Curcumin protects against lung injury

An early feature of paraquat (PQ) toxicity (a weedkiller that produces delayed toxic effects on the lungs when ingested) is the inflow of inflammatory cells, releasing proteolytic enzymes and free radicals, which can destroy lung tissue and result in pulmonary fibrosis (chronic inflammation and formation of fibrous tissue). Therefore, suppressing early lung injury is an appropriate therapy of pulmonary damage before the development of irreversible fibrosis. Curcumin confers remarkable protection against PQ lung injury. In a study, 50 mg/kg of PQ resulted in a significant rise in the levels of certain proteins and enzymes, thiobarbituric acid reactive substances (TBARS), and neutrophils in the fluid of the bronchioles and alveoli in the lungs (bronchoalveolar lavage fluid (BALF)), while decreasing glutathione levels. In addition, the data also demonstrated that PQ caused a decrease in angiotension converting enzyme (ACE), glutathione levels and an increase in levels of TEARS and myeloperoxidase (MPO) (leads to impaired bacterial killing) activity in the lung. Interestingly, curcumin prevented the general toxicity and mortality induced by PQ and blocked the rise in proteins and enzymes such as BALF protein, ACE and neutrophils. Similarly, curcumin prevented the rise in TEARS content in both BAL cell and lung tissue and MPO activity of the lung. In addition, curcumin treatment abolished PQ induced reduction in lung ACE and BAL cell, and lung glutathione levels. Thus, curcumin has important therapeutic implications in facilitating the early suppression of PQ lung injury.

LIFE SCIENCES, 2000, Vol 66, Iss 2, pp PL21-PL28

Role of vitamin C in atherogenesis and vascular dysfunction

Free radicals have been implicated as an important causative factor in atherosclerosis and vascular dysfunction. Antioxidants can inhibit the development of atherosclerosis and improve vascular function by two different mechanisms. 1) Lipid-soluble antioxidants present in low-density lipoprotein (LDL), including vitamin E and water-soluble antioxidants present in the extracellular fluid of the arterial wall, including vitamin C, inhibit LDL oxidation through an LDL-specific antioxidant action. 2) Antioxidants present in the cells of the vascular wall decrease cellular production and the release of free radicals, inhibit endothelial activation (i.e., activity of molecules that adhere to one another), and improve the biologic activity of endothelium-derived nitric oxide (EDNO) through a cell- or tissue-specific antioxidant action. Vitamin E and a number of thiol antioxidants have been shown to decrease the adhesion of molecules. Vitamin C has been demonstrated to make EDNO activity