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Produktinformation

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

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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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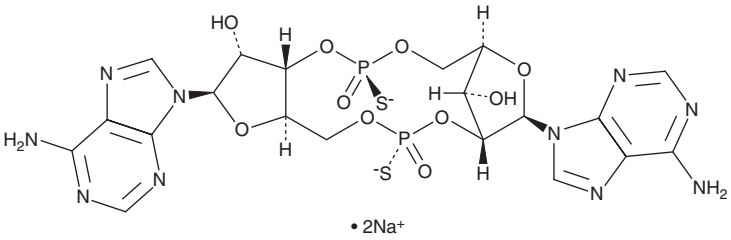
PRODUCT INFORMATION

ADU-S100

Item No. 27901

CAS Registry No.: 1638750-95-4
Formal Name: [P(R)]-5′-O-[(R)-hydroxymercaptophosphinyl]-P-thioadenylyl-(2′→5′)-adenosine cyclic nucleotide, disodium salt
Synonyms: MIW815, ML RR S2-CDA
MF: $C_{20}H_{22}N_{10}O_{10}P_2S_2$ • 2Na
FW: 734.5
Purity: ≥98%
Supplied as: A solid
Storage: -20°C
Stability: ≥4 years



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

Laboratory Procedures

ADU-S100 is supplied as a solid. A stock solution may be made by dissolving the ADU-S100 in the solvent of choice, which should be purged with an inert gas. ADU-S100 is slightly soluble (0.1-1 mg/ml) in acetonitrile. It is also sparingly soluble (1-10 mg/ml) in water. We do not recommend storing the aqueous solution for more than one day.

Description

ADU-S100 is a cyclic dinucleotide STING agonist.[1] It induces IFN-β-dependent reporter activity in HEK293T cells expressing human STING, the human STING variants STING HAQ, STING REF, STING AQ, STING Q, or mouse STING when used at a concentration of 10 µM. Intratumoral administration of ADU-S100 (50 µg/tumor per day for three days) reduces tumor growth in wild-type, but not in *Sting1*$^{-/-}$, mice in a B16/F10 murine melanoma model. It decreases primary and secondary tumor volume in a 4T1 murine breast cancer model of tumor rechallenge when intratumorally administered. Unilateral intratumoral administration of ADU-S100 reduces tumor volumes in a bilateral CT26 murine colorectal cancer model. It increases tumor infiltration of natural killer (NK) cells, $CD4^+$ T cells, and $CD8^+$ T cells, as well as increases immune cell-induced tumor necrosis, in a GL261 murine glioma model when intratumorally administered at a dose of 50 µg/tumor.[2]

References

1. Corrales, L., Glickman, L.H., McWhirter, S.M., *et al.* Direct activation of STING in the tumor microenvironment leads to potent and systemic tumor regression and immunity. *Cell Rep.* **11(7),** 1018-1030 (2015).
2. Berger, G., Knelson, E.H., Jimenez-Macias, J.L., *et al.* STING activation promotes robust immune response and NK cell-mediated tumor regression in glioblastoma models. *Proc. Natl. Acad. Sci. U.S.A.* **119(28),** e2111003119 (2022).

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