PRODUCT INFORMATION

PDE3A (human, recombinant)

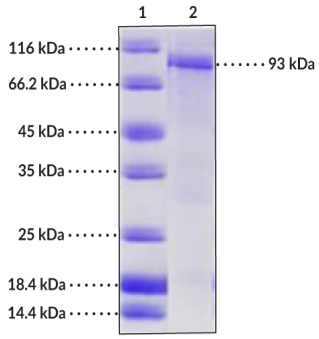
Item No. 32041

Overview and Properties

Synonyms:	CGI-PDE A, cGMP-inhibited 3',5'-Cyclic Phosphodiesterase A, PDE3A, Phosphodiesterase 3A, CGMP-inhibited
Source:	Recombinant human N-terminal His-GST-tagged PDE3A expressed in insect cells
Amino Acids:	669-1141
Uniprot No.:	Q14432
Molecular Weight:	81.7 kDa
Storage:	-80°C (as supplied)
Stability:	≥1 year
Purity:	≥90% estimated by SDS-PAGE
Supplied in:	Lyophilized from sterile 20 mM Tris, pH 7.4, with 500 mM sodium chloride and 10% glycol, 3 mM DTT
Endotoxin Testing:	<1.0 EU/µg, determined by the LAL endotoxin assay

Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Image



Lane 1: MW Markers
Lane 2: PDE3A

SDS-PAGE Analysis of PDE3A. This protein consists of 710 amino acids and has a calculated molecular weight of 81.7 kDa. By SDS-PAGE, under reducing conditions, the molecular mass of the protein is 93 kDa due to apparent post-translational modifications.

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Description

Phosphodiesterase 3A (PDE3A) is a cyclic nucleotide phosphodiesterase that hydrolyzes cGMP and cAMP.[1,2] It is a 125 kDa protein composed of an N-terminal domain important for localization, a linker region, and a conserved C-terminus that contains the catalytic domain.[2] Alternative splicing produces three isomers of PDE3A, PDE3A1, PDE3A2, and PDE3A3, that vary in N-terminal hydrophobic membrane association region (NHR) composition but all contain the same conserved C-terminal catalytic domain. PDE3A is found in the heart, vascular smooth muscle, platelets, and oocytes.[3] Gain-of-function mutations in *PDE3A* have been found in individuals with Mendelian hypertension and brachydactyly type E.[4] *PDE3A* expression is decreased in drug-resistant non-small cell lung cancer (NSCLC) cells, and overexpression of *PDE3A* restores sensitivity to cisplatin (Item No. 13119) in cisplatin-resistant A549 cells.[5] Female *Pde3a* knockout mice exhibit inhibited oocyte maturation and complete sterility.[6] *Pde3a* knockout mice also exhibit increased survival compared with *Pde3a* wild-type mice in a model of collagen and epinephrine-induced pulmonary thromboembolism. Cayman's PDE3A (human, recombinant) protein consists of 710 amino acids and has a calculated molecular weight of 81.7 kDa. By SDS-PAGE, under reducing conditions, the molecular mass of the protein is 93 kDa due to apparent post-translational modifications.

References

1. Küthe, A., Eckel, H., Stief, C.G., *et al.* Molecular biological characterization of phosphodiesterase 3A from human corpus cavernosum. *Chem. Biol. Interact.* **119-120**, 593-598 (1999).
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3. Lugnier, C. Cyclic nucleotide phosphodiesterase (PDE) superfamily: A new target for the development of specific therapeutic agents. *Pharmacol. Ther.* **109(3)**, 366-398 (2006).
4. Maass, P.G., Aydin, A., Luft, F.C., *et al. PDE3A* mutations cause autosomal dominant hypertension with brachydactyly. *Nat. Genet.* **47(6)**, 647-653 (2015).
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6. Begum, N., Shen, W., and Manganiello, V. Role of PDE3A in regulation of cell cycle progression in mouse vascular smooth muscle cells and oocytes: Implications in cardiovascular diseases and infertility. *Curr. Opin. Pharmacol.* **11(6)**, 725-729 (2011).

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