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Epigenetics of epithelial Na+ channel-dependent sodium uptake and blood pressure regulation

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Abstract

The epithelial Na^+ channel (ENaC) consists of α, β, γ subunits. Its expression and function are regulated by aldosterone at multiple levels including transcription. ENaC plays a key role in Na^+ homeostasis and blood pressure. Mutations in ENaC subunit genes result in hypertension or hypotension, depending on the nature of the mutations. Transcription of αENaC is considered as the rate-limiting step in the formation of functional ENaC. As an aldosterone target gene, αENaC is activated upon aldosterone-mineralocorticoid receptor binding to the cis-elements in the αENaC promoter, which is packed into chromatin. However, how aldosterone alters chromatin structure to induce changes in transcription is poorly understood. Studies by others and us suggest that Dot1a-Af9 complex represses *αENaC* by directly binding and regulating targeted histone H3 K79 hypermethylation at the specific subregions of *αENaC* promoter. Aldosterone decreases Dot1a-Af9 formation by impairing expression of Dot1a and Af9 and by inducing Sgk1, which, in turn, phosphorylates Af9 at S435 to weaken Dot1a-Af9 interaction. MR attenuates Dot1a-Af9 effect by competing with Dot1a for binding Af9.

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