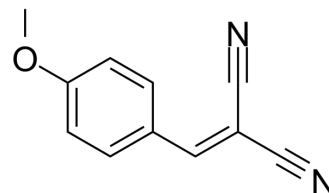


Tyrphostin A1

Cat. No.:	HY-16668		
CAS No.:	2826-26-8		
Molecular Formula:	C ₁₁ H ₈ N ₂ O		
Molecular Weight:	184.19		
Target:	Interleukin Related		
Pathway:	Immunology/Inflammation		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	2 years
		-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (542.92 mM)
 * "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	5.4292 mL	27.1459 mL	54.2918 mL
	5 mM	1.0858 mL	5.4292 mL	10.8584 mL
	10 mM	0.5429 mL	2.7146 mL	5.4292 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
 Solubility: ≥ 2.08 mg/mL (11.29 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Tyrphostin A1 (AG9) inhibits CD40L-stimulated IL-12 production in macrophage cultures and antigen-induced generation of Th1 cells. IC₅₀ value: Target: IL-12 production inhibitor. Addition of increasing concentration of A1 resulted in a dose dependent decrease of IL-12 p40, with maximal inhibition (62.5%) occurring at a dose of 10 μM. Tyrphostin A1 blocks CD40L-induced translocation of NF-κB to the nucleus, and reduces the activation of IL-12 p40 gene. In vivo therapy with A1 leads to decrease in generation of myelin basic protein (MBP) specific encephalitogenic T cells. In addition, treatment of SJL/J mice with A1 results in attenuation of experimental allergic encephalomyelitis (EAE) [1]. Tyrphostin A1 is a much weaker inhibitor of TK than other tyrphostins (IC₅₀>1250 μM for epidermal growth factor receptor (EGFR) kinase), and therefore often used to differentiate TK-mediated effects of tyrphostins from other non-specific effects [2].

IC₅₀ & Target

IL-12

REFERENCES

- [1]. Du C, et al. Inhibition of CD40 signaling pathway by tyrphostin A1 reduces secretion of IL-12 in macrophage, Th1 cell development and experimental allergic encephalomyelitis in SJL/J mice. *J Neuroimmunol.* 2001 Mar 1;114(1-2):69-79.
- [2]. Ogura T, et al. Activation of background membrane conductance by the tyrosine kinase inhibitor tyrphostin A23 and its inactive analog tyrphostin A1 in guinea pig ventricular myocytes. *Jpn J Pharmacol.* 2001 Nov;87(3):235-9.
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Caution: Product has not been fully validated for medical applications. For research use only.

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