# Erratum

In the article by Bellamkonda K. Kishore, Carissa M. Krane, Max Reif & Anil G. Menon, *Molecular physiology of urinary concentration defect in elderly population*, appearing in *International Urology and Nephrology* 33(2), pp. 235–248 was incorrectly printed.

The two figures on pages 238 and 239 should have been printed in colour. We therefore reprint the two colour figures on the following pages.

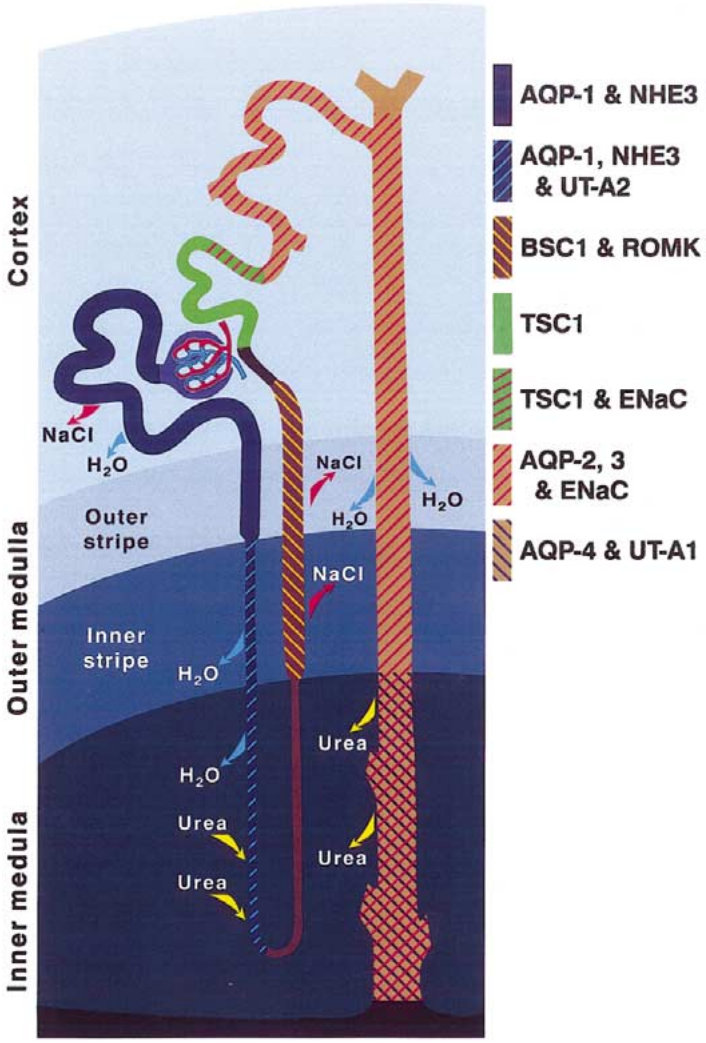


*Figure 1.* Rat nephron and collecting duct showing the segmental localization of key solute transporters and water channels that are involved in the urinary concentration mechanism. AQP1 is abundantly expressed on both apical and basolateral domains of proximal nephron up to the tip of Henle's loop. It is also expressed in the outer medullary descending vasa recta (not depicted in the Figure). The vasopressin-regulated apical water channel (AQP2) is expressed throughout the collecting duct system, and in the connecting tubules of superficial nephrons (not shown here) and the arcade segments of deep or juxtamedullary nephrons (shown here). The basolateral water channel AQP3 follows similar distribution, except that it is predominantly expressed in the cortical and outer medullary regions. The other basolateral water channel (AQP4) is expressed in the inner medullary collecting duct. The sodium hydrogen exchanger isoform 3 (NHE3) is the major salt transporter in the proximal nephron, following a distribution that is similar to that of AQP1. NHE3 is also expressed in thick ascending limb, but not in the thin ascending limb of Henle's loop (not depicted in the Figure). The major thick ascending limb salt transporters are the apical bumetanide-sensitive cotransporter-1 (BSC1; Na-K-2Cl contransporter) and the renal outer medullary potassium channel (ROMK). By virtue of these major apical salt transporters and the basolateral sodium pump, the TAL segment is responsible for generation and maintenance of medullary osmotic gradient or hypertonicity. ROMK is also expressed in connecting tubules and cortical collecting duct principal cells (not depicted in the Figure). The thiazide-sensitive cotransporter-1 (TSC1) is localized in the distal convoluted tubule beyond the macula densa. In addition, the amiloride-sensitive apical sodium channel (ENaC) is expressed in the principal cells of connecting tubules and the collecting duct system, although its abundance is less in IMCD. ENaC is also expressed in the distal part of the distal convoluted tubule, where it is co-expressed with the TSC. The basolateral sodium-pump (Na-K-ATPase) is expressed throughout the nephron and collecting duct system (not depicted in the Figure). The activity of this pump in the thick ascending limb contributes the driving force for transepithelial salt transport, thus generating the medullary hypertonicity. The urea transporter isoforms expressed in the descending limb of Henle's loop (UT-A2), terminal inner medullary collecting duct (UT-A1) and the descending vasa recta (UT-B; not shown in the figure) are responsible for the generation and maintenance of urea concentration gradient in deep inner medulla. For more details see the text.

