PRODUCT INFORMATION

STING H232 variant (human, recombinant)

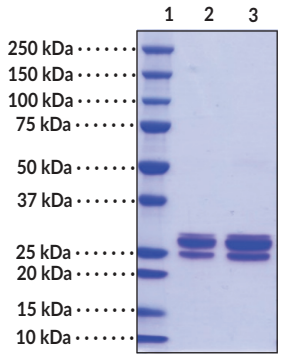
Item No. 22815

Overview and Properties

Synonyms:	Endoplasmic Reticulum Interferon Stimulator, ERIS, hSTING, MITA, MPYS, Stimulator of Interferon Genes, TMEM173
Source:	Recombinant N-terminal His-tagged STING catalytic domain purified from *E. coli*
Amino acids:	138-379 (N-terminal truncation)
Uniprot No.:	Q86WV6
Molecular Weight:	28.8 kDa
Storage:	-80°C (as supplied); avoid freeze/thaw cycles by storing protein in aliquots
Stability:	≥2 years
Purity:	***batch specific*** (≥70% estimated by SDS-PAGE)
Supplied in:	50 mM HEPES, pH 8.0, with 150 mM sodium chloride, 10% glycerol
Protein Concentration:	***batch specific*** mg/ml
Activity:	Serial dilutions of canonical 3'3'-cGAMP were incubated with 5 µg recombinant human STING H232 variant in 50 mM HEPES, pH 7.5, with 150 mM NaCl, 10% glycerol, and SYPRO® Orange dye at 4°C.[1] The reaction was read on a BioRad CFX96 Touch™ Real-Time PCR Detection System at 4-100°C.[2] The binding of the ligand stabilizes the protein structure, increasing the melting temperature (T_m), which is detected *via* a thermal shift assay (TSA), also known as a differential scanning fluorimetry (DSF) assay.[2]

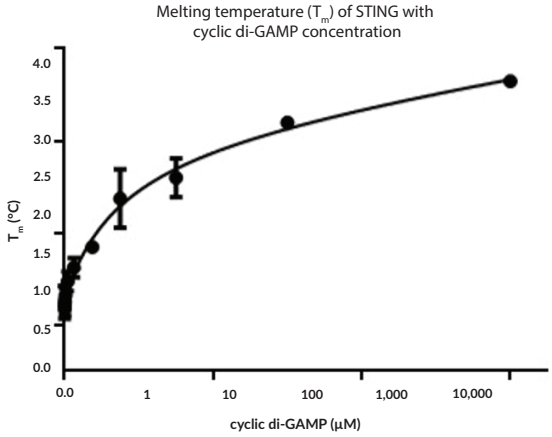
Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Image



Lane 1: MW Markers
Lane 2: STING H232 variant (2 µg)
Lane 3: STING H232 variant (4 µg)

SDS-PAGE Analysis of STING H232.



Binding Activity of STING H232 variant (Item No. 22815). STING H232 variant (5 µg) was incubated with serial dilutions of 3'3'-cGAMP (Item No. 17966) and SYPRO® Orange dye. The detected increase in T_m indicates binding.

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Description

Stimulator of interferon genes (STING) is a component of the innate immune response that binds to cyclic dinucleotides, which are bacterial second messengers, leading to activation of NF-κB and transcription of immunomodulatory genes, including type I interferon (IFN).[3-6] The H232 variant of STING is found at a 13.7% frequency in the 1000 Genome Project.[7] The SNP variant R232 (Item No. 22816) is the most common variant in the human population, found at a frequency of 57.9%. Various mutations in STING either reduce or increase its activity. Gain-of-function mutations in STING, including R284M (Item No. 23594) and V155M, lead to constitutive activation and enhancement of the type I IFN response.[7,8] The V155M mutation is associated with a systemic inflammatory condition, including pulmonary fibrosis and autoimmune factors.[8] Mutations that reduce STING activity include K224R (Item No. 23593), which reduces ubiquitination of STING thereby disrupting its localization within the cell, and the double mutation G230A, R293Q (Item No. 23592), which reduces the IFN response.[7,9] A T596A mutation present in the mouse strain Goldenticket leads to a complete loss of STING protein and lack of a type I IFN response to infection by *Listeria*.[10]

References

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6. Konno, H., Konno, K., and Barber, G.N. Cyclic dinucleotides trigger ULK1 (ATG1) phosphorylation of STING to prevent sustained innate immune signaling. *Cell* **155(3)**, 688-698 (2013).
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9. Ni, G., Konno, H. and Barber, G.N. Ubiquitination of STING at lysine 224 controls IRF3 activation. *Sci. Immunol.* **2(11):eaah7119** (2017).
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