6,2',4'-Trimethoxyflavone

Cat. No.: HY-103220

CAS No.: 720675-74-1

Molecular Formula: $C_{18}H_{16}O_5$

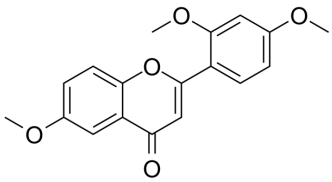
Molecular Weight: 312.32

Target: Aryl Hydrocarbon Receptor

Pathway: Immunology/Inflammation

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 5 mg/mL (16.01 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.2018 mL	16.0092 mL	32.0184 mL
	5 mM	0.6404 mL	3.2018 mL	6.4037 mL
	10 mM	0.3202 mL	1.6009 mL	3.2018 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 0.5 mg/mL (1.60 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 0.5 mg/mL (1.60 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

6,2',4'-Trimethoxyflavone is a potent aryl hydrocarbon receptor (AHR) antagonist. 6,2',4'-Trimethoxyflavone represses AHR-mediated gene induction[1].

In Vitro

6,2',4'-trimethoxyflavone (TMF) as an AHR ligand that possesses the characteristics of an antagonist having the capacity to compete with agonists, such as 2,3,7,8-tetrachlorodibenzo-p-dioxin and benzo[a]pyrene, thus effectively inhibiting AHR-mediated transactivation of a heterologous reporter and endogenous targets, e.g., CYP1A1, independent of cell lineage or species. Furthermore, TMF displays superior action by virtue of having no partial agonist activity, in contrast to other documented antagonists, e.g., alpha-napthoflavone, which are partial weak agonists. TMF also exhibits no species or promoter dependence with regard to AHR antagonism[1].

6,2',4'-Trimethoxyflavone (0-100 μM; 72 hours) shows an inhibitory activity of TNF-α production in THP-1 cells, with IC_{50} of

2.38 μM. 6,2',4'-Trimethoxyflavone shows an inhibitory activity of TNF-α production in B16-F10 cells with IC_{50} of 1.32 μM[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[2]

Cell Line:	THP-1 cells, B16-F10 cells
Concentration:	0-100 μM
Incubation Time:	72 hours
Result:	Showed inhibitory activity of TNF-α production in THP-1 cells and B16-F10 cells.

In Vivo

6,2',4'-trimethoxyflavone-treated (5 mg/kg/day; i.p.) WT mice shows significantly decreased infarct volume, improved sensorimotor, and nonspatial working memory functions compared with their respective controls[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male C57BL/6 wild-type (WT) mice, AHRcKO mice[3]
Dosage:	5 mg/kg/day
Administration:	I.p.
Result:	Both TMF-treated and AHRcKO mice attenuated acute cerebral infarction and functional impairments.

REFERENCES

[1]. Murray IA, et al. Antagonism of aryl hydrocarbon receptor signaling by 6,2',4'-trimethoxyflavone [published correction appears in J Pharmacol Exp Ther. 2018 Nov;367(2):291]. J Pharmacol Exp Ther. 2010;332(1):135-144.

[2]. Apaza T L, et al. Flavonoids of Tripodanthus acutifolius inhibit TNF-α production in LPS-activated THP-1 and B16-F10 cells. J Ethnopharmacol. 2019;242:112036.

[3]. Chen WC, et al. Aryl hydrocarbon receptor modulates stroke-induced astrogliosis and neurogenesis in the adult mouse brain. J Neuroinflammation. 2019;16(1):187. Published 2019 Oct 12.

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