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Atherosclerosis: Molecular Mechanisms and Therapeutic Advances

Guest Editors:

Prof. Dr. Maria Luisa Balestrieri
Department of Biochemistry, Biophysics and General Pathology, School of Medicine, University of Campania "Luigi Vanvitelli", 80138 Naples, Italy

Prof. Dr. Nunzia D'Onofrio
Department of Precision Medicine, University of Campania Luigi Vanvitelli, Via L. de Crecchio 7, 80138 Naples, Italy

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Message from the Guest Editors

The development of atherosclerosis, a complex multifactorial process, is, at least in part, controlled by the functional state of the vascular endothelium, influenced by a broad set of cardiovascular risk factors. Hypercholesterolemia, hypertension, and diabetes mellitus enhance reactive oxygen species generation, resulting in oxidative modification of lipoproteins and phospholipids, all mechanisms that contribute to atherogenesis. A pivotal role for inflammation in the pathogenesis of atherosclerosis has been recognized and proved at molecular levels. However, despite current knowledge, results coming from genome wide association studies are expected to uncover the complex inflammatory process subtending atherosclerosis, thus opening a new scenario for tailored target therapy. Moreover, several pieces of evidence point towards the crosstalk between long non-coding RNAs and vasculature in regulating the development of vessel lining and recruitment of immune cells, such as macrophages, at the site of injury and inflammation. In particular, the modulation of atherosclerosis by long non-coding RNAs has brought significant attention over the past few years.