



# Histology, Effects, Secretion and Regulation of Adrenal Cortex Hormones

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## DESCRIPTION

An infant's adrenal gland weighs twenty times its total weight compared to an adult's gland. The foetal zone, which is considerably wide in the fetus, is found in the place of reticular zone, although it is not formed in anencephali. Maternal cortisol is transferred into the foetus circulation and affects feedback ACTH production through the foetus pituitary gland. Maternal hormones maintain electrolyte balance. Invasion of the Foetal zone begins 3-4 days after birth and proceeds very fast, so that the basic remodeling of the adrenal cortex is finished by the third week after delivery. The newborn child can maintain homeostasis in normal conditions, although the adrenal cortex undergoes rapid remodeling. The exceptions are inherited defects of steroidogenic enzymes in the adrenal cortex, which may lead to, for example, can lead to congenital adrenal hyperplasia. From a histological point of view, the adrenal cortex is differentiated into three zones which also differ in gene expression of enzymes involved in corticoids synthesis. The Zona glomerulosa produces mainly aldosterone (the 11-beta-hydroxylase-free zone); zona fasciculata produces cortisol (P450-aldosynthase-free zone, i.e, 18-hydroxylase/18-oxidase); zona reticularis produces corticoids with androgenic effects such as Dehydroepiandrosterone (DHEA). DHEA levels are high in young age and decrease with growing age. Foetal DHEA is converted to oestrogen in the placenta. Steroid Factor 1 (SF1) is a specific protein involved in corticoid synthesis. It is a transcription factor associated with the TGG CTA motif of the gene regulation domain. If absent, the adrenal glands and gonads are lost.

The basic glucocorticoid is cortisol, whose secretion is regulated by Corticoliberin or Corticotropin-Releasing Hormone (CRH), Adrenocorticotrophic Hormone (ACTH) from the hypothalamus and adenohipophysis respectively. Approximate daily excretion is cortisol 10-20 mg, corticosterone 3 mg, and aldosterone 0.3 mg. The biological half-life of cortisol is about 100 minutes. This process usually involves feedback through glucocorticoid receptors in the hypothalamus and pituitary gland. Disorders may also occur at non-steroidogenic level. A gene defect in the glucocorticoid receptor and a gene mutation for the ACTH receptor

have been described (leading to familial glucocorticoid deficiency syndrome). Essential for hypothalamic-pituitary-adrenal axis function is the nuclear hormone receptor (DAX-1) gene, whose mutations or deletions cause congenital hypoplasia linked with the X chromosome. High cortisol levels reduce CRH and ACTH production within minutes. Long-term high cortisol levels also result in low Pro-opiomelanocortin (POMC) precursor synthesis. Important stimuli for increasing cortisol secretion are stressful situations, including hypoglycemia and anxious reactions (important in pediatrics). The pronounced circadian rhythm of cortisol is used for laboratory diagnosis. The lowest levels are usually around 4 am, but the highest levels of cortisol and ACTH reach 8 am. Cortisol in the blood is bound to protein (about 90%), transcortin (Cortisol-Binding Globulin-CBG), and albumin. Cortisol is mainly metabolized by hepatocytes to 17-hydroxysteroids and excreted in the urine. More than 95% of the metabolites conjugate with glucuronic acid (3 $\alpha$ -hydroxy group) in the liver, and a smaller part is sulfatized at the 21-hydroxy group. The conversion of cortisone (a biologically inactive form that does not bind to glucocorticoid receptors) to cortisol also occurs in the liver. Only a small part is excreted in the urine as free cortisol. Cortisol affects the conversion of basic metabolites. It stimulates the conversion of amino acids to glucose (gluconeogenesis) in the liver and stimulates proteolysis in muscle and lipolysis in adipose tissue (through hormone-sensitive lipase). It has a significant effect on the body's immune response and has anti-allergic, anti-oedematous, anti-inflammatory and anti-exudative effects. Cortisol acts through nuclear steroid receptors. Cortisol or aldosterone first penetrates into the cytoplasm, binds to steroid receptors, and releases heat shock proteins (Hsp90) that previously bound to this structure at the carboxy terminus. Receptors to which hormones are not bound are prevented from translocating to the cell nucleus by hsp. Steroid hormone receptors are dimeric transcription factors.

After binding to steroids, the complex penetrates the cell nucleus, the conformation changes, and the DNA binding site (with "zinc finger") affects the promoter areas involving negative and positive Hormone Response Elements (HREs). These are short (15 bp) palindrome sequences.

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