**CARDIOVASCULAR RISK FACTORS: AN UPDATE**

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Cardiovascular disease (CVD) still remains the number one cause of death in the United States. Hyperuricemia has long been established as the major etiologic factor in gout. Only in the last few years, several large clinical studies have confirmed that hyperuricemia is a significant and independent risk factor for CVD after an extensive adjustment for almost all of the possible confounding conditions. High levels of serum uric acid (SUA) directly impair the cardiovascular system in a concentration-dependent matter; and xanthine oxidoreductase (XOR)-induced oxidative stress also significantly contributes to the development of CVD. Accordingly, urate-lowering drugs such as XOR-inhibitors could play an important role in the prevention and treatment of CVD. However, due to their potential adverse effects, clinically available XOR-inhibitor drugs, Allopurinol and Febuxostat, are not indicated for clinical management of hyperuricemia-related CVD. Currently, the development of newer agents with differing pharmacological mechanisms and less toxicity is an active field of research. We have recently found a natural substance derivative, 3,4-dihydroxy-5-nitrobenzaldehyde (DHNB), which has a strong XOR-inhibitory effect in a cell-free system and in mouse models. DHNB displays potent mixed-type inhibition of XOR and shows an additive effect with Allopurinol at low concentrations. In addition, DHNB directly scavenged reactive oxygen species (ROS). DHNB has a different chemical structure from the current clinical XOR-inhibitor drugs, and shows much less toxicity in mouse models as compared with Allopurinol. More preclinical studies and clinical trials are warranted to accelerate the clinical application of DHNB as a new, safer, and effective XOR-inhibitor drug for the treatment of gout and a symptomatic hyperuricemia, especially in its long-term use for the prevention of hyperuricemia-induced CVD.