Effect of peptide extract from beef myofibrillar protein on oxidative stress in the brains of spontaneously hypertensive rats

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Introduction: Hypertension is associated with increased oxidative stress in brain [1], and our previous studies showed the ACE inhibitory activity and anti-hypertension effects of peptides [2,3]. The current review suggests that brain health is an important factor in late stage hypertensive disease because of the correlation of hypertension with the aging of the brain [4,5]. A previous study showed long-lasting activity of ACE inhibitors as hypertension agents in spontaneous hypertensive rat (SHR) brains [6].

Materials and methods: This study was conducted to determine the effect of beef peptide extract on oxidative stress in the brains of spontaneously hypertensive rats (SHRs). A 3-kDa peptide extract was obtained from beef myofibrillar protein using alkaline-AK (AK3K). Oxidative stress in SHR brains was measured by assessing malondialdehyde (MDA) and reactive oxygen species (ROS) concentrations and superoxide dismutase (SOD), catalase, and glutathione peroxidase (GPx) activity.

Results: This study was conducted to determine the anti-oxidative stress effect of the peptide extract from beef myofibrillar protein on the SHR brain by examining different oxidative stress factors such as MDA, ROS, SOD, catalase, and GPx activity. The MDA concentration in SHR brains treated with the AK3K peptide extracted at 400 and 800 mg/mL was 0.33 ± 0.09 and 0.40 ± 0.10 μM, respectively, which was significantly lower than that of the control and captopril treatment groups ($p < 0.05$). In addition, the ROS content of SHR brains treated with captopril and AK3K peptide extracts at 400, 800 mg/mL was 23.92 ± 4.56, 23.12 ± 1.00, and 21.71 ± 4.16 μM, respectively. The decrease in ROS content in the SHR brain was significantly greater in the AK3K peptide extract treatment groups compared to that in the control group ($p < 0.05$). The SOD activity in SHR brains was significantly increased by treatment with AK3K400 and captopril compared to the control ($p < 0.05$). However, the SOD activity of the SHR brain treated with AK3K800 was not significantly different from that in the control group. The catalase activity and GPx activity were not significantly different between groups. Therefore, SOD plays a key role in controlling oxidative stress in the SHR brain via the inhibition of ROS and MDA production. Furthermore, this study showed that the peptide extract from beef myofibrillar protein could help to reduce neuronal hypertension through the inhibition of oxidative stress in the brain.

Conclusions: This study showed that the peptide extract from beef myofibrillar protein obtained using alkaline-AK has an anti-oxidative stress effect related to hypertension in the SHR brain. The SHRs treated with AK3K peptide extracts showed a significant decrease in the MDA level and ROS generation compared to the control. The SOD activities in SHRs treated with AK3K peptide extract (400 mg/mL) and captopril were significantly higher compared to those in the other treatment groups. However, the catalase and GPx activities in SHRs treated with each sample were not significantly different between groups. Based on the results of this study, we assume that the anti-oxidative stress property of the AK3K peptide extract can alleviate neuronal hypertension. Furthermore, the AK3K peptide extract could possibly be used in neuronal hypertension therapy.

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