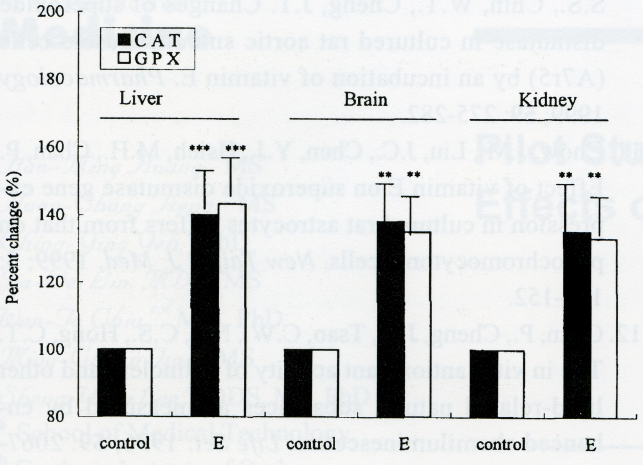

Fig. 3. Percentage of changes of catalase (CAT) and glutathione peroxidase (GPX) activity in rat organs ($n = 8$) after 3 days of supplementation with vitamin E (200 mg/ml/kg + 0.5% ethanol saline). The control group used 0.5% ethanol in saline. E = vitamin E. $^{**}p < 0.01$, $^{***}p < 0.001$. (vitamin E vs. control).

Other natural antioxidant enzymes such as catalase and glutathione peroxidase, which were employed as references, also showed similar changes to those of SOD (Fig. 3).

DISCUSSION

Enzymes protecting against free radicals include SOD, catalase, and glutathione peroxidase. SOD catalyzes the conversion of the superoxide anion radical into H_2O_2. H_2O_2 is removed by glutathione peroxidase which catalyzes its reduction to H_2O, while converting the reduced glutathione (GSH) into oxidized glutathione (GSSG). Catalase also decomposes H_2O_2 to oxygen and water. The brain appears to be relatively poorly endowed with glutathione peroxidase and catalase,[8] and this, in conjunction with its relatively high concentrations of polyunsaturated fatty acids (PUFAs), may account for its susceptibility to ischemia/reperfusion. Furthermore, many regions of the brain are enriched in iron,[9] which possesses an ability to catalyze the formation of oxygen radicals. Vitamin E (α-tocopherol) is a lipophilic compound which is present in relatively high concentrations in both plasma and mitochondrial membranes. It reacts with lipid peroxyl radicals to form lipid hydroperoxides which can then be removed by the phospholipase-glutathione peroxidase system.

Our previous study showed that incubation of rat aortic smooth muscle cells with vitamin E at an optimal concentration can increase the activity of SOD.[10] In this study, we demonstrate that the administration of vitamin E is not only beneficial in the treatment to OFR injury, but also prevents vital organ damage through better tolerability to OFRs. This phenomenon is compatible with findings of our previous study that vitamin E can also increase SOD gene expression in cultured brain astrocytes of the rat.[11] Moreover, the increasing mRNA of antioxidant enzymes suggests that vitamin E promotes antioxidant activity through a change in gene expression. The limitation of this study is that we did not perform dose-response experiments. However, in our previous report, we found that vitamin E at this concentration has the most potent antioxidant activity.[12]

In conclusion, the lipophilic antioxidant, vitamin E, is effective in increasing SOD activity and mRNA gene expression. Whether this phenomenon is beneficial in a clinical setting needs further evaluation.

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