - 7

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