WEHI-9625

Cat. No.:	HY-128777			
CAS No.:	2595314-46-6			
Molecular Formula:	C ₃₄ H ₂₇ NO ₅ S ₂			
Molecular Weight:	593.71			
Target:	VDAC; Apoptosis			
Pathway:	Membrane Transporter/Ion Channel; Apoptosis			
Storage:	Powder	-20°C	3 years	
		4°C	2 years	
	In solvent	-80°C	2 years	
		-20°C	1 year	

SOLVENT & SOLUBILITY

Preparing Stock Solutions		Mass Solvent Concentration	1 mg	5 mg	10 mg			
		1 mM	1.6843 mL	8.4216 mL	16.8432 mL			
	5 mM	0.3369 mL	1.6843 mL	3.3686 mL				
		10 mM	0.1684 mL	0.8422 mL	1.6843 mL			
	Please refer to the sc	Please refer to the solubility information to select the appropriate solvent.						
n Vivo	1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: 2.5 mg/mL (4.21 mM); Suspended solution; Need ultrasonic							
		2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: 2.5 mg/mL (4.21 mM); Suspended solution; Need ultrasonic						
	3. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (4.21 mM); Clear solution							

BIOLOGICAL ACTIVITY				
Description	WEHI-9625 is a tricyclic sulfone, first-in-class inhibitor of apoptosis with an EC ₅₀ of 69 nM. WEHI-9625 binds to VDAC2 and promotes its ability to inhibit apoptosis driven by mouse BAK. WEHI-9625 is completely inactive against both human BAK and the closely related apoptosis effector BAX ^[1] .			
IC ₅₀ & Target	Bak	Bax		
In Vitro	WEHI-9625 (0-10 µM; Mcl1 ^{?/?} B	ax ^{?/?} MEFs cells) treatment could prevent cell death mediated by BAK and potently inhibits BIM		

Product Data Sheet

ِّە ە م

ŃН



		BH3-induced loss of mitochondrial membrane potential in Bax ^{?/?} , but not Bak ^{?/?} , cells ^[1] . MCE has not independently confirmed the accuracy of these methods. They are for reference only. Cell Viability Assay ^[1]		
	Cell Line: Mcl1 ^{-/-} Bax ^{-/-} MEFs cells with ABT-737-pretreated			
	Concentration:	0-10 μΜ		
	Incubation Time:			
	Result:	Could prevent cell death.		
In Vivo	WEHL-9625 demonstrate	es that blocking apoptosis at an early stage was both advantageous and pharmacologically tractable		
	WEHI-9625 demonstrates that blocking apoptosis at an early stage was both advantageous and pharmacologically tractable ^[1] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.			

REFERENCES

[1]. van Delft MF, et al. A small molecule interacts with VDAC2 to block mouse BAK-driven apoptosis. Nat Chem Biol. 2019 Oct 7.

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