In the journal Circulation, Ulrich Laufs, MD and his colleagues report an association between long-term intense exercise and a reduction in the shortening of telomeres that occurs with aging.*

The researchers assessed telomere length in blood samples from professional runners whose age averaged 20, middle-aged athletes who had engaged in endurance exercise since youth, and young and old groups of untrained athletes who did not engage in regular exercise. Age-dependent telomere loss was found to be lower in the middle aged athletes who had engaged in endurance exercise for several decades compared to the older, untrained men. “The most significant finding of this study is that physical exercise of the professional athletes leads to activation of the important enzyme telomerase and stabilizes the telomere,” noted Dr. Laufs. “This is direct evidence of an anti-aging effect of physical exercise. Physical exercise could prevent the aging of the cardiovascular system, reflecting this molecular principle.”

Editor’s note: Several factors have been associated with reduced telomere shortening, including multivitamin supplementation and other lifestyle improvements.

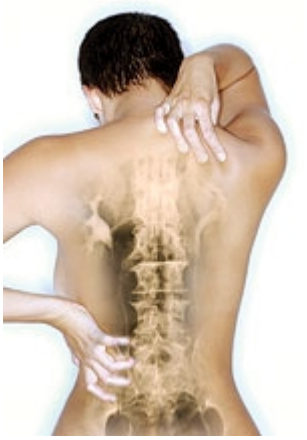
—Dayna Dye

Reference

* Circulation. 2009 Dec 15;120(4).

Zinc Plays Role in the Prevention of Osteoporosis

The Department of Medicine at Emory University School of Medicine recently studied zinc’s role as an essential nutritional component in the development of humans and animals.* Researchers noticed several important factors relating zinc content in bones to the strength of bones. In particular, increased bone zinc content has been shown to decrease bone aging, skeletal unloading, and postmenopausal conditions, suggesting its role in bone health. Zinc has also demonstrated a stimulatory effect on osteoblastic bone formation and mineralization.



Researchers also noted that the oral administration of beta-alanyl-L-histidinato zinc (AHZ) or zinc acexamate has a restorative effect on bone loss under various pathophysiologic conditions including aging, aluminum bone toxicity, calcium and vitamin D deficiency, adjuvant arthritis, estrogen deficiency, diabetes, and fracture healing. The study concluded that zinc compounds may be designed as a new supplementation factor in the prevention and therapy of osteoporosis.

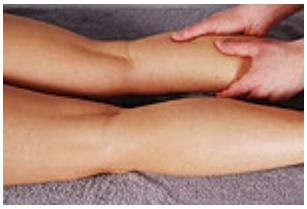
—Jon Finkel

Reference

* Mol Cell Biochem. 2009 Dec 25.

Magnesium Deficiency May Be Linked to Restless Leg Syndrome

In a study from the Romanian *Journal of Neurology and Psychiatry,* researchers conducted biochemical and neurological tests in 10 cases of restless leg syndrome. The investigators reported important disorders of sleep organization. They found agitated sleep with frequent periods of nocturnal awakenings, and a decrease of the duration and percentage of the deeper rapid eye movement (REM) sleep—also found in other forms of insomnia caused by magnesium deficiency. [1]



According to the U.S. Dept. of Agriculture’s Human Nutrition Research Center in North Dakota, “Magnesium plays a key role in the body’s chemistry that regulates sleep. This may be why persons with long-term lack of sleep, or abnormal brain waves during deep sleep, often have low magnesium in their blood….Magnesium treatment increased deep sleep and improved brain waves during sleep in 12 elderly subjects. Magnesium treatment also decreased time to fall asleep and improved sleep quality of 11 alcoholic patients who often have a low magnesium status.”[2]

—Jon Finkel

Reference

1. Rom J Neurol Psychiatry.1993 Jan-Mar; 31(1):55-61.
2. www.ars.usda.gov.

Coenzyme Q10 and Creatine Combination Produces Additive Neuroprotective Effects in Models of Parkinson's and Huntington's Diseases

Researchers from the Department of Neurology and Neuroscience at the Weill Medical College of Cornell University have discovered that coenzyme Q10 (CoQ10) and creatine are promising agents for neuroprotection in neurodegenerative diseases via their effects on improving mitochondrial function and cellular bioenergetics and their properties as antioxidants.*

The researchers examined whether a combination of CoQ10 with creatine can exert additive neuroprotective effects in a mouse model of Parkinson's disease, a rat model of Huntington's disease (HD), and a transgenic mouse model of HD. The combination of the two agents produced additive neuroprotective effects against dopamine depletion in the striatum and loss of tyrosine hydroxylase neurons in the substantia nigra following chronic administration of a neurotoxic agent. These findings suggest that combination therapy using CoQ10 and creatine may be useful in the treatment of neurodegenerative diseases such as Parkinson's disease and HD.

—Jon Finkel

Reference

* J Neurochem. 2009 Jun;109(5):1427-39.