

Product Data Sheet

Product Name: CD 2665

Cat. No.: GC12072

Chemical Properties

Cas. No. 170355-78-9

Chemical Name 4-(7-((1s,3R,5S,7s)-adamantan-1-yl)-6-((2-methoxyethoxy)methoxy)naphthalen-2-yl)benzoic acid

SMILES OC(C1=CC=C(C=C1)C2=CC=C3C(C=C(C(OCOCOC)=C3)[C@]4(C[C@H](C5)C6)C[C@@H]5C[C@H]6C4)=C2)=O

Formula $C_{31}H_{34}O_5$

M.Wt 486.6

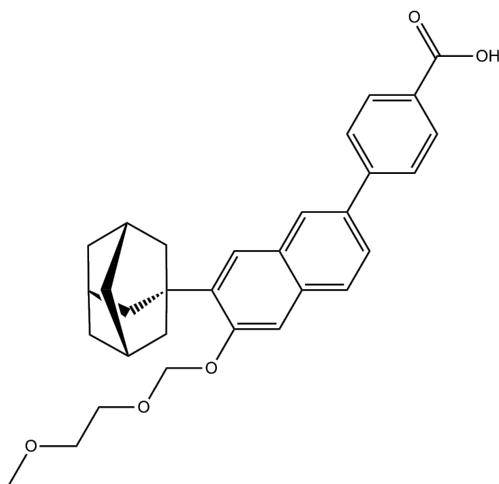
Solubility DMF: 30 mg/mL, DMF:PBS(pH 7.2) (1:1): 0.5 mg/mL, DMSO: 25 mg/mL

Storage Store 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

CD 2665 is a retinoic acid receptor β (RAR β) / RAR γ antagonist [1] [2] [3] with binding K_d values of 306 nM and 110 nM to RAR β and RAR γ , respectively [3].

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

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Retinoic acid receptors include RAR α , RAR β and RAR γ . They are able to bind 9-cis and all-trans stereoisomers of retinoic acid (RA) [1], and mediate RA signal as transcription factors [2].

CD 2665 (1 μ M) did not antagonize the luciferase activity induced by 9-cis- or all-trans-RA in HiB5 cells, but partially blocked the luciferase activity in 3XbRARE-Luc-transfected cells induced by CD 666 (a RAR γ -selective agonist, 100 nM). CD 2665 at 1 μ M reduced the viable cell number to about 60% level of vehicle-treated cells. CD 2665 (1 μ M) clearly antagonized the increase in cell number resulted from the treatment with CD 666 (100 nM) [4].

The biosynthesis of retinoic acid can also be disordered by chronic ethanol consumption. CD 2665 administration did not modify blood alcohol levels in alcohol-treated mice, but totally reversed the impairment of spontaneous alternation rates resulted from alcohol consumption. Administration with CD2665 for 22 days in alcohol-treated mice significantly decreased both mRNA expression and enzymatic activity of tTG (a retinoic acid-target gene) and normalized the expression levels to the level in control mice [2].

References:

- [1]. Yasmin Marikar, ZengQuan Wang, Elizabeth A. Duell, et al. Retinoic Acid Receptors Regulate Expression of Retinoic Acid 4-Hydroxylase that Specifically Inactivates All-Trans Retinoic Acid in Human Keratinocyte HaCaT Cells. The Journal of Investigative Dermatology, 1998, 111(3):434-439.
- [2]. Serge Alfos, Catherine Boucheron, Véronique Pallet, et al. A Retinoic Acid Receptor Antagonist Suppresses Brain Retinoic Acid Receptor Overexpression and Reverses a Working Memory Deficit Induced by Chronic Ethanol Consumption in Mice. Alcohol Clin Exp Res., 2001, 25(10): 1506-1514.
- [3]. Zsuzsa Szondy, Uwe Reichert, Jean-Michel Bernardon, et al. Inhibition of activation-induced apoptosis of thymocytes by all-trans- and 9-cis-retinoic acid is mediated via retinoic acid receptor α . Biochem. J., 1998, 331:767-774.
- [4]. Jean-Ju Chung, Sehyung Cho, Yunhee Kim Kwon, et al. Activation of retinoic acid receptor γ induces proliferation of immortalized hippocampal progenitor cells. Molecular Brain Research, 2000, 83:52-62.

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