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Oxidative Phosphorylation System Dysfunction Role and Mechanisms in Cancer and Its Therapies

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Message from the Guest Editors

OXPHOS dysfunction, which can be also caused by mtDNA copy number and expression alterations or by mito-nuclear mismatch, directly impacts ATP and ROS production and can have, among others, effects on metabolic remodeling, on the control of apoptosis or on gene expression through epigenetic modifications. These effects can drive the transformation process or facilitate cancer cell adaptation to its microenvironment, having consequences in all stages of tumorigenesis, including the escape from immune surveillance or response to treatment. Integration of different data types, from high-throughput analysis comparing tumor and non-tumor cells in large patient cohorts to the identification of relevant associations and candidate mutations/variants, to single detailed functional studies of particular mutations in specific cancer types, will be needed to elucidate the involved pathways and mechanisms. This knowledge will help us to design more efficient therapeutic strategies.

The aim of this Special Issue is to present and review advances in the understanding of the molecular mechanisms that explain the multiple influences of OXPHOS dysfunction in the carcinogenesis process.







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Message from the Editor-in-Chief

Cancers is an international online journal addressing both clinical and basic science issues related to cancer research. The journal is publishing in Open Access format, which will certainly evolve to ensure that the journal takes full advantage of the rapidly changing world of information and knowledge dissemination. It publishes high-quality clinical, translational, and basic science research on cancer prevention, initiation, progression, and treatment, as well as other related topics, particularly to capture the most seminal studies in the rapidly growing area of immunology, immunotherapy, and tumor microenvironment.

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