Effects of the Streptomyces Metabolites, Homogentisic Acid and β-Phenylpyruvic Acid Against Oxidative Stress

中文摘要

英文摘要

Free radicals and reactive oxygen species (ROS), generated in most biologicals vstems by metabolic reactions, ultraviolet rays or radioirradiation, have been implicated as the causative factors in cell injury, aging processes and the p athogenesis of numerous diseases. Althought such kinds of oxidative damages ca n be prevented or reduced by the endogenous or exogenous antioxidant defending mechanisms of oxidation, it still have a need to obtain sufficient quantity o f antioxidants through daily diet to prevent the deleterious effects exerted b y free radicals and ROS. To meet this requirements, it is important to search for more potent and reliable antioxidants from environmental sources. In the pr evious study, we have isolated two Streptomyces metabolities, homogentisic aci d (HA) and β -phenylpyruvic acid (β -PPA), which have been proved to posscess inhibitory activity against lipid peroxidation. In contiuning our work to furt her understand the detailed mechanism and the antioxidant performance of these two microbial metabolites, we have established several in vitro evaluation sy stems for the investigation of antioxidant activities of the two compounds. Th ey include: (1) human erythrocyte ghost membrane system, (2) rat liver microso me system, (3) rat brain homogenate system, (4) use of intact erythrocyte as t he model for detecting antioxidant activity against lipid peroxidation by the method of flowcytometry, (5) use of rat primary hepatocyte as the model for de tecting antioxidant activity against oxidative stress. In the results, HA and β -PPA showed antioxidant effects against (1) peroxyl radical-induced lipid pe roxidation both in erythrocyte ghost membrane and rat liver microsome; (2) iro n-induced lipid peroxidation in rat brain homogenate, (3) lipid peroxidation i n intact human erythrocyte measured by flow cytometry. Nevertheless, HA and β -PPA did not show any effects in rat primary hepatocyte against oxidant-induce d oxidative stress.