Asparagus Racemosus Supplementation Ameliorate Age-related Oxidative Damage in Skeletal Muscle Lysosome of Aged Rats

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ABSTRACT: Aging is the accumulation of diverse deleterious changes in the cells and tissues leading to increased risk of diseases. Oxidative stress is considered as a major risk factor and contributes to age-related increase in oxidative damage during aging. Age associated changes occur in many cellular organelles. Changes in the lysosomes of senescent tissues and organisms are common and have been used as biomarkers of aging. Cellular aging represents slowly developing functional decline of lysosomes compartment and secondary to oxidant-induced damage and lipofuscin accumulation. Lipofuscin consider as a hallmark of aging and their deposition ultimately increases the oxidative damage. In the present study, we have evaluated the salubrious role of asparagus racemosus root extract (ARRE) on accumulation of oxidative damage products such Malondialdehyde (MDA), Protein carbonyls (PCO), lysosomal marker enzymes acid phosphatase and Cathepsin D activity, aging marker lipofuscin and membrane bound H⁺ATPase activity in skeletal muscle lysosome of aged rats. Our results, thus, revealed that ARRE has ameliorating effect on the accumulation of age-related oxidative damages and restored the enzyme activity and decreased the lipofuscin content in skeletal muscle lysosomes. This ameliorating activity of ARRE mainly attributed to the presence of enriched therapeutic phytochemical constituents, which act synergistically to alleviate the indices of oxidative stress, is associated with aging.

KEY WORDS: Asparagus racemosus root extract, Lysosome, Aging, Lipofuscin, Malondialdehyde, Protein carbonyl.

Introduction

Aging is characterized by slow, progressive, structural and functional changes that take place at cellular, tissue and organ level. These changes resulting in gradual functional decline, decreased adaptability and ability to face stress and increased probability of age-associated diseases including cardio vascular disease, cancer, diabetic, Alzheimer’s etc. Age associated changes occur in many cellular organelles including lysosomes and mitochondria. Changes in the lysosomes of senescent tissues and organisms are common and have been used as biomarkers of aging. Lysosomes of skeletal muscle show the most remarkable age-related changes because they are non-proliferative and play a major role in cellular aging and death (Cuervo and Dice, 2000). Cellular aging represents slowly developing functional decline of lysosomes compartment and secondary to oxidant-induced damage and lipofuscin accumulation (Terman et al., 2006).

Lysosomes are membrane bound vesicles involved in intracellular digestion. They contain a variety of hydrolytic enzymes that are optimally active at an acidic pH (Holtzman, 1989; Kornfeld and Mellman, 1989; Futai et al., 1998). These hydrolases require an acidic environment for activity and become inactivated at a neutral pH. The intralysosomal environment is maintained at (pH) 4.5 by membrane integrated H⁺ ATPase. (Dell’Angelica, 2000). Lysosomal enzymes degrade not only cytosolic biomolecule like protein, carbohydrate, lipid and nucleic acid but also whole organelles including mitochondria, endoplasmic reticulum, ribosome, peroxisome and proteosomes (Cuervo, 2004; Levine and Klipnsky, 2004). The lysosomal compartment has multiple functions and it may be indicator of adaptive abilities. It is known that lysosomes are especially sensitive to oxidative stress (Li et al., 1998). It was investigated that hydroxyl radicals destabilize lysosomal membranes and thereby cause leakage of lysosomal enzymes to the cytosol with ensuing cellular degeneration or even death (Hellquist et al., 1997). Normally damaged macromolecules and organelles are efficiently degraded in the lysosome, resulting in the successful recycling (Brunk and Terman, 2002b).

As age advances, lysosomal degradation capacity decreases that contribute to increased accumulation of incompletely degraded intralysosomal waste material as lipofuscin (age pigment) which starts to accumulate in postmitotic cells from early life and then gradually increases with age advancing (Terman et al., 2006). Lipofuscin consider as a hallmark of aging and their deposition ultimately decreases cellular adaptability and promotes the development of age-related pathologies, including neuro degenerative diseases, heart failure and macular degeneration (Terman and Brunk, 2004).

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