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Renal Expression of Ammonia Transporter (RhCG, RhBG) in Chronic Renal Failure

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In chronic renal failure (CRF), acid-base balance is maintained until late in the course of the disease by adaptive changes in acid excretion. The current study examines whether changes in the ammonia transporter family members, Rhbg and Rhcg, occur in response to reduced renal mass. CRF was induced in Sprague-Dawley rats by 5/6 nephrectomy; control rats were sham-operated. One week later blood and 24 hr urine samples were collected and kidneys were obtained. Creatinine clearance (ClCr) was decreased (0.25±0.07 mL/min, CRF, vs 0.65±0.14, control, $p<0.05$), plasma HCO_3 was slightly increased (25.7±2.8, CRF, vs 23.0±2.1 mmol/L, control), and plasma Na and K were unchanged. Absolute urinary ammonia excretion was unchanged, but urinary ammonia adjusted for ClCr was increased (11.7±7.2, CRF, vs 4.4±1.6 mol/mL, control, $p<0.05$), indicating adaptations in ammonia metabolism. Single cell Rhcg expression was quantified using immunohistochemistry and quantitative morphometry. Cell height, total cellular expression, expression in the apical and basolateral 25% of the cell, and % of total expression in the apical and basolateral regions were quantified. CRF increased intercalated and principal cell height in the connecting segment (CNT), cortical collecting duct (CCD) and outer medullary collecting duct (OMCD). In intercalated cells in the CNT, CCD and OMCD CRF increased apical Rhcg expression, and in the OMCD CRF increased basolateral expression. In principal cells, CRF increased apical and basolateral Rhcg expression in CCD and OMCD, but not in the CNT. There were no significant changes in Rhbg expression. In summary, CRF induces cell hypertrophy and changes in Rhcg distribution in intercalated and principal cells, suggesting that adaptive changes in Rhcg expression may contribute to adaptive increases in ammonia secretion in CRF.