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암모니아운반단백질 발현에서 Foxi1의 역할

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Role of Foxi1 in Rhbg and Rhcg Expression

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Foxi1 is a transcription factor that mediates an important regulatory mechanism in collecting duct development. Genetic disruption of Foxi1 resulted in an intercalated cell-differentiation defect and display renal tubular acidosis. The purpose of this study was to examine the expression of the ammonia transporter family members, Rh B glycoprotein (Rhbg) and Rh C glycoprotein (Rhcg) in Foxi1 -/- mice. Kidney tissues were perfused with periodate-lysine-paraformaldehyde solution and processed for light microscope immunohistochemistry. In control mice, basolateral Rhbg immunoreactivity was heterogeneous, strong in intercalated cells and weak in principal cells. In Foxi1 -/- mice, in the cortical collecting duct (CCD) and outer medullary collecting duct (OMCD) Rhbg immunoreactivity was homogeneous, and overall expression was dramatically decreased. Similarly, Rhcg immunoreactivity was homogeneous and overall expression decreased dramatically in the CCD and OMCD as compared to its expression in wild-type littermates. However, comparison of expression of both Rhbg and Rhcg in Foxi -/- CCD and OMCD to its expression in principal cells in these segments in wild-type littermates showed that expression was similar to, if not slightly greater, to its expression in CCD and OMCD principal cells in wild-type mice. In the connecting segment (CNT), both Rhbg and Rhcg expression appeared unchanged, with basolateral Rhbg immunolabel and apical and basolateral Rhcg expression, and the intensity of immunolabel did not detectably differ between wild-type and Foxi1 -/- mice. Finally, we confirmed the absence of both AE1-positive and pendrin-positive intercalated cells in the CNT, CCD and OMCD. Carbonic anhydrase II (CA II) expression was present in the majority of CCD and OMCD cells, consistent with the known of expression of CA II in mouse principal cells. These findings suggest that Rhbg and Rhcg expression may be regulated through two independent pathways; one downstream of Foxi1-dependent stimulation of A-type intercalated cell and non-A non-B cell development and one independent of Foxi1 and primarily present in CNT cells and principal cells.

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