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## **Protection Effect of Cyanidin**

# **3-O-Glucoside Against Oxidative Stress-induced HepG2 Cell Death Through Activation of Akt and Extracellular Signal-regulated Kinase Pathways**

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## Abstract

The attack of reactive oxygen species (ROS) on unsaturated fatty acids causes peroxidation. The membranes of liver cells are rich in unsaturated fatty acids, which are a target of lipid peroxidation by free radicals. The purpose of the present study was to identify the hepatoprotective potential of *Lonicera caerulea* and its active compound, cyanidin 3-O-glucoside, on tertiary butyl hydroperoxide (tBHP)-induced oxidative damage in HepG2 cells. *L. caerulea* extract and its active compound, cyanidin 3-O-glucoside, showed a dose-dependent hepatoprotective effect on tBHP-induced HepG2 cell damage. In addition, *L. caerulea* and cyanidin 3-O-glucoside inhibited the production of intracellular ROS in tBHP-treated HepG2 cells. The protein expression of phosphorylated extracellular signal-regulated kinases and Akt, which are responsible for cell protection against ROS, were increased after treatment with cyanidin 3-O-glucoside (50 and 100  $\mu$ M) in a dose-dependent manner. Therefore, cyanidin 3-O-glucoside is the active compound in *L. caerulea*, which is responsible for the hepatoprotective effects through

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the inhibition of ROS and the activation of antioxidant mechanisms.

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