Tranilast sodium

Cat. No.: HY-B0195A

CAS No.: 104931-56-8

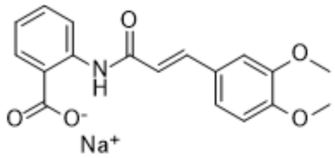
Molecular Formula: $C_{18}H_{16}NNaO_5$

Molecular Weight: 349.31

Target: Prostaglandin Receptor; Angiotensin Receptor

Pathway: GPCR/G Protein

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Tranilast sodium (MK-341 sodium) acts as an anti-atopic agent. Tranilast suppresses production of prostaglandin D2 (PGD2, IC_{50}= 0.1 mM). Tranilast sodium exhibits anti-inflammatory and immunomodulatory effects[1]. Tranilast sodium antagonizes angiotensin II and inhibits its biological effects in vascular smooth muscle cells[2].

IC_{50} & Target		
	Angiotensin II	DP2 0.1 mM (IC_{50})

In Vitro

Tranilast exhibits significant immunomodulatory activity inhibiting Endotoxin-induced prostaglandin E2 (PGE2; IC_{50}=~1-20 μM), thromboxane B2 (IC_{50}=~10-50 μM), (TGF-β1; IC_{50}=~100-200 μM), and IL-8 (IC_{50}=~100 μM) formation. A23187-induced monocyte leukotriene C4 or PGE2 formation is inhibited by Tranilast at IC_{50}s of 10-40 μM and 2-20 μM, respectively[3]. Tranilast (10-200 μM) exhibits the anti-proliferative effect in a dose-dependent manner in both MCF-7 and MDA-MB-231 cell lines. Tranilast also (10-200μM) enhances the anti-tumor effects of Tamoxifen (1-20 μM) on human breast cancer cells in vitro [4].

Tranilast (12.5, 25, 50, 100 μg/mL; 72 hours) inhibits proliferation of HDMECs[5].

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

Cell Proliferation Assay[4]

Cell Line:	MCF-7 and MDA-MB-231 cells
Concentration:	10, 20, 50, 100, and 200 μM
Incubation Time:	48 hours
Result:	Anti-proliferative effect in a dose-dependent manner in both cell lines.

Cell Proliferation Assay[5]

Cell Line:	Human dermal microvascular endothelial cells (HDMECs)
Concentration:	12.5, 25, 50, 100 μg/mL
Incubation Time:	72 hours
Result:	IC_{50} value was 44.3 μg/mL (136 μM).

In Vivo

Tranilast (300 mg/kg; administered orally twice a day for 3 days) dose-dependently suppresses angiogenesis in mice[5].

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Animal Model:	Nine-week-old male C57BL/6 mice[5]
Dosage:	300 mg/kg
Administration:	Administered orally twice a day for 3 days
Result:	Suppressed the VEGF-induced angiogenesis in matrigel; 58% of significant suppression was observed at a dose of 300 mg/kg. The ED_{50} value and 95% confidence limits were 165 mg/kg and 162±169 mg/kg, respectively.

CUSTOMER VALIDATION

- Cell Metab. 2022 Feb 7;34(3):424-440.e7.
- Autophagy. 2021 Nov;17(11):3592-3606.
- Pharmacol Res. 2017 Nov;125(Pt B):150-160.
- J Interferon Cytokine Res. 2021 Mar;41(3):102-110.

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REFERENCES

[1]. K Ikai , et al. Inhibitory Effect of Tranilast on Prostaglandin D Synthetase. Biochem Pharmacol. 1989 Aug 15;38(16):2673-6.

[2]. E A Capper, et al. Modulation of Human Monocyte Activities by Tranilast, SB 252218, a Compound Demonstrating Efficacy in Restenosis. J Pharmacol Exp Ther. 2000 Dec;295(3):1061-9.

[3]. Sara Darakhshan, et al. Tranilast Enhances the Anti-Tumor Effects of Tamoxifen on Human Breast Cancer Cells in Vitro. J Biomed Sci. 2013 Oct 21;20(1):76.

[4]. M Isaji , et al. Tranilast Inhibits the Proliferation, Chemotaxis and Tube Formation of Human Microvascular Endothelial Cells in Vitro and Angiogenesis in Vivo. Br J Pharmacol. 1997 Nov;122(6):1061-6.

[5]. K Miyazawa , et al. Tranilast Antagonizes Angiotensin II and Inhibits Its Biological Effects in Vascular Smooth Muscle Cells. Atherosclerosis. 1996 Apr 5;121(2):167-73.

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

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA