Dysregulation of Renal Aquaporins and Sodium Transporters in Diseases Associated with Water and Sodium Imbalance

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Renal sodium transporters play key roles in counter current multiplication and urinary concentration. Active sodium cloride transport is critically involved in establishing the driving force for water reabsorption in the proximal nephron and collecting duct. It has recently become clear that dysregulation of aquaporins, and especially AQP2 is critically involved in many water balance disorders. Lack of functional AQP2 is seen in primary forms of diabetes insipidus, and reduced expression and targeting is seen in several diseases associated with urinary concentrating defects such as acquired nephrogenic diabetes insipidus, postobstructive polyuria, as well as acute and chronic renal failure. In contrast, in conditions with water retention such as severe congestive heart failure, pregnancy and SIADH both AQP2 expression levels and apical plasma membrane targetting is increased suggesting a role for AQP2 in the development of water retention. In addition to changes in aquaporin targeting and expression key sodium transporters are also dysregulated in urinary concentrating defects such as hypercalcemia-induced NDI, postobstructive polyruria as well as in acute and chronic renal failure. Continued analysis of the aquaporins and sodium transporters are providing detailed molecular insight into the fundamental physiology and pathophysiology of water and sodium balance disorders.