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Prevention of mutation, cancer, and other age-associated diseases by optimizing micronutrient intake.

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Abstract

I review three of our research efforts which suggest that optimizing micronutrient intake will in turn optimize metabolism, resulting in decreased DNA damage and less cancer as well as other degenerative diseases of aging. (1) Research on delay of the mitochondrial decay of aging, including release of mutagenic oxidants, by supplementing rats with lipoic acid and acetyl carnitine. (2) The triage theory, which posits that modest micronutrient deficiencies (common in much of the population) accelerate molecular aging, including DNA damage, mitochondrial decay, and supportive evidence for the theory, including an in-depth analysis of vitamin K that suggests the importance of achieving optimal micronutrient intake for longevity. (3) The finding that decreased enzyme binding constants (increased Km) for coenzymes (or substrates) can result from protein deformation and loss of function due to an age-related decline in membrane fluidity, or to polymorphisms or mutation. The loss of enzyme function can be compensated by a high dietary intake of any of the B vitamins, which increases the level of the vitamin-derived coenzyme. This dietary remediation illustrates the importance of understanding the effects of age and polymorphisms on optimal micronutrient requirements. Optimizing micronutrient intake could have a major effect on the prevention of cancer and other degenerative diseases of aging.

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