

GENETICS OF ENDOCRINE DISEASE

The Molecular Pathogenesis of Hereditary and Sporadic Adrenocortical and Adrenomedullary Tumors

CHRISTIAN A. KOCH, KAREL PACAK, AND GEORGE P. CHROUSOS

Pediatric and Reproductive Endocrinology Branch, National Institute of Child Health and Human Development, National Institutes of Health (C.A.K., K.P., G.P.C.), Bethesda, Maryland 20892; and Department of Medicine III, University of Leipzig (C.A.K.), 04103 Leipzig, Germany

Modern imaging modalities lead to frequent detection of adrenal masses, most of them incidental findings. Although the majority of adrenocortical and adrenomedullary tumors are benign, there are no reliable clinical and laboratory markers to distinguish most of them from malignant neoplasms. The molecular mechanisms underlying the pathogenesis of these tumors have recently begun to be unraveled. A fruitful avenue for the elucidation of tumorigenesis has been the study of adrenal tumors that are manifestations of hereditary or

postzygotic genetic syndromes, because one knows the "first hit", i.e. the primary gene defect. In contrast, in sporadic adrenal tumors the first hit, possibly a somatic mutation of a tumor-related gene, is unknown, and therefore the sequence of genetic alterations is difficult to establish. In this article we review in addition to our own work the literature on molecular aspects of adrenocortical and adrenomedullary tumorigenesis. (*J Clin Endocrinol Metab* 87: 5367-5384, 2002)

ADRENAL TUMORS HAVE been identified in up to 9% of all autopsy cases, with most of them appearing in hypertensive and diabetic subjects (1-3). Most of these masses (found in ~7% of patients older than age 50 yr) are detected incidentally, during assessment of patients by modern imaging modalities. Although the majority of adrenal tumors are nonfunctional, as demonstrated by biochemical investigations, some are hormone secreting and responsible for endocrine pathology (4, 5). The clinician often faces the difficult task of assessing a patient with an adrenal incidentaloma for the presence of subclinical disease or for the possibility and potential of malignancy. If initial imaging characteristics suggestive of malignancy, such as intratumor necrosis, irregular margins and local metastasis, are not present, regular imaging follow-up should be performed to help detect malignant masses in a timely fashion. Generally, adrenal tumors with a malignant potential grow faster than those with a benign phenotype. However, it should be noted that even adrenal tumors smaller than 2 cm at detection might already be metastatic (6).

The molecular pathogenesis steps of hereditary/genetic adrenal tumors that occur after the "first hit" (the primary gene defect) are not well understood, whereas those of adrenal tumors that occur sporadically are even more obscure. In addition, histological or molecular tumor markers that can reliably distinguish between benign and malignant adrenal

masses are generally lacking (6-12). In 1984, Weiss (7) suggested the introduction of a malignancy score determined by histopathological features of adrenocortical tumors. He acknowledged three limitations of his multifactorial analysis. First, only 25 patients with adrenal adenomas and 18 with carcinomas were studied. Second, the follow-up period of benign tumors was between 5 and 11 yr, whereas it was up to 23 yr for malignant tumors. Third, tumor tissue could be heterogeneous within the same lesion or classified as borderline, and therefore the score, even if established by experienced pathologists, could not be completely reliable.

Some limitations also characterize the diagnostic and prognostic values of genetic and molecular markers studied by Gicquel *et al.* (9). For the 96 patients diagnosed with localized adrenal disease, the follow-up ranged between 5 months and 11.5 yr. A longer follow-up period might have demonstrated a higher number of malignant adrenal tumors, initially classified as benign lesions. Gicquel *et al.* (9) found loss of heterozygosity (LOH) at 17p13 in 23 of 76 (30%) tumors restricted to the adrenal gland and in 11 of 13 (85%) adrenal cancers. Similarly, LOH at 11p15 was observed in 32 of 94 (34%) tumors restricted to the adrenal gland, whereas it was present in 15 of 18 (83%) of adrenal carcinomas. Furthermore, overexpression of IGF2 was demonstrated in 26 of 94 (28%) tumors restricted to the adrenal gland, whereas it was shown in 13 of 18 (83%) of adrenal cancers. These findings suggest that none of the aforementioned genetic and molecular alterations would reliably predict malignancy.

Elucidating the molecular pathogenesis of adrenal tumors may help improve the diagnosis and treatment of patients with such masses. Here, we review genetic/molecular aspects of adrenal tumorigenesis in masses arising from the adrenal cortex and medulla. These structures have embry-

Abbreviations: CDK, Cyclin-dependent kinase; CGH, comparative genomic hybridization; EGF, epidermal growth factor; EGFR, epidermal growth factor receptor; FH, familial hyperaldosteronism; LOH, loss of heterozygosity; MEN1, multiple endocrine neoplasia type 1; MEN2, multiple endocrine neoplasia type 2; NF1, neurofibromatosis type 1; SDHB and SDHB, succinate dehydrogenase subunits D and B; TRAP, telomeric repeat amplification protocol; VHL, von Hippel-Lindau.

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