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Produktinformation

- Forschungsprodukte & Biochemikalien
- Zellkultur & Verbrauchsmaterial
- Diagnostik & molekulare Diagnostik
- Laborgeräte & Service

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See the following pages for more information!

Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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PRODUCT INFORMATION

CdnP (*Mycobacterium tuberculosis* strain ATCC 25618/H37Rv recombinant)

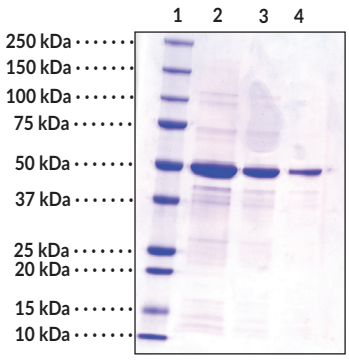
Item No. 22809

Overview and Properties

Synonyms:	Bifunctional Oligoribonuclease and PAP Phosphatase NrnA, 3'(2'),5'-Bisphosphate Nucleotidase, Cyclic di-NMP Phosphodiesterase, EC: 31.13.7, PAP Phosphatase, 3'-Phosphoadenosine 5'-phosphate Phosphatase, Rv2837c
Source:	N-terminal Trx-tagged, His-tagged, and S-tagged *M. tuberculosis* cyclic di-NMP phosphodiesterase protein (full length) purified from *E. coli*
Amino acids:	1-336 (full length)
Uniprot No.:	P71615
Molecular Weight:	52.71 kDa (includes purification tag)
Storage:	-80°C (as supplied) ; avoid freeze/thaw cycles by storing protein in aliquots
Stability:	≥1 year
Purity:	***batch specific*** (≥70% estimated by SDS-PAGE)
Supplied in:	50 mM HEPES, pH 8.0, containing 150 mM sodium chloride
Protein Concentration:	***batch specific*** mg/ml

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

Image



Lane 1: MW Markers
Lane 2: CdnP (5 µg)
Lane 3: CdnP (2 µg)
Lane 4: CdnP (1 µg)

Representative gel image shown; actual purity may vary between batches.

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PRODUCT INFORMATION

Description

Cyclic di-nucleotide phosphodiesterase (CdnP, also known as Rv2837c) is a soluble, stand-alone phosphodiesterase in regulating cyclic dinucleotide signaling during intracellular infections of *M. tuberculosis*.[1] Studies have found that CdnP is capable of hydrolyzing cyclic di-adenosine monophosphate (c-di-AMP; Item No. 17753) into two AMPs as a strategy to avoid activation of the innate immune response of the host. *M. tuberculosis* infection leads to cytosolic release of c-di-AMP, which is recognized by stimulator of interferon genes (STING) and subsequently triggers type I interferon response.[2] Other studies have shown that deletion or inhibition of CdnP attenuates the *M. tuberculosis* virulence both *in vitro* and *in vivo*.[3,4]

References

1. He, Q., Wang, F., Liu, S., *et al*. Structural and biochemical insight into the mechanism of Rv2837c from *Mycobacterium tuberculosis* as a c-di-NMP phosphodiesterase. *J. Biol. Chem.* **291(27)**, 14386-14387 (2016).
2. Dey, B., Dey, R.J., Cheung, L.S., *et al*. A bacterial cyclic dinucleotide activates the cytosolic surveillance pathway and mediates innate resistance to tuberculosis. *Nat. Med.* **21(4)**, 401-406 (2015).
3. Yang, J., Bai, Y., Zhang, Y., *et al*. Deletion of the cyclic di-AMP phosphodiesterase gene (cnpB) in *Mycobacterium tuberculosis* leads to reduced virulence in a mouse model of infection. *Mol. Microbiol.* **93(1)**, 65-79 (2014).
4. Dey, R.J., Dey, B., Zheng, Y., *et al*. Inhibition of innate immune cytosolic surveillance by an *M. tuberculosis* phosphodiesterase. *Nat. Chem. Biol.* **13(2)**, 210-217 (2017).

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