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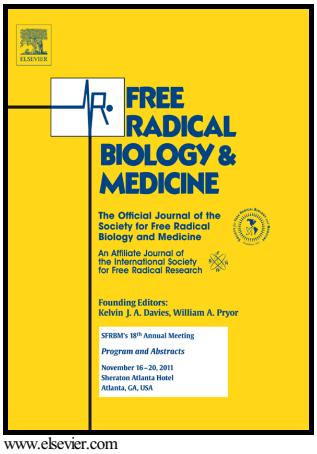

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Beneficial and Paradoxical Roles of Selenium at Nutritional Levels of Intake in Healthspan and Longevity

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Abstract

Accumulation of genome and macromolecule damage is a hallmark of aging, age-associated degeneration, and genome instability syndromes. Although processes of aging are irreversible, they can be modulated by genome maintenance pathways and environmental factors such as diet. Selenium (Se) confers its physiological functions mainly through selenoproteins, but Se compounds and other proteins that incorporate Se nonspecifically also impact optimal health. Bruce Ames proposed that the aging process could be mitigated by a subset of low-hierarchy selenoproteins whose levels are preferentially reduced in response to Se deficiency. Consistent with this notion, results from two selenotranscriptomic studies collectively implicate three low-hierarchy selenoproteins in age or senescence. Experimental evidence generally supports beneficial roles of selenoproteins in the protection against damage accumulation and redox imbalance, but some selenoproteins have also been reported to unexpectedly display harmful functions under sporadic conditions. While longevity and healthspan are usually thought to be projected in parallel, emerging evidence suggests a trade-off between

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