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## Renal expression of ammonia transporters in the rat with bilateral ureteral obstruction

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**Background:** Bilateral ureteral obstruction is a clinical problem that is often associated with a urinary acidification defect caused by decreased net acid excretion, predominantly due to a decrease in urinary ammonia excretion. The present study aimed at examining the changes in renal expression of the ammonia transporter family members, Rhbg and Rhcg in response to bilateral urinary obstruction and release to identify the cellular and molecular mechanisms for the urinary acidification defect associated with ureteral obstruction.

**Methods:** Male Sprague-Dawley rats underwent 24-hour bilateral ureteral obstruction (BUO), bilateral ureteral obstruction release followed by for 1 day (BUO-R1), 3 days (BUO-R3), and 14 days (BUO-R14). Ureter was exposed and a 4 mm long piece of polyethylene tubing (PE) was placed around the midportion of ureter. The ureter was then occluded by tightening the tubing with a 4.0 silk ligation. The obstructed ureters were decompressed by removal of the ligature and the PE tubing. Animals were placed in metabolic cages and urine was collected under mineral oil. Rhbg, Rhcg and H<sup>+</sup>-ATPase expression was evaluated with immunohistochemistry. Cell height, total cellular expression, expression in the apical 25% of the cell, and % of total expression in the apical regions of Rhcg were quantified using immunohistochemistry with quantitative morphometric analysis.

**Results:** After 24-hour of BUO, the serum bicarbonate (BUO, 27.5 ± 1.4 mmol/L; C, 25.0 ± 1.7; p<0.05), potassium (BUO, 6.9 ± 1.1 mmol/L; C, 4.4 ± 0.9 1.1 mmol/L; p<0.05), creatinine (BUO, 4.9 ± 0.2 mg/dL; C, 1.1 ± 0.8 mg/dL; p<0.05) levels were increased comparing control. 24-hour urinary ammonia excretion were significantly increased in BUO-R1 (1.10±0.79 mmol; p<0.05), BUO-R3 (0.78±0.56 mmol; p<0.05), BUO-R14 (0.43±0.34 mmol; p<0.05) compared with controls (0.12±0.09 mmol). Light microscopy and immunohistochemistry with quantitative morphometric analysis demonstrated that total Rhcg and H<sup>+</sup>-ATPase expression was decreased in the cortical collecting duct and outer medullary collecting duct in BUO, BUO-R1 and BUO-R3, and recovered to control level BUO-R14. Rhbg immunoreactivity was unchanged in bilateral ureteral obstruction and release.

**Conclusion:** The rats with bilateral ureteral obstruction and release showed the changes in renal expression of Rhcg expression in the CCD and OMCD associated with urinary ammonia excretion. These changes suggest that

the ammonia transporter Rhcg mediates a urinary acidification defect associated with bilateral ureteral obstruction.

**Keywords:** Ureteral obstruction; Acidosis; Ammonia; Kidney tubules; Collecting duct