SANTA CRUZ BIOTECHNOLOGY, INC.

PDE7A (C-21): sc-11131

BACKGROUND

Phosphodiesterases (PDE, also designated cyclic nucleotide phosphodiesterase) are important for the downregulation of the intracellular level of the second messenger cyclic adenosine monophosphate (cAMP) by hydrolyzing cAMP to 5'AMP. Phosphodiesterase type 3 isoforms, PDE3A and 3B, are expressed primarily in cardiovascular tissue and adipose tissue, respectively. PDE3A, is found in myocardium and platelets and PDE3B is found in lymphocytes. The PDE7A1 (HCP1) isozyme and the PDE7A2 proteins, alternate splice products of PDE7A, are highly expressed in skeletal muscle. PDE7B is most highly expressed in pancreas. The PDE family contains proteins that serve tissue-specific roles in regulation of lipolysis, glycogenolysis, myocardial contractility, and smooth muscle relaxation.

REFERENCES

1. Bloom, T.J. and Beavo, J.A. 1996. Identification and tissue-specific expression of PDE7 phosphodiesterase splice variants. Proc. Natl. Acad. Sci. USA 93: 14188-14192.
2. Han, P., et al. 1997. Alternative splicing of the high affinity cAMP-specific phosphodiesterase (PDE7A) mRNA in human skeletal muscle and heart. J. Biol. Chem. 272: 16152-16157.
3. Sheth, S.B., et al. 1997. Cyclic AMP phosphodiesterases in human lymphocytes. Br. J. Haematol. 99: 784-789.
4. Fisher, D.A., et al. 1998. Isolation and characterization of PDE8A, a novel human cAMP-specific phosphodiesterase. Biochem. Biophys. Res. Commun. 246: 570-577.
5. Gantner, F., et al. 1998. Phosphodiesterase profile of human B lymphocytes from normal and atopic donors and the effects of PDE inhibition on B cell proliferation. Br. J. Pharmacol. 123: 1031-1038.

CHROMOSOMAL LOCATION

Genetic locus: PDE7A (human) mapping to 8q13.1; Pde7a (mouse) mapping to 3 A2.

SOURCE

PDE7A (C-21) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the C-terminus of PDE7A 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.

Blocking peptide available for competition studies, sc-11131 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

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.

APPLICATIONS

PDE7A (C-21) is recommended for detection of PDE7A1 and PDE7A2 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) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

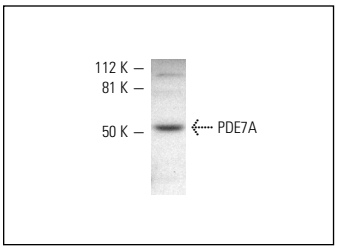
PDE7A (C-21) is also recommended for detection of PDE7A1 and PDE7A2 in additional species, including equine, canine and porcine.

Suitable for use as control antibody for PDE7A siRNA (h): sc-44005, PDE7A siRNA (m): sc-41609, PDE7A shRNA Plasmid (h): sc-44005-SH, PDE7A shRNA Plasmid (m): sc-41609-SH, PDE7A shRNA (h) Lentiviral Particles: sc-44005-V and PDE7A shRNA (m) Lentiviral Particles: sc-41609-V.

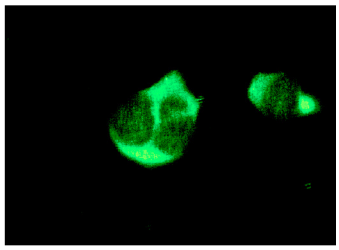
Molecular Weight of PDE7A: 57/50 kDa.

Positive Controls: HuT 78 whole cell lysate: sc-2208.

DATA



PDE7A (C-21): sc-11131. Western blot analysis of PDE7A expression in HuT 78 whole cell lysate.



PDE7A (C-21): sc-11131. Immunofluorescence staining of methanol-fixed HuT 78 cells showing cytoplasmic staining.

SELECT PRODUCT CITATIONS

1. Smith, S.J., et al. 2003. Ubiquitous expression of phosphodiesterase 7A in human proinflammatory and immune cells. Am. J. Physiol. Lung Cell. Mol. Physiol. 284: L279-L289.
2. Asirvatham, A.L., et al. 2004. A-kinase anchoring proteins interact with phosphodiesterases in T lymphocyte cell lines. J. Immunol. 173: 4806-4814.
3. Baxendale, R.W., et al. 2005. Mammalian sperm phosphodiesterases and their involvement in receptor-mediated cell signaling important for capacitation. Mol. Reprod. Dev. 71: 495-508.
4. Dong, H., et al. 2010. Inhibition of PDE3, PDE4 and PDE7 potentiates glucocorticoid-induced apoptosis and overcomes glucocorticoid resistance in CEM T leukemic cells. Biochem. Pharmacol. 79: 321-329.