

Bioactive compounds extracted from *Ecklonia cava* by using enzymatic hydrolysis protects high glucose-induced damage in INS-1 pancreatic β -cells.

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Abstract

Pancreatic β -cells are very sensitive to oxidative stress and this might play an important role in β -cell death in diabetes. In the present study, we investigated whether the brown alga *Ecklonia cava* has protective effects against high glucose-induced damage in INS-1 pancreatic β -cells. For that purpose, we prepared an enzymatic hydrolysate from *E. cava* (EHE) by using the carbohydrase, Celluclast. High-glucose (30 mM) treatment induced glucotoxicity, whereas EHE prevented cells from high glucose-induced damage then restoring cell viability was significantly increased. Furthermore, lipid peroxidation, intracellular reactive oxygen species (ROS) and nitric oxide (NO) were overproduced as the result of the treatment by high glucose; however, these lipid peroxidation, ROS and NO generations were effectively inhibited by addition of EHE in a dose-dependent manner. Moreover, EHE treatment increased activities of antioxidant enzymes including catalase (CAT), superoxide dismutase (SOD), and glutathione peroxidase (GSH-px) in high glucose pretreated INS-1 pancreatic β -cells. EHE slightly reduced the expression of pro-apoptotic protein Bax induced by high glucose but increased the expression of Bcl-2, an anti-apoptotic protein. These findings indicate that EHE might be used as potential nutraceutical agent which will protect the glucotoxicity caused by hyperglycemia-induced oxidative stress associated with diabetes.