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New insights into sodium transport regulation in the distal nephron: Role of GPCRs

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Abstract

The renal handling of Na⁺ balance is a major determinant of the blood pressure level. The inability of the kidney to excrete the daily load of Na⁺ represents the primary cause of chronic hypertension. Among the different segments that constitute the nephron, those present in the distal part (i.e. the cortical thick ascending limb, the distal convoluted tubule, the connecting and collecting tubules) play a central role in the fine-tuning of renal Na⁺ excretion and are the target of many different regulatory processes that modulate Na⁺ retention more or less efficiently. G-protein coupled receptors (GPCR) are crucially involved in this regulation and could represent efficient pharmacological targets to control blood pressure levels. In this review, we describe both classical and novel GPCR-dependent regulatory systems that have been shown to modulate renal Na⁺ absorption in the distal nephron. In addition to the multiplicity of the GPCR that regulate Na⁺ excretion, this review also highlights the complexity of these different pathways, and the connections between them.

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