PRODUCT INFORMATION

STING A162 variant (human, recombinant)

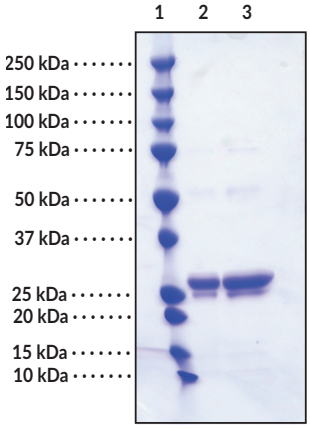
Item No. 25306

Overview and Properties

Source:	N-terminal histidine-tagged human recombinant STING R232, S162A mutant expressed in *E. coli*
Amino Acids:	138-379
Uniprot No.:	Q86WV6
Molecular Weight:	28.8 kDa
Storage:	-80°C (as supplied)
Stability:	≥2 years
Purity:	***batch specific*** (≥75% estimated by SDS-PAGE)
Supplied in:	50 mM HEPES, pH 8.0, 150 mM sodium chloride, 1 mM DTT, and 10% glycerol
Protein Concentration:	***batch specific*** mg/ml
Activity:	Serial dilutions of canonical 3'3'-cGAMP were incubated with 5 µg recombinant human STING A162 variant in 50 mM HEPES, pH 7.5, 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]

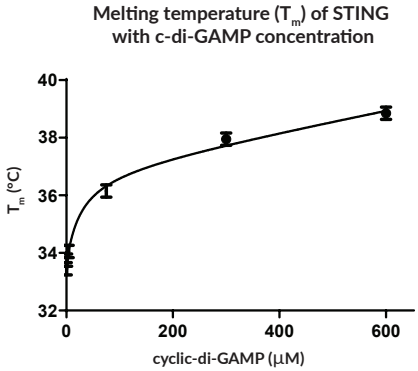
Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Images



Lane 1: MW Markers
Lane 2: STING A162 variant (2 µg)
Lane 3: STING A162 variant (4 µg)

Representative gel image shown; actual purity may vary between each batch.



Binding Activity of STING A162 variant (Item No. 25306). STING A162 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

STING A162 variant (human recombinant) contains amino acids 138-379 of the wild-type variant (R232) with an alanine substituted for serine at position 162. 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 R232 variant of STING is the most common variant in the human population, found at a frequency of 57.9% in the 1000 Genome Project.[7] The SNP variant H232 is found at a 13.7% frequency. The S162A point mutation is located in the cyclic dinucleotide binding site of the human STING variants R232 and H232 and allows human STING to bind to DMXAA, a compound previously known to bind mouse, but not human, STING.[8,9] When STING S162A is bound to DMXAA, it adopts the closed conformation, similar to the conformation it has when bound to the second messenger 2'3'-cGAMP (Item No. 19887), and activates the IFN pathway similarly to mouse STING.[6]

References

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3. Sun, L., Wu, J., Du, F., *et al.* Cyclic GMP-AMP synthase is a cytosolic DNA sensor that activates the type I interferon pathway. *Science* **339(6121)**, 786-791 (2013).
4. Wu, J., Sun, L., Chen, X., *et al.* Cyclic GMP-AMP is an endogenous second messenger in innate immune signaling by cytosolic DNA. *Science* **339(6121)**, 826-830 (2013).
5. 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).
6. Burdette, D.L., Monroe, K.M., Sotelo-Troha, K., *et al.* STING is a direct innate immune sensor of cyclic-di-GMP. *Nature* **478(7370)**, 515-518 (2011).
7. Yi, G., Brendel, V.P., Shu, C., *et al.* Single nucleotide polymorphisms of human STING can affect innate immune response to cyclic dinucleotides. *PLoS One* **8(10)**, e77846 (2013).
8. Gao, P., Ascano, M., Zillinger, T., *et al.* Structure-function analysis of STING activation by c[G(2',5')pA(3',5')p] and targeting by antiviral DMXAA. *Cell* **154(4)**, 748-762 (2013).
9. Conlon, J., Burdette, D.L., Sharma, S., *et al.* Mouse, but not human STING, binds and signals in response to the vascular disrupting agent 5,6-dimethylxanthenone-4-acetic acid. *J. Immunol.* **190(10)**, 5216-5225 (2013).

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