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Pathogenesis and Treatment of Adrenal Tumors

Guest Editors:

Dr. Anna Angelousi
Department of Endocrinology, First Department of Internal Medicine, Laiko Hospital, National and Kapodistrian University of Athens, 11527 Athens, Greece

Dr. Danae Delivanis
Division of Endocrinology, Diabetes, Metabolism, and Nutrition, Mayo Clinic, Rochester, MN 55905, USA

Deadline for manuscript submissions:
closed (15 May 2022)

Message from the Guest Editors

Dear Colleagues,

Advances in genomics have enormously improved our understanding of adrenal tumorigenesis and led to the development of prognostic markers and therapeutic targets. Bilateral nodular hyperplasias causing Cushing's syndrome are frequently caused by germline alterations leading to cAMP/PKA pathway activation (micronodular) and ARMC5 inactivation (macronodular). Somatic mutations of β-catenin and PRKACA are observed in non-secreting or cortisol producing adenomas, respectively. Alterations of the β-catenin (CTNN1B, ZNFR3) or TP53 pathways are found in carcinomas. Mutations in cancers are more common in aggressive tumors and correlate with transcriptome or methylation profiles. Identification of these alterations helps to refine the molecular classification of these tumors and to develop molecular targeted treatment.

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Editor-in-Chief

Prof. Dr. Felipe Fregni

1. Neuromodulation Center and Center for Clinical Research Learning, Spaulding Rehabilitation Hospital and Massachusetts General Hospital, Harvard Medical School, Boston, MA 02114, USA
2. Department of Epidemiology, Harvard T.H. Chan School of Public Health, Boston, MA 02115, USA

Message from the Editor-in-Chief

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Biomedicines Editorial Office
MDPI, St. Alban-Anlage 66
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