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PRODUCT INFORMATION

STING R232 variant (Y240A mutant; human, recombinant)

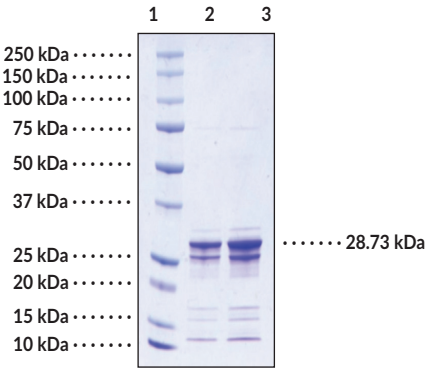
Item No. 40240

Overview and Properties

Synonyms:	Endoplasmic Reticulum Interferon Stimulator, ERIS, Mediator of IRF3 Activation, MITA, Mitochondrial Mediator of IRF3 Activation, MPYS, Stimulator of Interferon Genes, Stimulator of Interferon Response cGAMP Interactor 1, STING1, N-Terminal Methionine-Proline-Tyrosine-Serine Plasma Membrane Tetraspanner, TMEM173, Transmembrane Protein 173
Source:	Recombinant human N-terminal His-tagged STING R232 variant (Y240A mutant) expressed in *E. coli*
Amino Acids:	138-379
Uniprot No.:	Q86WV6
Molecular Weight:	28.73 kDa
Storage:	-80°C (as supplied)
Stability:	≥1 year
Purity:	≥75% estimated by SDS-PAGE
Supplied in:	50 mM HEPES, pH 8.0, 100 mM sodium chloride, 10% glycerol
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: STING R232 variant (2 µg)
Lane 2: STING R232 variant (4 µg)

SDS-PAGE Analysis of STING R232 variant (Y240A mutant; human, recombinant). This protein has a calculated molecular weight of 28.73 kDa.

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PRODUCT INFORMATION

Description

Stimulator of interferon genes (STING) is a component of the innate immune response that recognizes and binds to cyclic dinucleotides (CDNs), which either originate from bacteria or are signals of intracellular stress, leading to activation of NF-κB and transcription of immunomodulatory genes, including type I interferon (IFN).[1-5] The R232 variant is the most common variant in the human population, found at a frequency of 57.9% in the 1000 Genome Project.[6] STING is composed of four transmembrane domains at the N-terminus, a helix α1 domain involved in protein dimerization and ligand sensing, and a cytoplasmic C-terminal domain containing the cyclic dinucleotide-binding domain, as well as the TBK1/IRF1-binding site, TBK1 phosphorylation site, and IRF3 docking site.[7] Various mutations in STING either reduce or increase its activity or binding affinity.[6,8,9,10] The tyrosine-to-alanine substitution at position 240 (Y240A) reduces 2'3'-cGAMP binding to STING and prevents activation of the IFN pathway.[11]

References

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