**THE EFFECTS OF RISK FACTORS ON ENDOTHELIAL PROGENITOR CELLS IN CARDIOVASCULAR DISEASES**

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Atherosclerosis is initiated by endothelial damage. Neighboring healthy endothelial cells can migrate and proliferate to restore the injured endothelium. The injured endothelial monolayer may also be regenerated partly by circulating bone marrow-derived endothelial progenitor cells (EPCs). These circulating EPCs are mobilized endogenously triggered by tissue ischemia or exogenously by cytokine stimulation. Clinical studies demonstrated that levels of circulating EPCs are associated with vascular endothelial function and cardiovascular risk factors. A number of risk factors, such as aging, hypertension, diabetes mellitus, and hypercholesterolemia have been shown to exert detrimental effects on EPC number and function. Our studies demonstrated increased endothelial apoptotic microparticles and decreased circulating EPC levels and function in hypertensive patients with microalbuminuria and left ventricular hypertrophy. Reduced levels of circulating EPCs independently predict atherosclerotic disease progression and development of cardiovascular events. Animal and clinical studies of cell therapy have shown that transplantation of autologous EPCs or other cellular pools enriched with vascular progenitors is feasible in both coronary and peripheral atherosclerotic diseases. Our studies have shown that high glucose impairs EPCs by modifying nitric oxide-related but not oxidative stress-mediated mechanisms. MMP-9 is essential for EPCs in ischemia-induced angiogenesis. There are several ways to increase levels of circulating EPCs and improve their function by pharmacological strategies and lifestyle modification. In a series of study, we found that statins, red wine intake, adiponectin, far infrared may increase the number and function of EPCs.