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Commentary

Aldosterone: A Homeostatic Stabilizer

Astha Sharma*

Department of Pharmacy, School of Medical and Allied Sciences, Galgotias University, Greater Noida, Uttar Pradesh, India

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Aldosterone is important for electrolyte and fluid homeostasis, as well as blood pressure management. The "classical" concept of aldosterone activity is that it stimulates sodium reabsorption and potassium secretion by targeting epithelia of the distal colon and renal nephron. Aldosterone interacts to steroid receptors in these cells, causing them to translocate to the nucleus, where they control gene expression with the induced proteins that stimulate transport. This "genomic" activity is 0.5 h-1.0 h late and is dependent on transcription and translation. Aldosterone's activities have recently been reported as being more fast and independent of transcription and translation. Aldosterone, a steroid hormone produced in the zona glomerulosa of the adrenal cortex, is important for electrolyte and fluid balance. Over the last half-century, it has been the subject of much important research, and several outstanding recent review papers provide in-depth discussion of all areas of aldosterone in physiology. Various sites of aldosterone action inside the central nervous system have been postulated or assumed with little evidence throughout the last few decades. In this review, we describe a series of recent studies that led to the identification and characterization of the first aldosterone-sensitive cells in the brain. Aldosterone has physiological effects on epithelia to maintain fluid and electrolyte homeostasis, as well as proinflammatory effects on a number of non-epithelial cells when salt levels are too high. Mineralocorticoid receptors, members of a wide family of nuclear transcription factors, mediate these effects through DNA-directed, RNA-mediated protein synthesis. The nongenomic actions of aldosterone, which are unaffected by actinomycin D or cycloheximide, have been successfully established in a variety of epithelial and nonepithelial tissues. Despite valiant efforts, no nonclassical aldosterone membrane receptor has been identified, and rapid nongenomic effects mediated by classical mineralocorticoid receptors are being more recognised in the kidney, heart, and vascular wall. Aldosterone affects blood volume and pressure via altering epithelial fluid and electrolyte excretion, as well as blood volume and pressure. Mineralocorticoid receptors are located in epithelium and non-epithelial tissues (vessels, walls, heart, and brain) and have a high affinity for the glucocorticoids cortisol and corticosterone. Blocking mineralocorticoid receptors with spironolactone or eplerenone improves cardiovascular outcomes. Activation of cardiovascular MR reflects aldosterone levels that are inappropriate for salt status in specific instances (primary aldosteronism, experimental mineralocorticoid administration). In contrast to hormone secretion regulation in peptide-secreting cells, where both release and synthesis are significant variables, steroid secretion regulation simply requires an assessment of (those factors). Managing the process of synthesis This mechanism is quite simple in most steroid-secreting tissues, with one or two variables usually regulating the biosynthetic pathways. The regulation of aldosterone secretion is the one exception. The function of aldosterone secretion contributes to the complexity of its control. The main function of aldosterone is to alter sodium (volume) and potassium homeostasis. It performs these duties primarily by enhancing salt reabsorption at the collecting duct and/or distal tubule of the kidney through an energy-dependent process. The excretion of potassium and hydrogen ions increases in response to increased sodium reabsorption. Angiotensins, potassium, pituitary hormones, neurotransmitters, and other aldosterone-stimulating factors are the five subgroups of agents that induce aldosterone release. We will only discuss the three most important secretagogues—angiotensins, potassium, and ACTH-as well as neurotransmitters, because little is known about this final group.

Correspondence to: Astha Sharma, Department of Pharmacy, School of Medical and Allied Sciences, Galgotias University, Greater Noida, Uttar Pradesh, India, E-mail: bharadwajashi@gmail.com

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