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Trace elements and aging

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Abstract:
Functionally altered proteins are reported to be present in various organs of aged animals. The accumulation of such proteins is implicated in the mechanism of age-associated deterioration of the physiological functions of tissues. However, the mechanisms of the generation of altered proteins are not fully understood.
Protein carbonylation has been regarded as a marker of oxidative damage to proteins and an increase in the amount of such oxidized protein has been reported in many experimental aging models. It has been suggested or shown that protein carbonyls drive from the direct oxidation of side chains of amino acid residues or the reaction of lipid peroxidation products with amino acid residues or non-enzymatic glycation phenomena. Interestingly, the rates of these chemical modifications of proteins are often enhanced in the presence of metal ions such as iron and copper. Because age-related accumulation of iron but not copper appears to be a common phenomenon in different tissues of various animals, including humans, iron may acts as a major catalyst which promotes direct or indirect protein carbonylation in vivo during aging. Furthermore, it has been shown that decreased iron stores and low iron diet extend the life span of animals. These findings suggest that iron withholding or removal can possibly prevent age-related dysfunctions and certain diseases caused by iron-mediated oxidative damage.

Key words: aging, longevity, iron, oxidation, protein carbonyl

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