







Special Issue Reprint

Hypoxia-Inducible Factors: Regulation and Therapeutic Potential

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Oxygen is an essential molecule in the production of adenosine triphosphate (ATP) in cells, and a lack of energy due to O_2 deficiency makes the maintenance of biological functions and human life improbable. Since oxygen functions as the final electron acceptor in the series of ATP synthesis reactions in conjunction with oxidative phosphorylation in mitochondria, its deficiency causes the oxidation of a series of coenzymes, such as nicotinamide and flavin adenine dinucleotide, and the reduction in oxygen molecules to water molecules (H_2O). Mammals do not have a mechanism for biosynthesizing oxygen in their bodies. In higher organisms such as vertebrates, which possess many organs, oxygen in the body is always "scarce,"; therefore, the dominant view is that organisms have evolved mechanisms to respond to the lack of this essential molecule (hypoxia), and actively use it to maintain bodily integrity. Anatomically complex, higher multicellular organisms are equipped with specialized mechanisms to enable all cells to obtain sufficient oxygen. The proper development and preservation of these systems requires the harmonious expression of thousands of genes. The transcription factor responsible for this gene expression is hypoxia-inducible factor 1 (HIF-1).

In this Special Issue, we invited research and review papers on various areas of oxygen biology research that focused on the fundamental understanding of HIF signaling pathways and related gene expression profiling, as well as pharmacogenomic biomarkers, molecular targets driving the regulation of human physiology and pathophysiology, and validation in



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