Regulation of cellular aging in rat embryonic fibroblast cells using gallic acid

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Abstract

Introduction: Aging is a process characterized by an irreversible growth arrest in somatic cells which caused reactive oxygen species (ROS) products, lipid peroxidation, and DNA and proteins damage. The specific objective of this study was to assess a well-known natural antioxidant compound, Gallic acid (GA) for its anti-aging potential, and evaluate the mechanisms involved in attenuating H_2O_2 induced cellular senescence in rat embryonic fibroblast (REF) cells.

Methods and Results: To begin this process, REF cells were pre-incubated with GA for 24 hours, subsequently were exposed to hydrogen peroxide (H_2O_2) for 2 hours. After the incubation time, cell viability, ROS level as well as senescence-associated (beta)-galactosidase (SA-β-GAL) activity, mitochondrial complex I, II and IV enzyme activities, and cell cycle distribution via flow cytometry were investigated. GA declined the cytotoxic effects of H_2O_2 in REF cells. Analysis of cell cycle showed in REF cells treated by GA the percentage of G0/G1 arrest was diminished compared to the H_2O_2 group. Additionally, GA potently decreased the levels of ROS as well as mitochondrial complex activities. Furthermore, qualitative and quantitative investigation of SA-β-GAL activity demonstrated GA can also decrease cellular senescence.

Conclusions: The findings of this study offer some important insights into the protective effect of GA on controlling cellular senescence and aging process. The results presented support to these hypotheses that GA diminish the oxidative stress of REF cells in cellular senescence. Moreover, incorporation of GA as a protective antioxidant agent works by attenuating the ROS, subsiding mitochondrial complex activities, and affecting cell division.

Keywords: Aging, Gallic acid, Mitochondrial complex, Oxidative stress, Cell cycle