# ADU-S100 disodium salt

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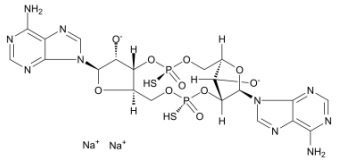
| **Cat. No.:** | HY-12885A |
| **CAS No.:** | 1638750-95-4 |
| **Molecular Formula:** | $C_{20}H_{22}N_{10}Na_2O_{10}P_2S_2$ |
| **Molecular Weight:** | 734.51 |
| **Target:** | STING |
| **Pathway:** | Immunology/Inflammation |
| **Storage:** | Please store the product under the recommended conditions in the COA. |



## BIOLOGICAL ACTIVITY

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| **Description** | ADU-S100 disodium salt is an activator of stimulator of interferon genes (**STING**). |
| **$IC_{50}$ & Target** | STING[1] |
| **In Vitro** | ADU-S100 shows enhanced type I IFN production over CDA in THP-1 human monocytes. In contrast, the dithio, mixed-linkage cyclic dinucleotide (CDN) derivatives (ML RR-CDA, ML RR-S2 CDG, and ML RR-S2 cGAMP) potently activate all five hSTING alleles, including the refractory $hSTING^{REF}$ and $hSTING^{Q}$ alleles. ADU-S100 induces the highest expression of IFN-β and the pro-inflammatory cytokines TNF-α, IL-6, and MCP-1 on a molar equivalent basis, as compared to endogenous ML cGAMP and the TLR3 agonist poly I:C. ADU-S100 is also found to induce aggregation of STING and induce phosphorylation of TBK1 and IRF3 in mouse bone marrow macrophage (BMM). ADU-S100 induces significantly higher levels of IFN-α when compared to ML cGAMP[1]. |
| **In Vivo** | ADU-S100 shows higher anti-tumor control than the endogenous ML cGAMP. A dose response of the ADU-S100 compound is performed in B16 tumor-bearing mice, which identifies an optimal antitumor dose level that also elicites maximum tumor antigen-specific $CD8^+$ T cell responses, and improves long-term survival to 50%[1]. |

## PROTOCOL

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| **Cell Assay [1]** | Cryopreserved hPBMCs are thawed and $1\times10^6$ cells per well are plated in a 96 well plate in RPMI media supplemented with 10% FBS, 1% non-essential amino acids, 1% penicillin/streptomycin, L-glutamine, 10 mM HEPES buffer, 1 mM Sodium Pyruvate, 0.055 mM β-ME at 37°C with 5% $CO_2$. Cells are stimulated with 10 μM ADU-S100 or ML cGAMP for 6 hours and supernatants are harvested. Supernatants are diluted 1:2 and assayed for IFN-α protein using Cytometric Bead Array (CBA) Human Flex Set. Data is collected using a FACSVerse cytometer and analyzed by FCAP Array Software[1].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **Animal Administration [1]** | Mice[1]<br>WT C57BL/6 mice are inoculated with $5\times10^4$ B16.F10 cells in the left flank (n=8). When tumor volumes are 100 $mm^3$ mice receive three IT doses of either ML RR-S2 CDG (25 μg), ADU-S100 (50 μg), or HBSS as control. WT C57BL/6 mice are inoculated with $5\times10^4$ B16.F10 cells in the left flank (n=5). When tumor volumes are 100 $mm^3$ they received three |

IT doses of ADU-S100 at 5, 25, 50 or 100 μg or HBSS as control. WT C57BL/6 mice are inoculated with $5\times10^4$ B16.F10 cells in the left flank (n=8). When tumor volumes are 100 $mm^3$ they receive three IT doses of 100 μg ADU-S100 or HBSS as control. Treatments are administered on days 13, 17 and 20 and tumor measurements are taken twice weekly. Results are shown as percent survival by Log-rank (Mantel-Cox) test (A and C)[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## CUSTOMER VALIDATION

- **Biomaterials**. 2018 May;163:67-75.
- **J Immunother Cancer**. 2019 Sep 18;7(1):252.

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## REFERENCES

[1]. Corrales L, et al. Direct Activation of STING in the Tumor Microenvironment Leads to Potent and Systemic Tumor Regression and Immunity. Cell Rep. 2015 May 19;11(7):1018-30.



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