

# Hypoaldosteronism

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In **medicine** (**endocrinology**), **hypoaldosteronism** refers to decreased levels of the **hormone aldosterone**.

**Isolated hypoaldosteronism** is the condition of having lowered **aldosterone** without corresponding changes in **cortisol**.<sup>[1]</sup> (The two hormones are both produced by the adrenals.)

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## Causes

There are several causes for this condition, including primary **adrenal insufficiency**, **congenital adrenal hyperplasia**, and medications (certain **diuretics**, **NSAIDs**, and **ACE inhibitors**).

- **Aldosterone deficiency – primary (rare)**
  1. Primary **adrenal insufficiency**
  2. **Congenital adrenal hyperplasia** (**21** and **11β** but not 17)
  3. **Aldosterone synthase** deficiency
- **Hyporeninemic hypoaldosteronism** (due to decreased **angiotensin 2** production as well as intra-adrenal dysfunction)<sup>[2]</sup>
  1. Renal dysfunction-most commonly **diabetic nephropathy**
  2. **NSAIDs**
  3. **Ciclosporin**

## Treatment

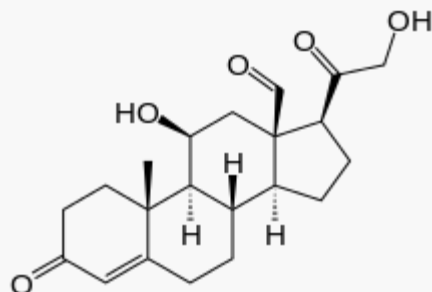
- Aldosterone deficiency should be treated with a **mineralocorticoid** (such as **fludrocortisone**), as well as possibly a **glucocorticoid** for **cortisol** deficiency, if present.
- Hyporeninemic hypoaldosteronism is amenable to **fludrocortisone** treatment,<sup>[2]</sup> but the accompanying **hypertension** and **edema** can prove a problem in these patients, so often a **diuretic** (such as the **thiazide** diuretic, **bendrofluzide**, or a **loop diuretic**, such as **furosemide**) is used to control the **hyperkalemia**.<sup>[3]</sup>

## Effects

This condition may result in **hyperkalemia**, when it is sometimes termed 'type 4 **renal tubular acidosis**' even though it doesn't actually cause **acidosis**. It can also cause urinary **sodium** wasting, leading to volume depletion and **hypotension**.

**Na+** is lost in the urine. **K+** is retained, and the plasma **K+** rises.<sup>[*citation needed*]</sup>

## Hypoaldosteronism



Aldosterone

### Classification and external resources

<b>Specialty</b>	Endocrinology
<b>ICD-10</b>	E27.4 <span><span><span></span></span></span>
<b>DiseasesDB</b>	20960 <span><span><span></span></span></span>
<b>MeSH</b>	D006994 <span><span><span></span></span></span>

[edit on Wikidata]

When adrenal insufficiency develops rapidly, the amount of Na<sup>+</sup> lost from the **extracellular fluid** exceeds the amount excreted in the urine, indicating that Na<sup>+</sup> also must be entering cells. When the **posterior pituitary** is intact, salt loss exceeds water loss, and the plasma Na<sup>+</sup> falls. However, the plasma volume also is reduced, resulting in hypotension, circulatory insufficiency, and, eventually, fatal **shock**. These changes can be prevented to a degree by increasing the dietary **NaCl** intake. Rats survive indefinitely on extra salt alone, but in dogs and most humans, the amount of supplementary salt needed is so large that it is almost impossible to prevent eventual collapse and death unless mineralocorticoid treatment is also instituted.<sup>[*citation needed*]</sup>

## See also [edit]

- [Addison's disease](#)
- [Adrenal gland](#)
- [Hyperaldosteronism](#)
- [Pseudohypoaldosteronism](#)

## References [edit]

- ↑ Becker, Kenneth L. (2001). *Principles and practice of endocrinology and metabolism*​. Lippincott Williams & Wilkins. pp. 785–. ISBN 978-0-7817-1750-2. Retrieved 15 July 2011.
- ↑ *a b* DeFronzo RA (1980). "Hyperkalemia and hyporeninemic hypoaldosteronism". *Kidney Int.* **17** (1): 118–34. doi:10.1038/ki.1980.14​. PMID 6990088​.
- ↑ Sebastian A, Schambelan M, Sutton JM (1984). "Amelioration of hyperchloremic acidosis with furosemide therapy in patients with chronic renal insufficiency and type 4 renal tubular acidosis". *Am. J. Nephrol.* **4** (5): 287–300. doi:10.1159/000166827​. PMID 6524600​.

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**Diseases of the endocrine system (E00–E35 , 240–259)**

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Categories: [Adrenal gland disorders](#)