Fenoterol hydrobromide

Cat. No.: HY-B0976A

CAS No.: 1944-12-3

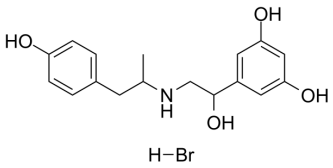
Molecular Formula: $C_{17}H_{22}BrNO_4$

Molecular Weight: 384.26

Target: Adrenergic Receptor

Pathway: GPCR/G Protein; Neuronal Signaling

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (260.24 mM)
H_2O : 25 mg/mL (65.06 mM; Need ultrasonic)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.6024 mL	13.0120 mL	26.0240 mL
5 mM	0.5205 mL	2.6024 mL	5.2048 mL
10 mM	0.2602 mL	1.3012 mL	2.6024 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 60 mg/mL (156.14 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (6.51 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.51 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.51 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Fenoterol hydrobromide (Th-1165a), a sympathomimetic agent, is a selective and orally active β2-adrenoceptor agonist. Fenoterol hydrobromide is an effective bronchodilator and can be used for bronchospasm associated with asthma, bronchitis and other obstructive airway diseases research[1][2].

In Vitro

Fenoterol (1 μM; pre-incubated 30 minutes) treatment reduces AICAR-induced AMPK activation, NF-κB activation and TNF-α

release, and also significantly downregulates the elevated phosphorylation levels of AMPK[2].
Fenoterol inhibits lipopolysaccharide (LPS)-induced AMPK activation and inflammatory cytokine production in THP-1 cells[2].
Fenoterol is also a potent exosome biogenesis and/or secretion activator in PC cells[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[2]

Cell Line:	THP-1 cells stimulated with AICAR
Concentration:	1 μM
Incubation Time:	Pre-incubated 30 minutes
Result:	Significantly downregulated the elevated phosphorylation levels of AMPK.

In Vivo

Fenoterol (0.7 mg/kg; intraperitoneal injection; twice a day; for 3 weeks) treatment suppresses mechanical allodynia during chronic treatment[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male C57BL/6J mice (6 weeks old) with neuropathy[3]
Dosage:	0.7 mg/kg
Administration:	Intraperitoneal injection; twice a day; for 3 weeks
Result:	Alleviated neuropathic allodynia during chronic treatment.

REFERENCES

[1]. Amrita Datta, et al. High-throughput screening identified selective inhibitors of exosome biogenesis and secretion: A drug repurposing strategy for advanced cancer. Sci Rep. 2018 May 25;8(1):8161.

[2]. R C Heel, et al. Fenoterol: a review of its pharmacological properties and therapeutic efficacy in asthma. Drugs. 1978 Jan;15(1):3-32.

[3]. Wei Wang, et al. Anti-inflammatory activities of fenoterol through β-arrestin-2 and inhibition of AMPK and NF-κB activation in AICAR-induced THP-1 cells. Biomed Pharmacother. 2016 Dec;84:185-190.

[4]. Nada Choucair-Jaafar, et al. Beta2-adrenoceptor agonists alleviate neuropathic allodynia in mice after chronic treatment. Br J Pharmacol. 2009 Dec;158(7):1683-94.

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