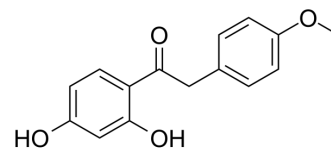


Ononetin

Cat. No.:	HY-108451		
CAS No.:	487-49-0		
Molecular Formula:	C ₁₅ H ₁₄ O ₄		
Molecular Weight:	258.27		
Target:	TRP Channel		
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling		
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 : 100 mg/mL (387.19 mM; Need ultrasonic)				
		Solvent Concentration	Mass 1 mg	5 mg	10 mg
	Preparing Stock Solutions	1 mM	3.8719 mL	19.3596 mL	38.7192 mL
		5 mM	0.7744 mL	3.8719 mL	7.7438 mL
10 mM		0.3872 mL	1.9360 mL	3.8719 mL	
Please refer to the solubility information to select the appropriate solvent.					
In Vivo	<ol style="list-style-type: none"> Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 2.5 mg/mL (9.68 mM); Clear solution Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.5 mg/mL (9.68 mM); Clear solution Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (9.68 mM); Clear solution 				

BIOLOGICAL ACTIVITY

Description	Ononetin, a natural deoxybenzoin, is a potent and selective TRPM3 channel blocker with an IC ₅₀ of 0.3 μM ^[1] .
IC₅₀ & Target	TRPM3 0.3 μM (IC ₅₀)
In Vitro	Ononetin (1-10 μM) completely and reversibly abrogates Ca ²⁺ entry and ionic currents through recombinantly expressed TRPM3α2 and block the pregnenolone sulphate-inducible Ca ²⁺ entry in primary cultures of mouse or rat dorsal root ganglia

(DRG) neurones, indicating biological activity towards endogenously expressed TRPM3^[1].
Oxidative intermediates of Ononetin may be involved in activating TRPA1, but not in the block of TRPM3 by Ononetin^[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Ononetin (10 mg/kg, i.p.) treatment completely reverses established Freund's Complete Adjuvant (FCA)-induced heat hypersensitivity, demonstrating that the loss of hypersensitivity in Trpm3^{-/-} mice is unlikely to be caused by developmental or compensatory mechanisms and suggests that TRPM3 may be a tractable target for inflammatory pain^[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. I Straub, et al. Citrus fruit and fabacea secondary metabolites potently and selectively block TRPM3. Br J Pharmacol. 2013 Apr;168(8):1835-50.

[2]. Omar Alkhatib, et al. Promiscuous G-Protein-Coupled Receptor Inhibition of Transient Receptor Potential Melastatin 3 Ion Channels by Gβγ Subunits. J Neurosci. 2019 Oct 2;39(40):7840-7852.

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

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