**OXIDATIVE STRESS AND ENDOTHELIAL DYSFUNCTION**

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Excessive production of reactive oxygen species (ROS) and/or insufficient activity of antioxidant defense mechanisms may result in oxidative stress, which could cause the damage to DNA, lipids, proteins, and carbohydrates, and abnormal gene expression, thereby contributing to cardiovascular disease and many other inflammatory and chronic diseases. Recently, we have demonstrated that several new cardiovascular risk factors, such as antiretroviral therapy drugs, adipokines, soluble CD40L, uric acid, nitrotyrosine and chlorotyrosine significantly increased oxidative stress of endothelial cells and impaired endothelial nitric oxide synthase (eNOS) system. This impairment is involved in the reduced activity and expression of eNOS, decreased sensitivity to nitric oxide (NO) or increased degradation of NO by reaction with superoxide. Antioxidants reduce oxidative stress; therefore, they may prevent or reduce the risk of oxidative stress-related endothelial dysfunction and cardiovascular disease. Recently, we have investigated new antioxidant functions and molecular mechanisms of ginsenoside Rb1, entacapone, and dihydroxy-nitrobenzaldehyde (DHNB) in the vascular system. These studies may provide a significant rationale for using new antioxidants in combination therapy with existing antioxidants such as vitamin C and vitamin E for cardiovascular disease.