Peptides from hydrolysate of lantern fish (Benthosema pterotum) proved neuroprotective in vitro and in vivo

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ABSTRACT

Benthosema pterotum is an underutilized deep-sea by-catch fish. Its protein hydrolysate (BPH) showed antioxidative activity. The BPH consisting of 13.2 mg/g of active peptides, Phe-Tyr-Tyr and Asp-Trp, significantly reduced H2O2-induced reactive oxygen species (ROS) and apoptotic cell death in human neuroblastoma SH-SY5Y cells through activation of intracellular antioxidant defence system. In Morris water-maze test, BPH was able to ameliorate memory and learning deficiency of D-galactose (D-gal)-induced neurodegenerative/ageing ICR mice. BPH-fed mice showed significantly lower levels of both D-gal-induced thiobarbituric acid reactive substances (TBARS) and endothelial nitric oxide synthase (eNOS), but higher levels of glucose-6-phosphate dehydrogenase (G6PDH) and brain-derived neurotrophic factor (BDNF) in brain of ageing mice in comparison to the control. In light of these results, BPH may be considered as a novel nutraceutical for easing the ageing and/or age-related neurodegenerative diseases.

1. Introduction

Oxidative stress and unexpected apoptotic cell death have been implicated in the pathogenesis of age-related neurodegenerative diseases, such as Alzheimer’s disease (AD) and Parkinson diseases. AD with high occurrence in ageing population manifests two noticeable symptoms, progressive memory loss and cognitive dysfunction (Wang et al., 2013a). Growing evidences showed that oxidative stress resulting from reactive oxygen species (ROS) seriously affects AD animals and patients (Lee et al., 2008; Nagatsu & Sawada, 2007). It is now clear that excessive ROS that results in oxidative stress is due to endogenous imbalance between the level of oxidants and antioxidants. In brain, antioxidants, such as superoxide dismutase (SOD), glutathione peroxidase (GPx) and catalase (CAT), scavenge surplus ROS and prevent brain cells from oxidative injury (Nagatsu & Sawada, 2007). Dietary antioxidants also play a role in the inactivation of endogenous antioxidant defence system in order to prevent and/or delay the age-related neurodegenerative