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

Guest Editors:

Dr. Patricio Fernández-Silva
GENOXPHOS Group, Department Biochemistry and Molecular and Cell Biology, Faculty of Sciences, University of Zaragoza, and Biocomputation and Complex Systems Physics Institute (BIFI), Zaragoza, Spain

Dr. Raquel Moreno-Loshuertos
GENOXPHOS Group, Department Biochemistry and Molecular and Cell Biology, Faculty of Sciences, University of Zaragoza, and Biocomputation and Complex Systems Physics Institute (BIFI), Zaragoza, Spain

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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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Editor-in-Chief

Prof. Dr. Samuel C. Mok
Department of Gynecologic Oncology and Reproductive Medicine, The University of Texas MD Anderson Cancer Center, Houston, TX 77030, USA

Message from the Editor-in-Chief

Cancers (ISSN 2072-6694) is an international, online journal addressing both clinical and basic science issues related to cancer research. The journal will continue its 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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Contact Us

Cancers Editorial Office
MDPI, Grosspeteranlage 5
4052 Basel, Switzerland

Tel: +41 61 683 77 34
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cancers@mdpi.com
𝕏@Cancers_MDPI