Molecular mechanisms of cigarette smoke – induced myocardial infarction and its prevention

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Myocardial injury, including myocardial infarction, was produced in cigarette smoke-exposed marginal vitamin C-deficient guinea pigs (450-500g body weight) as evidenced by release of cardiac Troponin T and I in the serum, oxidative stress, inflammation, apoptosis, thrombosis and collagen deposition in the myocardium. Treatment of rat cardiomyocyte cells (H9c2) *in vitro* and guinea pigs *in vivo* with p-benzoquinone in amounts derived from cigarette smoke with and without antibody to p-benzoquinone (*in vitro*) revealed that p-benzoquinone was a major factor responsible for cigarette smoke-induced myocardial damage. Population based studies indicated that plasma vitamin C levels of smokers without disease were significantly lower (p=0,0000) than that of non-smokers. Vitamin C levels of cigarette smoke-related cardiovascular patients were further lower (p= 0.0000) than that of smokers without disease. A moderately large dose of vitamin C (15 mg/day) prevented cigarette smoke/p-benzoqunone-induced myocardial injury. This dose in the guinea pig is approximately 2g vitamin C/adult/day.

Biotechnological application

Serum level of p-benzoquinone in chronic smokers may be used as marker for identification of high risk smokers. Dietary supplementation of vitamin C may be a novel and simple therapy for the prevention of pathological cardiovascular events in the smokers.

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