SANTA CRUZ BIOTECHNOLOGY, INC.

V-ATPase D2 (7A4): sc-517031

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 V_1 domain, which is responsible for ATP hydrolysis, and a integral V_0 domain, which is responsible for proton translocation, compose V-ATPase. Nine subunits (A-H) make up the V_1 domain and five subunits (a, d, c, c' and c'') make up the V_0 domain. Like F-ATPase, V-ATPase most likely operates through a rotary mechanism. V-ATPase D2 is a 350 amino acid protein that is expressed in kidney, lung and osteoclast. V-ATPase D2 has been implicated as a regulator of urine acidification, osteoclast fusion and bone formation. Furthermore, V-ATPase D2 has been identified as a dendritic cell marker.

REFERENCES

1. Smith, A.N., et al. 2002. Molecular cloning and characterization of novel tissue-specific isoforms of the human vacuolar H^+-ATPase C, G and d subunits, and their evaluation in autosomal recessive distal renal tubular acidosis. Gene 297: 169-177.
2. Sun-Wada, G.H., et al. 2003. Diversity of mouse proton-translocating ATPase: presence of multiple isoforms of the C, d and G subunits. Gene 302: 147-153.
3. Smith, A.N., et al. 2005. Vacuolar H^+-ATPase d2 subunit: molecular characterization, developmental regulation, and localization to specialized proton pumps in kidney and bone. J. Am. Soc. Nephrol. 16: 1245-1256.
4. Pietrement, C., et al. 2006. Distinct expression patterns of different subunit isoforms of the V-ATPase in the rat epididymis. Biol. Reprod. 74: 185-194.
5. Sato, K., et al. 2006. Selective expression of vacuolar H^+-ATPase subunit d2 by particular subsets of dendritic cells among leukocytes. Mol. Immunol. 43: 1443-1453.
6. Lee, S.H., et al. 2006. v-ATPase V_0 subunit d2-deficient mice exhibit impaired osteoclast fusion and increased bone formation. Nat. Med. 12: 1403-1409.
7. Xu, J., et al. 2007. Structure and function of V-ATPases in osteoclasts: potential therapeutic targets for the treatment of osteolysis. Histol. Histopathol. 22: 443-454.
8. Wu, H., et al. 2009. Atp6v0d2 is an essential component of the osteoclast-specific proton pump that mediates extracellular acidification in bone resorption. J. Bone Miner. Res. 24: 871-885.

CHROMOSOMAL LOCATION

Genetic locus: ATP6V0D2 (human) mapping to 8q21.3.

SOURCE

V-ATPase D2 (7A4) is a mouse monoclonal antibody raised against amino acids 238-306 representing partial length V-ATPase D2 of human origin.

RESEARCH USE

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

PRODUCT

Each vial contains 100 µg IgG_{2b} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

V-ATPase D2 (7A4) is recommended for detection of V-ATPase D2 of 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)] and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

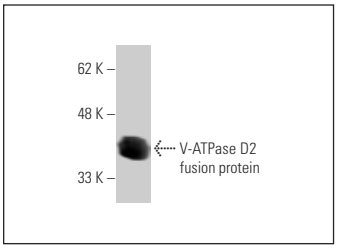
Suitable for use as control antibody for V-ATPase D2 siRNA (h): sc-76885, V-ATPase D2 shRNA Plasmid (h): sc-76885-SH and V-ATPase D2 shRNA (h) Lentiviral Particles: sc-76885-V.

Molecular Weight of V-ATPase D2: 40 kDa.

RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-IgGκ BP-HRP: sc-516102 or m-IgGκ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz Marker™ Molecular Weight Standards: sc-2035, UltraCruz® Blocking Reagent: sc-516214 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml).

DATA



V-ATPase D2 (7A4): sc-517031. Western blot analysis of human recombinant V-ATPase D2 fusion protein.

SELECT PRODUCT CITATIONS

1. Zhuang, J., et al. 2019. Sema6A-plexin-A2 axis stimulates RANKL-induced osteoclastogenesis through PLCγ-mediated NFATc1 activation. Life Sci. 222: 29-35.
2. Delong, C., et al. 2020. Arctiin abrogates osteoclastogenesis and bone resorption via suppressing RANKL-induced Ros and NFATc1 activation. Pharmacol. Res. 159: 104944.
3. Lee, E.J., et al. 2020. Coumarin ameliorates impaired bone turnover by inhibiting the formation of advanced glycation end products in diabetic osteoblasts and osteoclasts. Biomolecules 10: E1052.

STORAGE

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