

BACKGROUND

Aquaporins (AQPs) are membrane water channels that play critical roles in controlling the water contents of cells. These channels are widely distributed in all kingdoms of life, including bacteria, plants, and mammals. More than ten different aquaporins have been found in human body, and several diseases, such as congenital cataracts and nephrogenic diabetes insipidus, are connected to the impaired function of these channels. They form tetramers in the cell membrane, and facilitate the transport of water and, in some cases, other small solutes across the membrane. However, the water pores are completely impermeable to charged species, such as protons, a remarkable property that is critical the conservation of membrane's electrochemical potential, but paradoxical at the same time, since protons can usually be transfered readily through water molecules. Water molecules passing the channel are forced, by the protein's electrostatic forces, to flip at the center of the channel, thereby breaking the alternative donoracceptor arrangement that is necessary for proton translocation.1

AQP2 is expressed in the principal cells of the collecting ducts in the kidney and plays a critical role in the urine concentration. A unique feature of AQP2 is that it is stored in the intracellular compartment, and upon stimulation of an antidiuretic hormone (ADH, vasopressin), AQP2 translocates to the apical plasma membrane, where it serves in the uptake of water from the lumen of the collecting duct. It has been shown that AQP2 vesicles constitute a distinct intracellular compartment mostly in the apical cytoplasm and partially overlapping with early endosomes in collecting duct cells in the rat kidney. The AQP2 compartment is distinct from lysosomes, trans-Golgi network (TGN), Golgi apparatus, and endoplasmic reticulum. Numerous studies have focused on AQP2 trafficking from the intracellular compartment to the apical plasma membrane in response to ADH, and a signaling pathway via phosphorylation by protein kinase A and protein kinase G has been proposed. Moreover, retrieval may use the apical endosomal system and the phosphatidylinositol 3-kinase-dependent pathway.² In addition, the mutation of AQP2 leads to nephrogenic diabetes insipidus, the inability to concentrate urine.3

References:

- 1. Jensen, M. Ø. Et al: Structure 9:1083-1093, 2001
- 2. Tajika, Y. et al: Endocrinol. 145: 4375-83, 2004
- 3. Tamarappoo, B.K. & Verkman, A.S.: J. Clin. Investig. 101:2257-67, 1998

TECHNICAL INFORMATION

Source: Anti-AQP2 is a rabbit polyclonal antibody raised against a synthetic peptide corresponding to a sequence mapping near the C-terminal of human AQP2, identical to the related rat sequence.

Specificity and Sensitivity: Anti-AQP2 reacts specifically with AQP2 of human, rabbit, mouse & rat origin in Immunohistochemistry and western blotting procedures, no cross-reactivity with other members of the family.

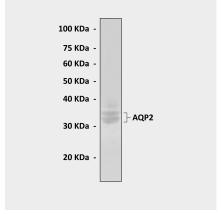
Storage Buffer: PBS and 30% glycerol.

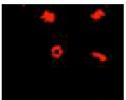
Storage: Store at -20°C for at least one year. Store at 4°C for frequent use. Avoid repeated freeze-thaw cycles.

APPLICATIONS

Application:	*Dilution:
WB	1:500 - 1:1000
IP	n/d
IHC	1:50 - 1:200
ICC	n/d
FACS	n/d
*Optimal dilutions must be determined by end user.	

QUALITY CONTROL DATA





Top: Detection of Aquaporin-2 from rat kidney lysate in Western blot assay, using Anti-AQP2) Antibody. **Bottom:** Immunohistochemical staining of frozen rat kidney tissue using Anti-AQP2 (SABC-CY3 method).





