



Product Information

Anti-Chloride Channel CLC-5 (CLCN5)

Developed in Rabbit, Affinity Isolated Antibody

Product Number **C 1116**

Product Description

Anti-Chloride Channel CLC-5 (CLCN5) was developed in rabbit using a synthetic peptide CDYENHFNTSKG-GEL corresponding to amino acid residues 401-415 of rat CLC-5 as the immunogen. This sequence is identical in mouse and has 14/15 residues identical in human. The antibody was affinity isolated on immobilized immunogen.

Anti-Chloride Channel CLC-5 (CLCN5) detects CLC-5 in Western blot with rat kidney membranes and in immunohistochemistry with rat kidney sections. The antibody is specific for CLC-5; it does not crossreact with other CLC family proteins

Chloride channels represent a functionally and structurally diverse group of anion channels and are located both in the plasma membrane and intracellular organelles.^{1,2} The plasma membrane channels are involved in ionic homeostasis, cell volume regulation, transepithelial transport, and the regulation of excitability in neurons, as well as smooth, cardiac and skeletal muscles. The channels located in intracellular organelles, such as mitochondria, lysosomes, and the Golgi apparatus, are implicated in the passage of anionic substrates; they regulate cellular volume and are required for acidification of these compartments.¹ Chloride channels are divided into five subtypes: voltage-sensitive CLC channels, volume-regulated channels, cAMP-regulated cystic fibrosis transmembrane conductance regulator channels, calcium-activated channels, and maxi or high conductance channels.³

The Voltage-Sensitive CLC family is composed of nine members, CLC-1 through CLC-7 and hCLC-Ka and hCLC-Kb. Each CLC subunit consists of 18 membrane-associated α -helices and contributes a single pore to a dimeric "double-barrelled" channel that contains two independently gated pores, which can be closed together by a common gate.^{1,4,5} These channels have intracellular N- and C-termini and 10-12 transmembrane domains.

The CLC-5 channel is mainly expressed in the kidney, although it has been found also in small intestine, brain and liver. It is predominantly intracellular, mainly found in intracellular vesicles, but can be expressed on plasma membrane.⁶ CLC channels are involved in the intraventricular acidification needed for endocytosis. Mutations in the CLC-5 gene cause Dent's disease, a X-linked renal disorder associated with proteinuria, hypercalciuria, and formation of kidney stones.⁷

Reagent

The antibody is supplied as lyophilized powder from phosphate buffered saline containing 1% bovine serum albumin and 0.025% sodium azide as preservative.

Precautions and Disclaimer

Due to the sodium azide content, a material safety data sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazards and safe handling.

Preparation Instructions

Reconstitute the lyophilized vial with 0.05 ml or 0.2 ml deionized water, depending on the package size purchased. Antibody dilutions should be made in buffer containing 1% bovine serum albumin.

Storage/Stability

Lyophilized powder can be stored intact at room temperature for several weeks. For extended storage, it should be stored at -20°C or below. The reconstituted solution can be stored at $2-8^{\circ}\text{C}$ for up to 2 weeks. For longer storage, freeze in working aliquots. Repeated freezing and thawing is not recommended. Storage in "frost-free" freezers is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Centrifuge all antibody preparations before use (10000 x g 5 min). Working dilution samples should be discarded if not used within 12 hours.

Product Profile

The recommended working dilution is 1:200 for immunoblotting.

Note: In order to obtain best results in different techniques and preparations we recommend determining optimal working concentration by titration test.

References

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4. Dutzler, R., et al., *Nature*, **415**, 287-294 (2002).
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6. Waldegger, S., and Jentsch, T.J., *J. Am. Soc. Nephrol.*, **11**, 1331-1339 (2000).
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