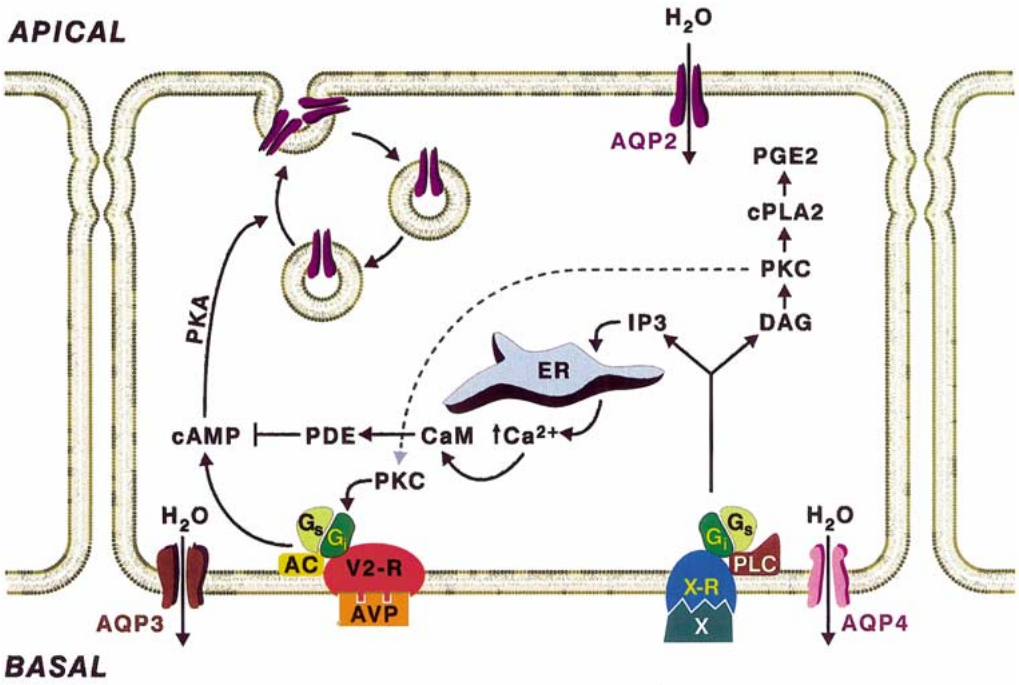


*Figure 2.* Schematic representation of the two mutually opposing signaling pathways and the corresponding membrane receptors involved in the regulation of osmotic water permeability of the collecting duct principal cells. Arginine vasopressin (AVP), or antidiuretic hormone binds to its V2 receptor (V2-R), a G protein-coupled receptor associated with the cyclic-AMP (cAMP) second messenger system. The increased intracellular cAMP levels and the ensuing activation of protein kinase A (PKA) increases the osmotic water permeability of the collecting duct cells by translocating the aquaporin-2 (AQP2) water channel-containing vesicles from subapical region to the apical plasma membrane. The apical plasma membrane is the rate-limiting barrier for transepithelial water flow, as aquaporin-3 (AQP3) and aquaporin-4 (AQP4) are constitutively expressed on the basolateral domain of the collecting duct principal cells under normal conditions. On the other hand, a variety of autacoids and paracrine agents, such as prostaglandin E2, endothelin and extracellular ATP (represented by X), acting through their respective receptors (represented by X-R), and the accompanying phosphoinositide signaling pathway, decrease the osmotic water permeability of the collecting duct. The scheme also illustrates the points where the two signaling pathways interact, such as activation of phosphodiesterases (PDE) by calcium-calmoduling (CaM) pathway, and the activation of inhibitory protein (Gi) associated with the V2 receptor complex by protein kinase C (PKC). Activation of phosphoinositide signaling pathway also results in the increased production of prostaglandin E2 (PGE2) through the activation of cytosolic phospholipase A2 (cPLA2) by calcium ($Ca^{2+}$), PKC and MAP-kinases (not illustrated here). For more details refer to the text. AC, adenylate cylcase; Gs, stimulatory G protein; DAG, diacylglycerol; cPLA2, cytosolic phosholipase A2; IP3, inositol triphosphate; ER, endoplasmic reticulum.