

Product Data Sheet

Product Name: 8-CPT-2Me-cAMP, sodium salt

Cat. No.: GC11483

Chemical Properties

Cas. No. 634207-53-7

Chemical Name sodium (4aR,6R,7R,7aR)-6-(6-amino-8-((4-chlorophenyl)thio)-9H-purin-9-yl)-7-methoxytetrahydro-4H-furo[3,2-d][1,3,2]dioxaphosphinin-2-olate 2-oxide

SMILES CO[C@]([C@]1([H])N2C3=NC=NC(N)=C3N=C2SC4=CC=C(Cl)C=C4)([H])[C@@](O5)([H])[C@@](O1)([H])COP5([O-])=O.[Na+]

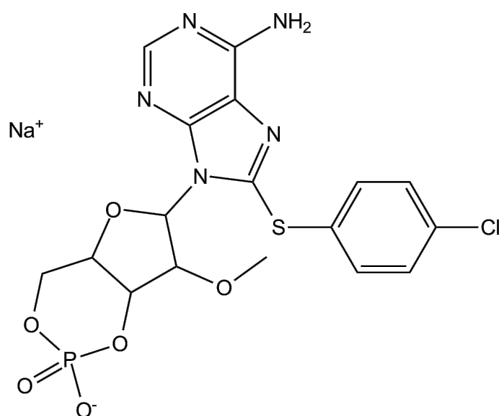
Formula $C_{17}H_{16}ClN_5O_6PS.Na$ M.Wt 507.82

Solubility 0.5mg/mL in ethanol, 25mg/mL in DMSO, 30mg/mL in DMF Storage Desiccate at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request.

Structure



Background

8-CPT-2Me-cAMP, sodium salt is a selective agonist of EPAC [1].

Cyclic AMP guanine nucleotide exchange factors (EPACs) are intracellular sensors for cAMP and function as nucleotide exchange factors for the Ras GTPase homologues Rap1 and Rap2 [1].

Caution: Product has not been fully validated for medical applications. For research use only.

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8-CPT-2Me-cAMP, sodium salt is a selective EPAC agonist. 8-CPT-2Me-cAMP increased Rap1 activation by EPAC1. Meantime, light chain 2 (LC2) of the microtubule-associated protein MAP1A increased this response. In LC2- and EPAC1-transfected cells, 8-CPT-2Me-cAMP increased cell adhesion to laminin [1]. In Jurkat Tcells, 8-CPT-2Me-cAMP (100 μ M) activated Rap1, which was not affected by H-89, a PKA inhibitor [2]. In 1-LN prostate cancer cells, 8-CPT-2Me-cAMP increased Epac1, p-AktS473 and p-AktT308 in a dose-dependent way. 8-CPT-2Me-cAMP increased p-AktS473 and AktS473 kinase activity by two-three fold. Also, 8-CPT-2Me-cAMP activated mTORC1 and mTORC2 [3].

In human prostate cancer cells, 8-CPT-2Me-cAMP increased the levels of p-cPLA2S505, COX-2 and PGE2. However, COX-2, EP4 or mTOR inhibitors inhibited this effect and reduced protein and DNA synthesis induced by Epac1. These results suggested Epac1 was a pro-inflammatory modulator and promoted cell proliferation [4].

References:

- [1]. Gupta M, Yarwood SJ. MAP1A light chain 2 interacts with exchange protein activated by cyclic AMP 1 (EPAC1) to enhance Rap1 GTPase activity and cell adhesion. J Biol Chem, 2005, 280(9): 8109-8116.
- [2]. Fuld S, Borland G, Yarwood SJ. Elevation of cyclic AMP in Jurkat T-cells provokes distinct transcriptional responses through the protein kinase A (PKA) and exchange protein activated by cyclic AMP (EPAC) pathways. Exp Cell Res, 2005, 309(1): 161-173.
- [3]. Misra UK, Pizzo SV. Upregulation of mTORC2 activation by the selective agonist of EPAC, 8-CPT-2Me-cAMP, in prostate cancer cells: assembly of a multiprotein signaling complex. J Cell Biochem, 2012, 113(5): 1488-1500.
- [4]. Misra UK, Pizzo SV. Evidence for a pro-proliferative feedback loop in prostate cancer: the role of Epac1 and COX-2-dependent pathways. PLoS One, 2013, 8(4): e63150.

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