SANTA CRUZ BIOTECHNOLOGY, INC.

V-ATPase B1/2 (H-180): sc-20943

SANTA CRUZ BIOTECHNOLOGY
The Power to Question

BACKGROUND

Vacuolar-type H^+-ATPase (V-ATPase) is a multisubunit enzyme responsible for acidification of eukaryotic intracellular organelles. V-ATPases pump protons against an electrochemical gradient, while F-ATPases reverse the process, thereby synthesizing ATP. A peripheral V1 domain, which is responsible for ATP hydrolysis, and a integral V0 domain, which is responsible for proton translocation, compose V-ATPase. Nine subunits (A–H) make up the V1 domain and five subunits (a, d, c, c' and c") make up the V0 domain. Like F-ATPase, V-ATPase most likely operates through a rotary mechanism. The V-ATPase V1 B subunit exists as two isoforms. In the inner ear, the V-ATPase B1 isoform functions in proton secretion and is required to maintain proper endolymph pH and normal auditory function. The gene encoding the human V-ATPase B1 isoform maps to chromosome 2q13.1. Mutations in this gene cause distal renal tubular acidosis associated with sensorineural deafness. The V-ATPase B2 isoform is expressed in kidney and is the only B isoform expressed in osteoclasts. The gene encoding the human V-ATPase B2 isoform maps to chromosome 8p21.3.

CHROMOSOMAL LOCATION

Genetic locus: ATP6V1B1 (human) mapping to 2q13.1, ATP6V1B2 (human) mapping to 8p21.3; Atp6v1b1 (mouse) mapping to 6 C3, Atp6v1b2 (mouse) mapping to 8 B3.3.

SOURCE

V-ATPase B1/2 (H-180) is a rabbit polyclonal antibody raised against amino acids 334-513 mapping at the C-terminus of V-ATPase B1 of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

V-ATPase B1/2 (H-180) is recommended for detection of V-ATPase B1 and V-ATPase B2 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

V-ATPase B1/2 (H-180) is also recommended for detection of V-ATPase B1 and V-ATPase B2 in additional species, including canine, bovine and porcine.

Molecular Weight of V-ATPase B1/2: 56 kDa.

Positive Controls: V-ATPase B1 (h): 293T Lysate: sc-116833, V-ATPase B1 (m2): 293T Lysate: sc-124513 or JAR cell lysate: sc-2276.

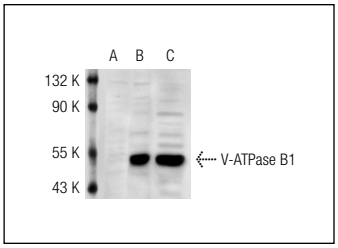
STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

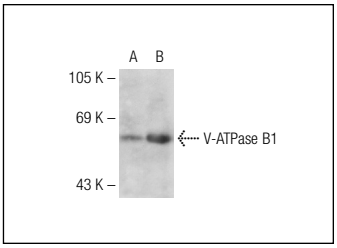
RESEARCH USE

For research use only, not for use in diagnostic procedures.

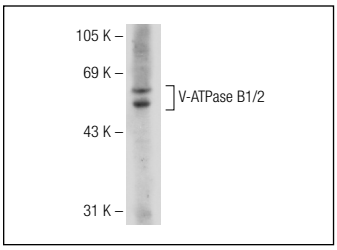
DATA



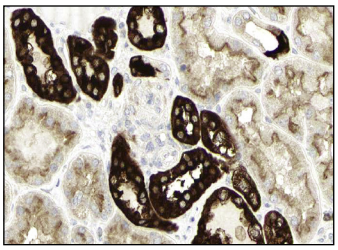
V-ATPase B1/2 (H-180): sc-20943. Western blot analysis of V-ATPase B1 expression in non-transfected 293T: sc-117752 (**A**), human V-ATPase B1 transfected 293T: sc-116833 (**B**) and JAR (**C**) whole cell lysates.



V-ATPase B1/2 (H-180): sc-20943. Western blot analysis of V-ATPase B1 expression in non-transfected: sc-117752 (**A**) and mouse V-ATPase B1 transfected: sc-124513 (**B**) 293T whole cell lysates.



V-ATPase B1/2 (H-180): sc-20943. Western blot analysis of V-ATPase B1/2 expression in mouse kidney tissue extract.



V-ATPase B1/2 (H-180): sc-20943. Immunoperoxidase staining of formalin fixed, paraffin-embedded human kidney tissue showing cytoplasmic staining of cells in glomeruli and tubuli, with strong staining of distal tubules. Kindly provided by The Swedish Human Protein Atlas (HPA) program.

SELECT PRODUCT CITATIONS

1. Kujala, M., et al. 2005. SLC26A6 and SLC26A7 anion exchangers have a distinct distribution in human kidney. Nephron Exp. Nephrol. 101: e50-e58.
2. Han, K.H., et al. 2006. Expression of the ammonia transporter, rh C glycoprotein, in normal and neoplastic human kidney. J. Am. Soc. Nephrol. 17: 2670-2679.
3. Kujala, M., et al. 2007. Expression of ion transport-associated proteins in human efferent and epididymal ducts. Reproduction 133: 775-784.
4. Han, K.H., et al. 2007. Effects of ischemia-reperfusion injury on renal ammonia metabolism and the collecting duct. Am. J. Physiol. Renal Physiol. 293: F1342-F1354.
5. Gao, X., et al. 2010. Deletion of hensin/DMBT1 blocks conversion of β- to α-intercalated cells and induces distal renal tubular acidosis. Proc. Natl. Acad. Sci. USA 107: 21872-21877.