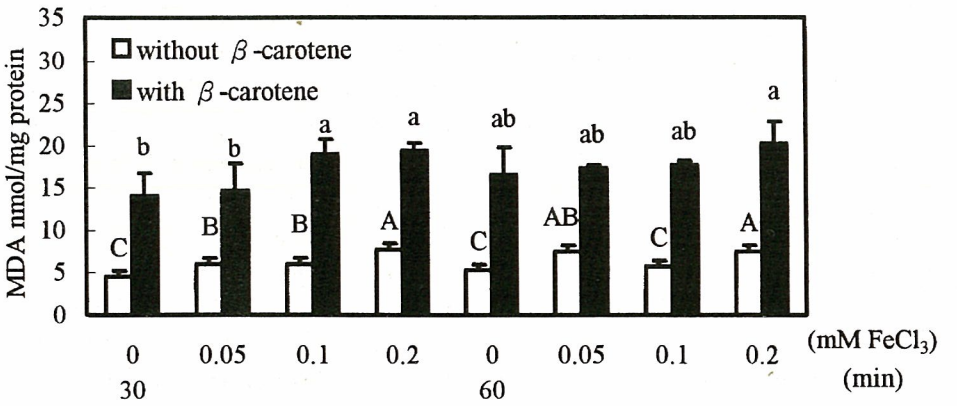


Fig. 5. Effect of $FeCl_3$ on thiobarbituric acid-reactive substances (malondialdehyde, MDA) in primary rat hepatocytes isolated from rats fed an AIN-76 diet with or without 0.01% β-carotene.

* Values with different superscripts in the same color bar significantly differ from one another at $p<0.05$ as determined by ANOVA and Duncan`s multiple range test. a, b: β-carotene diet. A, B, C: β-carotene-free diet.

** The concentration of MDA significantly differ ($p = 0.0001$) between feeding the β- carotene and β-carotene-free diets.

were incubated with 0.05 mM $FeCl_3$ or without $FeCl_3$. But GSH-Px activity was significantly increased in rats fed a β-carotene diet than a β-carotene-free diet when primary rat hepatocytes were incubated with 0.1 and 0.2 mM $FeCl_3$ for 60 min ($p < 0.05$) (Fig. 4).

MDA concentrations of rats fed the β-carotene diet were significantly greater than those fed the β-carotene-free diet when primary rat hepatocytes were incubated with 0.05~0.2 mM $FeCl_3$ ($p = 0.0001$) (Fig. 5). MDA concentrations of rats fed the β-carotene diet were also significantly greater than those fed the β-carotene-free diet when primary rat hepatocytes were incubated without $FeCl_3$.

## DISCUSSION

The need to examine the extent and mechanisms of the prooxidant actions of β-carotene has resulted in an increasing number of in vitro studies.[18] In this present study, we measured the effect of $FeCl_3$ on cell integrity and lipid peroxidation in primary rat hepatocyes from rats fed an AIN-76 diet with or without β-carotene (Fig. 1). This study found that the LDH leakage percentage and MDA concentrations of cells from rats fed the β-carotene diet were significantly greater than those of rats fed the β-carotene-free diet when primary rat hepatocytes were incubated with 0.05~0.2 mM $FeCl_3$ ($p = 0.0001$). The LDH leakage and MDA concentrations of rats fed the β-carotene diet were also significantly greater than those of rats fed the β-carotene-free diet when primary rat hepatocytes were incubated without $FeCl_3$. These results show that β-carotene is a prooxidant agent in this biological system.

Many of the reported results have been demonstrated only in vitro and not in vivo. Moreover, the β-carotene products directly responsible for the prooxidant activity have not yet been identified.[18] Previous studies indicated the prooxidant-antioxidant actions of β-carotene in vivo. Very little work has been directed at investigating the effects of β-carotene supplementation in vivo, which at the same time observing in vitro cell viability and the antioxidative system of primary rat hepatocytes.

Hydroxyl radicals (•OH) formed by iron-catalyzed reactions and/or iron-oxygen species may initiate the lipid peroxidation process. In addition, iron can catalyze the breakdown of lipid peroxides into alkoxyl and peroxyl free radicals.[10] Free radicals are chemical species with 1 or more unpaired electrons. Under normal physiological conditions, organisms can prevent free-radical damage by protective mechanism which in-