

Product Data Sheet

Product Name: SR59230A

Cat. No.: GC39725

Chemical Properties

Cas. No. 174689-39-5

SMILES O[C@@H](CN[C@H]1CCCC2=C1C=CC=C2)COC3=CC=CC=C3CC.O=C(O)C(O)=O

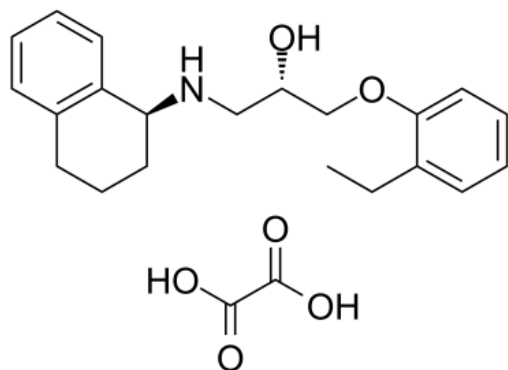
Formula $C_{23}H_{29}NO_6$ M.Wt 415.48

Solubility DMSO: 31.25 mg/mL (75.21 mM) 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



Protocol

Cell experiment [1]:

Cell lines Human umbilical vein endothelial cells (HUVECs)

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HUVECs were cultured in MCDB131 medium supplemented with 10% heat-inactivated fetal bovine serum, 2mM L-glutamine, 10U/ml heparin, 10ng/ml epidermal growth factor (EGF), and 5ng/ml basic fibroblast growth factor, at 37°C, 5% CO₂, and 95% humidity. The cells (2×10^4) were seeded in 24-well sterile plastic plates and allowed to adhere overnight. The cells were treated with SR59230A (0.001, 0.1, 1, 10, and 100µM) for 72 hours, or treated with solvent as a control to assess cell viability.

Reaction Conditions 0.001, 0.1, 1, 10, and 100µM; 72h

Applications SR59230A treatment inhibited the cell viability of HUVECs cells in a dose-dependent manner.

Animal experiment [2]:

Animal models Male C57BL/6J mice

Fifty male C57BL/6J mice (8 weeks old) were raised in a standard environment. The mice were injected with B16F10 cells and then received either SR59230A or L-748,337 treatment. SR59230A and L-748,337 were injected intratumorally at a dose of 5mg/kg/day. The treatment began on the 10th day (D10) after tumor cell injection and continued until D18. The length (L) and width (W) of the tumors were measured daily using a vernier caliper, and the tumor volume was calculated as the formula $L \times W^2 \times 0.5$.

Dosage form 5mg/kg/day for 9 days; intratumoral injection

Applications SR59230A treatment decreases the growth of melanoma in xenograft mouse model of B16F10 cells.

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References:

- [1] Bandini A, Biso L, Viaggi C, et al. Synergistic combination of the adrenergic antagonist SR59230A with common chemotherapeutic drugs and target therapies in cancer and endothelial cells[J]. Investigational New Drugs, 2025: 1-11.
- [2] Dal Monte M, Casini G, Filippi L, et al. Functional involvement of β 3-adrenergic receptors in melanoma growth and vascularization[J]. Journal of molecular medicine, 2013, 91(12): 1407-1419.

Background

SR59230A is a potent and selective β 3-adrenoceptor antagonist, with IC₅₀ values of 40, 408, and 648nM for β 3-, β 1-, and β 2-receptors, respectively [1]. SR59230A is a novel inhibitor of the Kir2.1 channel, with an IC₅₀ value of 33 μ M [2]. SR59230A inhibits the phosphorylation and thereby the activation of the mTOR/p70S6K pathway, crosses the blood-brain barrier, and acts on β 3 adrenoceptors [3-4]. SR59230A has been widely used to inhibit cancer cell progression and to develop novel combination therapies [5].

In vitro, SR59230A treatment for 72 hours significantly inhibited the proliferation of HUVEC, BRAF mutated 8505 C thyroid carcinoma cells, and U-87 cells with IC₅₀ values of 6.45 μ M, 13.09 μ M, and 18.21 μ M, respectively [6]. Treatment with 10 μ M SR59230A for 48 hours induced apoptosis in K562, KCL22, HEL and HL60 cells under hypoxic conditions (1% O₂) [7].

In vivo, SR59230A treatment via continuous intratumoral injection at a dose of 5mg/kg for 8 consecutive days led to a significant reduction in tumor volume and weight in the melanoma mouse model, and inhibited angiogenesis [8]. For a period of 5 weeks, 50 μ g of SR59230A (dissolved in DMSO) was injected into the tail vein every 3 days, which significantly inhibited the growth of breast cancer xenograft tumors in mice and reduced the expressions of p-mTOR S2448, p-4E-BP1 (T37/46), and p-mTOR S2481 [9].

References:

- [1] Kanzler S A, Januario A C, Paschoalini M A. Involvement of β 3-adrenergic receptors in the control of food intake in rats[J]. Brazilian Journal of Medical and Biological Research, 2011, 44: 1141-1147.
- [2] Kulzer M, Seyler C, Welke F, et al. Inhibition of cardiac Kir2. 1–2.3 channels by beta3

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adrenoreceptor antagonist SR 59230A[J]. Biochemical and Biophysical Research Communications, 2012, 424(2): 315-320.

[3] Deng J, Jiang P, Yang T, et al. Targeting β 3-adrenergic receptor signaling inhibits neuroblastoma cell growth via suppressing the mTOR pathway[J]. Biochemical and Biophysical Research Communications, 2019, 514(1): 295-300.

[4] Lob H E, Song J, Hurr C, et al. Deletion of p22phox-dependent oxidative stress in the hypothalamus protects against obesity by modulating β 3-adrenergic mechanisms[J]. JCI insight, 2017, 2(2): e87094.

[5] Ascone M, Banella C, Amato R, et al. Abstract A048 SR59230A-induced ferroptosis sensitization of Ewing sarcoma cells via Beta-3 adrenergic receptor modulation: A novel therapeutic target[J]. Cancer Research, 2024, 84(17_Supplement): A048-A048.

[6] Bandini A, Biso L, Viaggi C, et al. Synergistic combination of the adrenergic antagonist SR59230A with common chemotherapeutic drugs and target therapies in cancer and endothelial cells[J]. Investigational New Drugs, 2025: 1-11.

[7] Calvani M, Dabraio A, Bruno G, et al. β 3-adrenoreceptor blockade reduces hypoxic myeloid leukemic cells survival and Chemoresistance[J]. International Journal of Molecular Sciences, 2020, 21(12): 4210.

[8] Dal Monte M, Casini G, Filippi L, et al. Functional involvement of β 3-adrenergic receptors in melanoma growth and vascularization[J]. Journal of molecular medicine, 2013, 91(12): 1407-1419.

[9] Zhou Z, Zhan J, Luo Q, et al. ADRB3 induces mobilization and inhibits differentiation of both breast cancer cells and myeloid-derived suppressor cells[J]. Cell Death & Disease, 2022, 13(2): 141.

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