With the compliment of Dr. A. M. Tom 4B28

DIRECT CHEMICAL QUENCHING OF REACTIVE INTERMEDIATES FORMED DURING THE PROPAGATION PROCESS OF MICROSOMAL LIPID PEROXIDATION BY RETINOIDS

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It has been reported that a higher incidence of chemical-induced tumours in vitamin A-deficient animals could be due to alterations in the activity of certain microsomal oxidation-reduction enzymes. Previously, we have demonstrated that such changes could be related to a loss of vitamin A protection against free-radical induced membrane lipid peroxidation. In this study, we examined further the possible role of vitamin A in the protection of microsomal membrane lipids from oxidative damage. Retinyl acetate strongly suppressed NADPH-dependent hepatic microsomal lipid peroxidation (IC_{50} 0.4 μM) initiated by Fe(III)-ADP complex, however, both hydrogen peroxide and superoxide generation were not affected at 100 μM concentration. Cytochrome P-450 activities were only slightly affected by high concentrations of retinyl acetate (e.g. aminopyrine N-demethylation was inhibited by 15% at 100 μM concentration), suggesting that inhibition of microsomal electron transport is not a mechanism for suppression of lipid peroxidation. Moreover, retinyl acetate was found to be more effective in the suppression of propagation of free-radical intermediates following the initiation of lipid peroxidation. This further suggests that vitamin A acts mainly as a chemical quencher of reactive intermediate products formed during the lipid peroxidation process.
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