

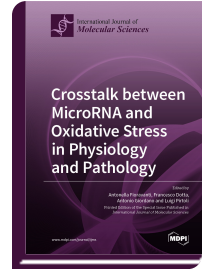


Special Issue Reprint

Crosstalk between MicroRNA and Oxidative Stress in Physiology and Pathology

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Edited by
Antonella Fioravanti
Francesco Dotta
Antonio Giordano
Luigi Pirtoli



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MicroRNAs (miRNAs) are small noncoding RNAs that are 19–24 nucleotides in length, following maturation. Recent evidence has demonstrated their key role as post-transcriptional regulators of gene expression through the binding of specific sequences within target messenger RNA (mRNA). miRNAs are involved in the synthesis of a very large number of proteins, and it is speculated that they could regulate up to 30% of the human genome. They control virtually every cellular process and are essential for animal development, cell differentiation, and homeostasis. Altered miRNA expression has been linked to such pathological events as inflammatory, degenerative, or autoimmune processes and have been associated with several diseases, including cancer, cardiovascular diseases, diabetes mellitus, and rheumatic and neurological disorders. Recently, miRNAs have been found in many different biological fluids, and this observation suggests the potential of miRNAs as new candidate biomarkers for diagnosis, classification, prognosis, and responsiveness in the treatment of different pathological conditions. Furthermore, the development of therapeutic strategies that involve either restoring or repressing miRNAs expression and activity has attracted much attention. Significant progress has been made in the systems for delivery of miRNAs, even if substantial improvements in this area are still necessary. Although they have been extensively studied, a number of interesting questions regarding the physiological and pathological role of miRNAs have been postulated, and their diagnostic and therapeutic role remain yet unanswered. Reactive oxygen species (ROS) are radical-containing oxygen molecules derived from cellular oxidative metabolism, including enzyme activities and mitochondrial respiration, and play a pivotal role in many cellular functions. Whereas ROS are essential for normal cellular processes, their aberrant production, or failure of the capacity to scavenge excessive ROS, induces an altered



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