CORTISOL

A1 Group

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Cortisol

Cortisol, also known more formally as hydrocortisone, is a steroid hormone, more specifically a glucocorticoid, produced by the zona fasciculata of the adrenal gland. It is released in response to stress and a low level of blood glucocorticoids. Its primary functions are to increase blood sugar through gluconeogenesis; suppress the immune system; and aid in fat, protein and carbohydrate metabolism. It also decreases bone formation.

Various synthetic forms of cortisol are used to treat a variety of diseases.
PRODUCTION AND RELEASE

Cortisol is produced in the human body by the adrenal gland in the zona fasciculate (the second of three layers comprising the adrenal cortex). The release of cortisol is controlled by the hypothalamus, a part of the brain. The secretion of corticotropin-releasing hormone (CRH) by the hypothalamus triggers cells in the neighboring anterior pituitary to secrete another hormone, adrenocorticotropic hormone (ACTH), into the vascular system where it is carried by blood to the adrenal cortex.
MAIN FUNCTIONS IN THE BODY

- Cortisol stimulates gluconeogenesis, and it activates anti-stress and anti-inflammatory pathways.
- Elevated levels of cortisol, if prolonged, can lead to proteolysis and muscle wasting.
- Cortisol prevents the release of substances in the body that cause inflammation. This is why cortisol is used to treat conditions resulting from over activity of the B-cell-mediated antibody response such as inflammatory and rheumatoid diseases, and allergies.
- Another function is to decrease bone formation.
- Cortisol plays an important role in glycogenolysis, in liver and muscle tissue.
ADDITIONAL EFFECTS OF CORTISOL

- Increases **blood pressure** by increasing the sensitivity of the vasculature to epinephrine and norepinephrine; in the absence of cortisol, widespread vasodilation occurs.
- Inhibits secretion of corticotropin-releasing hormone (CRH), resulting in feedback inhibition of ACTH secretion.
- Causes the kidneys to produce **hypotonic** urine.
- Shuts down the reproductive system, resulting in an increased chance of miscarriage and (in some cases) temporary infertility. Fertility returns after cortisol levels return to normal.
- There are potential links between cortisol, appetite and obesity.
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In humans, the amount of cortisol present in the blood undergoes diurnal variation; the level peaks in the early morning (approximately 8 am) and reaches its lowest level at about midnight-4 am, or three to five hours after the onset of the light/dark cycle. Information about the sleep of the retina is transmitted from the paired suprachiasmatic nuclei in the hypothalamus to the hypothalamus. This pattern is not present at birth; estimates of when it begins vary from two weeks to nine months of age. Changed patterns of serum cortisol levels have been observed in connection with abnormal ACTH levels, clinical depression, psychological stress, and a.m. or three to five hours after the onset of physiological stressors such as hypoglycemia, illness, fever, trauma, surgery, fear, pain, transmitted from the retina to the physical exertion, or temperature extremes. Cortisol levels may also differ for individuals with autism or Asperger's syndrome.
The primary control of cortisol is the pituitary gland peptide, adrenocorticotropic hormone (ACTH). ACTH probably controls cortisol by controlling the movement of calcium into the cortisol-secreting target cells. ACTH is in turn controlled by the hypothalamic peptide corticotropin releasing hormone (CRH), which is under nervous control.

High potassium media (which stimulates aldosterone secretion in vitro) also stimulate cortisol secretion from the fasciculata zone of canine adrenals.

Potassium loading also increases ACTH and cortisol in humans. This is probably the reason why potassium deficiency causes cortisol to decline and causes a decrease in conversion of 11-deoxycortisol to cortisol. This may also have a role in rheumatoid-arthritis pain (cell potassium is always low in RA).
FACTORS GENERALLY INCREASING CORTISOL LEVELS

- **Caffeine**
- **Sleep deprivation**
- Intense or prolonged **physical exercise** stimulates cortisol release to increase gluconeogenesis and maintain blood glucose.
- **Hypoestrogenism** and **melatonin** supplementation increase cortisol levels in postmenopausal women.
- **Burnout** is associated with higher cortisol levels.
- Severe trauma or stressful events can elevate cortisol levels in the blood for prolonged periods.
- Subcutaneous adipose tissue regenerates cortisol from **cortisone**.
# THE DISORDERS OF CORTISOL SECRETION

<table>
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<tr>
<th>Plasma Cortisol</th>
<th>Plasma ACTH</th>
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<tr>
<td>Primary hypercortisolism (Cushing's syndrome)</td>
<td>Secondary hypercortisolism (pituitary or ectopic tumor, Cushing's disease, pseudo-Cushing's syndrome)</td>
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<tr>
<td>Secondary hypocortisolism (pituitary tumor, Sheehan's syndrome)</td>
<td>Primary hypocortisolism (Addison's disease, Nelson's syndrome)</td>
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A 2010 study has found that serum cortisol predicts increased cardiovascular mortality in patients with acute coronary syndrome.
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CUSHING'S SYNDROME

Cushing's syndrome describes the signs and symptoms associated with prolonged exposure to inappropriately high levels of the hormone cortisol. This can be caused by taking glucocorticoid drugs, or diseases that result in excess cortisol, adrenocorticotropic hormone (ACTH) or CRH levels.

**Cushing's disease** refers to a pituitary-dependent cause of Cushing's syndrome: a tumor (adenoma) in the pituitary gland produces large amounts of ACTH, causing the adrenal glands to produce elevated levels of cortisol. It is the most common non-iatrogenic cause of Cushing's syndrome, responsible for 70% of cases excluding glucocorticoid related
SIGNS AND SYMPTOMS

- extreme weight gain
- muscle and bone weakness
- osteoporosis
- diabetes mellitus
- hypertension
- immune suppression
- baldness
- moodiness, irritability, or depression
- hypercholesterolemia

As amenorrhea in women and decreased fertility in men.
CAUSES

The most common cause of Cushing's syndrome is **exogenous** administration of **glucocorticoids** prescribed by a health care practitioner to treat other diseases. **Endogenous** Cushing's syndrome results from some derangement of the body's own system of secreting cortisol. Normally, **ACTH** is released from the **pituitary gland** when necessary to stimulate the release of cortisol from the **adrenal glands**.
Addison's disease (also known as chronic adrenal insufficiency, and can be caused by damage by the body’s own immune system, certain infections, or various rare causes) is a rare, chronic disease in which the adrenal glands do not produce sufficient mineralocorticoids and glucocorticoids (hormones). It is characterised by a number of relatively nonspecific symptoms, such as abdominal pain and weakness, but under certain circumstances these may progress to Addisonian crisis, a severe illness which may include coma and low blood pressure. The condition arises from problems with the adrenal gland, “primary adrenal insufficiency”, and can be caused by damage by the body’s own immune system, certain infections, or various rare causes. Addison’s disease is also known as chronic primary adrenocortical insufficiency, to distinguish it from acute primary adrenocortical insufficiency, most often caused by Waterhouse-Friderichsen syndrome. Addison’s disease should also be distinguished from secondary and tertiary adrenal insufficiency, which are caused by deficiency of ACTH (produced by the pituitary gland) and CRH (produced by the hypothalamus), respectively. Despite this distinction, Addisonian crises can happen in all forms of adrenal insufficiency.
THANKS FOR LISTENING