



Pathogenesis and Treatment of Adrenal Tumors 2.0

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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 in the β -catenin (CTNN1B, ZNF3) 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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Message from the Editor-in-Chief

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