

Product datasheet for **TA328783**

CFTR Rabbit Polyclonal Antibody

Product data:

Product Type:	Primary Antibodies
Applications:	IHC, WB
Recommended Dilution:	WB: 1:200-1:2000; IHC: 1:100-1:3000
Reactivity:	Human, Rat
Host:	Rabbit
Clonality:	Polyclonal
Immunogen:	Peptide (C)KEETEEVQDTRL, corresponding to amino acid residues 1468-1480 of human CFTR . Cytoplasmic, C-terminal part.
Formulation:	Lyophilized. Concentration before lyophilization ~0.8mg/ml (lot dependent, please refer to CoA along with shipment for actual concentration). Buffer before lyophilization: Phosphate buffered saline (PBS), pH 7.4, 1% BSA, 0.05% NaN ₃ .
Reconstitution Method:	Add 50 ul double distilled water (DDW) to the lyophilized powder.
Purification:	Affinity purified on immobilized antigen.
Conjugation:	Unconjugated
Storage:	Store at -20°C as received.
Stability:	Stable for 12 months from date of receipt.
Gene Name:	cystic fibrosis transmembrane conductance regulator
Database Link:	NP_000483 Entrez Gene 24255 Rat Entrez Gene 1080 Human P13569



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Background:

The cystic fibrosis transmembrane conductance regulator (CFTR) is the most dominant Cl channel in several epithelial tissues, especially in lung and colon. Remarkably, CFTR is a member of the ATP-binding cassette (ABC) transporter superfamily that uses ATP hydrolyzation as the driving force for the translocation of a wide variety of substrates including sugars, amino acids, proteins and hydrophobic compounds, across cellular membranes. The CFTR is unique among ABC transporters in that it is a cAMP-regulated Cl channel. It shares the superfamily topology of 12 transmembrane domains with two nucleotide-binding domains (NBDs) and a regulatory (R) domain in the large third intracytoplasmic loop that is phosphorylated in multiple sites by PKA. Mutations in the CFTR gene cause channel dysfunction in several ways, ranging from complete loss of surface expression to diminished Cl secretion. Defects in the CFTR gene cause cystic fibrosis (CF), the most common genetic disease among Caucasians, as well as a form of male sterility. Regulation of the CFTR channel is accomplished through the activation of surface receptors that couple to adenylyl cyclase, raise cAMP cellular levels and thus activate PKA. This has been demonstrated for the adenosine and β_2 adrenergic receptor and the vasopressin hormone among others. Besides enhanced Cl conductance, activation of CFTR also leads to the regulation of other ion channels. The best-studied case is its interaction with the epithelial Na⁺ channels (ENaC), although it can probably regulate other ion channels as well (Kir1.1 for example). The mechanism by which CFTR regulates other ion channels is not clear, but it may involve protein-protein interactions via molecules that interact with its C-terminal PDZ binding motif, such as the NHERF adaptor protein.

Synonyms:

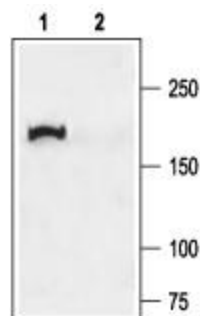
ABC35; ABCC7; CF; CFTR; dj760C5.1; MRP; MRP7; TNR-CFTR

Protein Families:

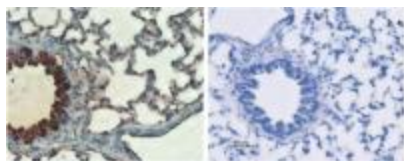
Druggable Genome, Transmembrane

Protein Pathways:

ABC transporters, Vibrio cholerae infection

Product images:

Western blot analysis of rat lung membranes: 1. Anti-CFTR antibody, (1:200). 2. Anti-CFTR antibody, preincubated with the control peptide antigen.



Expression of CFTR in rat lungs. Immunohistochemical staining of rat lungs sections using Anti-CFTR antibody (left panel). Strong staining of bronchial epithelial cells (red) and lighter staining of alveolar cells (red-brown) is apparent. There is also positive staining of macrophages while smooth muscle and endothelium are negative. Counterstain of cell nuclei appears blue. A negative control is shown in the right panel.