Keywords: Aequorin, Site Directed Mutagenesis, Luminescence Properties, Double Mutants.

Abstract No.258

Protective Effects of 6 Genotypes of Walnuts Against free Radical-Mediated Protein Oxidation

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The role of oxidative protein damages in the pathophysiology of human diseases is currently a topic of considerable interest as oxidized proteins has been implicated in a wide spectrum of clinical disorders. In this study, the antioxidant activity of 6 genotypes of walnuts, were investigated employing various established in vitro systems including ferric reducing ability (FRAP), 1,1-diphenyl-2-picrylhydrazyl (DPPH), and inhibitory effect on protein oxidation as well as the inhibition of Fe2+/ascorbate induced lipid peroxidation in human plama samples. Total phenolic content (TPC) and total flavonoid content (TFC) of the samples were also determined by a colorimetric method. The addition of Fe2+/ascorbate to the plasma samples significantly increased the of protein oxidation by loss of protein-bound sulphydryl (P-SH) groups and increased lipid peroxidation (LPO) The plant extracts showed inhibitory effects against P-SH oxidation, and LPO to varying degrees. Based on this study, the protective effects of walnuts extract could be due to its TPC. In that respect, free radical induced protein oxidation was suppressed significantly by the addition of walnut over a range of concentration. These results clearly demonstrated that in the shells of walnut have higher antioxidant activities than the hulls of walnut. KH501, KH403, KH509 genotype with the highest phenolic content in its shells has more antioxidant activity against protein oxidation.

Keywords: Protein Oxidation, Juglans Regia, Antioxidant Capacity, Lipid Peroxidation.

Abstract No.259

The Fibrillation Study of β-Lactoglobulin Upon Incubation with Aflatoxin M1

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Aflatoxin M1(AFM1) appears in milk as a direct result of the ingestion of food contaminated with aflatoxin B1 by cattle. The role of milk in human nutrition is well-known. The formation of AFM1 occurs in liver and it is secreted into the milk, which is cytotoxic and genotoxic. AFM1 is bound to milk proteins. As a result of the binding affinity of AFM1 for milk proteins, the toxin is distributed unevenly between whey and curd. The purpose of this report is to study the effect of AFM1 on β-lactoglobulin (β-Lg) fibrillation. Regards to this proposal that AFM1 enters to whey proteins (especially, β-Lg), supposed it would interact with this protein and affects on β-Lg fibrillation. β-Lg solution with concentrations of 1(W/V%) at pH 2 was prepared and interacted with different concentration of AFM1. After heating of the solutions at 85 °C for 24 h, the fibrillation of them were investigated. Strange results showed that AFM1 reduces the intensity of fluorescence of fibrils. By increasing the concentration of AFM1, the intensity of fluorescence of fibrils was decreased. This means AFM1 as a toxin reduces the fibrillation of β-lactoglobulin.

Keywords: Aflatoxin M1, β-Lactoglobulin, Fibrillation, Milk proteins.

Abstract No.260

Fibrillar Protein Aggregation May be Detrimental Via Different Oxidative Routes: Relevance to the Etiology of Amyloid-Related Neurodegenerative Disorders Using the Experimental-Based Evidences

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The exact mechanism of cell death in neurodegenerative diseases remains obscure, but the aberrant assembly of proteins into fibrillar