more effective and normalize vascular function in individuals with coronary artery disease and associated risk factors, including hypercholesterolemia, hyperhomocysteinemia, hypertension, diabetes and smoking.

PROCEEDINGS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE, 1999, Vol 222, Iss 3, pp 196-204

Protective effect of aminoguanidine on brain damage

Nitric oxide (NO) produced by inducible NO synthase contributes to ischemic brain damage. However, the role of inducible NO synthase-derived NO on neonatal hypoxic-ischemic encephalopathy has not been clarified. We demonstrate here that aminoguanidine, a relatively selective inhibitor of inducible NO synthase, ameliorated neonatal hypoxic-ischemic brain damage and that temporal profiles of NO correlated with the neuroprotective effect of aminoguanidine. Seven-day-old rat pups were subjected to left carotid artery occlusion followed by 2.5 h of hypoxic exposure (8% oxygen). Infarct volumes (cortical and striatal) were assessed 72 h after the onset of hypoxia-ischemia by planimetric analysis of coronal brain slices stained with hematoxylin-eosin. Aminoguanidine (300 mg/kg i.p.) administered once before the onset of hypoxia-ischemia and then three times daily, significantly ameliorated infarct volume (89% reduction in the cerebral cortex and 90% in the striatum). NO metabolites were measured by means of chemiluminescence using an NO analyzer. In controls, there was a significant biphasic increase in NO metabolites in the ligated side at 1 hour (during hypoxia) and at 72 h after the onset of hypoxia (p < 0.05). Aminoguanidine did not suppress the first peak but significantly reduced the second one (p < 0.05), and markedly reduced infarct size in a neonatal ischemic rat model. Suppression of NO production after reperfusion is a likely mechanism of this neuroprotection.

PEDIATRIC RESEARCH, 2000, Vol 47, Iss 1, pp 79-83

Preventing hip fracture

Hip fractures are recognized as a major public health problem worldwide. Demographic changes will lead to enormous increases in the number of hip fractures and projections indicate that the number of hip fractures occurring worldwide each year will rise from 1.26 million in 1990 to 4.5 million by 2050. However, preventive strategies are available. Supplementation with calcium and vitamin D restores bone quality by suppressing excessive activity of the parathyroid glands and decreases the risk of falling by improving neuromuscular coordination. As a result, hip fracture risk is reduced by 43% in the vitamin D-insufficient elderly. Treatment with the bisphosphonate alendronate increases bone strength and results in a 51% reduction of hip fracture risk. Also, hip protectors absorb energy during a fall and reduce hip fracture risk by 56%. Combining these procedures could prevent a large proportion of hip fractures in the future.

TRENDS IN ENDOCRINOLOGY AND METABOLISM, 1999, Vol 10, Iss 10, pp 417-420

Antioxidants/caloric restriction protects against liver cancer

A study looked at the role of oxidative stress and oxidative damage in the induction of cancer by nongenotoxic carcinogens (Genotoxic = may cause mutation or cancer by damaging DNA). Liver carcinogenic compounds such as chlorinated hydrocarbons appeared to induce oxidative stress in the liver. This oxidative stress and oxidative damage in turn may be responsible for the tumor-promoting activity of these compounds. Reduction of oxidative damage by antioxidants, or dietary-restriction, results in a lessening of the selective cell growth by these carcinogens.

The free radical stress induced by non-genotoxic agents may influence cell proliferation and/or apoptosis (programmed cell death) in the pre-cancerous cells. A study with nongenotoxic liver carcinogens showed a dose-dependent increase in oxidative stress and an increase in liver local lesion growth. The use of antioxidant dietary supplementation or caloric restriction prevented the lesion growth. This restriction appeared to be through an increase in apoptosis (cell death) in the liver lesions.


Effect of L-carnitine on glycoprotein status

The effect of L-carnitine on glycoprotein status was studied in young and aged rats. The levels of protein, hexose, hexosamine, sialic acid and fucos were low in aged rats. Supplementation of L-carnitine (300 mg/kg body weight/day) for 7, 14 and 21 days demonstrated a normalization of glycoprotein status. There was no such significant effect upon carnitine administration to young rats. It was concluded that L-carnitine is effective in normalizing the age-associated alterations in glycoproteins and can minimize the age-associated disorders in which free radicals are the major cause.


Cardiovascular disease risk factors and menopausal status

A study examined cardiovascular risk factors in 93 pre- and 93 postmenopausal women age 43-55. Postmenopausal women who were at least 3 years after menopause or whose menses had stopped naturally before age 48 were age-matched with premenopausal women with regular menses and without menopausal complaints. Compared to premenopausal women,