Redox Cycle of Caffeic Acid with Endogenous Ascorbic Acid and α-Tocopherol Influences Lipid Oxidation in Tissues

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Lately the redox chemistry has demonstrated to be a key factor to elucidate protective or hazardous effects of phenolics on lipid oxidation occurring in cellular membranes and tissues. The present investigation was focused on the molecular mechanism governing the capacity of caffeic acid (Caf-OH) to prevent lipid oxidation in muscle tissues by exploring the redox cycles of Caf-OH with important endogenous reductants of tissues such as α-TOH and $AscH^{-}$. Fish muscle was the model adopted due to its elevated content in polyunsaturated fatty acids (PUFA) and a notable vulnerability to undergo lipid oxidation. The interaction of Caf-OH with α-TOH was firstly investigated in oil-in-water emulsions, a simple model that has shown similar behavior to muscle tissues in different studies conceived to evaluate prevention of lipid oxidation. In this medium, Caf-OH was very active in retarding the progress of lipid oxidation, whereas the presence of Caf-OH did not affect significantly the kinetic for α-TOH decay. On the contrary, the supplementation of muscle tissue with Caf-OH was found to reduce both degradation of endogenous α-TOH and propagation of lipid oxidation. A high capacity of Caf-OH to regenerate α-TOH through reduction of the α-tocopheroxyl radicals was revealed by Electron Spin Resonance (ESR) Spectroscopy. Simultaneously, higher concentrations of Caf-OH in muscle tissue were found to accelerate significantly the loss of endogenous ascorbate ($AscH^{-}$), suggesting an active electron donation from $AscH^{-}$ to Caf-OH. The data reveal that Caf-OH is able to protect actively the endogenous α-TOH of muscle tissues, whereas endogenous $AscH^{-}$ contributes to repair Caf-OH. This redox cycle contributes ultimately to protect the endogenous α-TOH, considered last barrier against lipid oxidation in cellular membranes. Such protection results in a significant resistance of muscle tissues to suffer lipid oxidation.