volve SOD, CAT, and GSH-Px. In this study, the SOD activity of rats fed the β-carotene diet was significantly lower than that of rats fed the β-carotene-free diet (p = 0.0023) when primary rat hepatocytes were incubated with 0.05~0.2 mM $FeCl_3$ for 30 and 60 min (Fig. 2). The SOD activity of rats fed the β-carotene diet was also significantly less than that of rats fed the β-carotene-free diet when primary rat hepatocytes were incubated without $FeCl_3$. This result shows that β-carotene may act as a prooxidant agent in this biological system.

However, the CAT activity of rats fed the β-carotene diet was significantly greater than that 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) (Fig. 3). The CAT activity of rats fed the β-carotene diet was significantly greater than that of rats fed the β-carotene-free diet when primary rat hepatocytes were incubated without $FeCl_3$. This indicates that the antioxidant enzyme activity was modified in this study.

The GSH-Px activity of cells from rats was not affected by diets with or without β-carotene supplementation in primary rat hepatocytes incubated with 0.05 mM $FeCl_3$ or without $FeCl_3$. But the GSH-Px activity of cells from rats fed the β-carotene diet was significantly greater than that of rats fed the β-carotene-free diet in the presence of 0.1~0.2 mM $FeCl_3$ for 60 min (p < 0.05) (Fig. 4). This indicates that the antioxidant enzyme activity was modified in this study.

β-carotene was effective in this study system at concentrations of 0.0532 nmol/g rat liver tissue when the rats were fed a diet with 0.01% β-carotene. A prooxidant is an agent that can induce oxidative stress, which is defined as a shift in the prooxidant-antioxidant balance towards oxidant activity. The oxidative stress induced by a prooxidant agent in biological systems manifests itself as increased production of bioactive free-radical species, a decrease of the antioxidant defenses, and/or an increase in oxidative damage (oxidation of lipids, proteins, and DNA).[18] Improved knowledge of the prooxidant role of carotenoids in vitro and in vivo will help us understand their potential to influence biological processes in humans.[18]

The role of β-carotene in antioxidative efficacy in vivo is still controversial, although it has been shown to function as an antioxidant in many in vitro systems.[3] A plausible mechanism for the potential anticarcinogenic effects of β-carotene is its ability to scavenge reactive oxygen species that cause oxidative DNA damage. However, 2 recent intervention trials, 1 in Finland[4] and 1 in the USA,[5] unexpectedly observed an increased risk of lung cancer in smokers who were given high-doses (20-30 mg) of β-carotene supplements each day. Van Poppel et al.[27] reported the effect of β-carotene on increased DNA damage as assessed by urinary excretion of 8-oxo-7,8- dihydro-2-deoxyguanosine in male cigarette smokers. However, other studies have shown the beneficial effects of β-carotene in reducing cancer risk.[6]

The liver, with its effects on homeostasis in mammals, plays a complex role in the intact organism. The hepatic parenchymal cells in this monolayer system are viable and behave in many respects like normal adult rat livers.[28]

Recently, we reported that in rats fed diets supplemented with 0.01%, 0.1%, 0.2% β-carotene for 6 weeks,[29,30] the β-carotene concentration of the liver was lower in rats fed the diet with 0.01% β-carotene as compared to those of the 0.1% and 0.2% groups. The liver is the main β-carotene storage organ in rats. In this study, the β-carotene concentration of the liver was 0.532 nmol/g after 6 weeks of feeding with a diet containing 0.01% β-carotene.

In conclusion, this study shows the efficacy of β-carotene in inhibiting $FeCl_3$-induced oxidative stress in a cell system using a primary culture of rat hepatocytes. These results indicate that β-carotene without $FeCl_3$-induced oxidative stress acts to shift the prooxidant-antioxidant balance towards antioxidant activity.

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