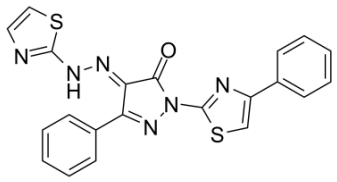
BTSA1

Cat. No.:	HY-123054
CAS No.:	314761-14-3
Molecular Formula:	$C_{21}H_{14}N_6OS_2$
Molecular Weight:	430.51
Target:	Bcl-2 Family; Apoptosis
Pathway:	Apoptosis
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 : 25 mg/mL (58.07 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3228 mL	11.6141 mL	23.2283 mL
	5 mM	0.4646 mL	2.3228 mL	4.6457 mL
	10 mM	0.2323 mL	1.1614 mL	2.3228 mL

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

BIOLOGICAL ACTIVITY

Description

BTSA1 is a potent, high affinity and orally active **BAX** activator with an **IC_{50}** of 250 nM and an **EC_{50}** of 144 nM. BTSA1 binds with high affinity and specificity to the N-terminal activation site and induces conformational changes to **BAX** leading to **BAX**-mediated **apoptosis**[1].

IC_{50} & Target

Bax 250 nM (IC_{50})	Bax 144 nM (EC50)

In Vitro

BTSA1 (5 μM; 6-24 hours; human AML cell lines) treatment reduced viability of all AML cell lines and displays substantial cell death activity within 6 hours[1].

BTSA1 (2.5-10 μM; 6 hours; NB4 cells) treatment induces BAX translocation coincided with the release of cytochrome c from the mitochondria to the cytosol. Significant BAX mitochondrial translocation is induced in a BTSA1 dose-dependent manner[1].

BTSA1 (0.15625-10 μM; 4-24 hours; OCI-AML3 cells) treatment induces dose-dependent caspase-3/7 activation in OCI-AML3 cells. Caspase-3/7 activation is monitored within 4-24 hours and maximal caspase-3/7 activation is

detected in 4 hours[1].

Cell Viability Assaysup>[1]

Cell Line:	Human AML cell lines<
Concentration:	5 μM
Incubation Time:	6 hours, 12 hours, 24 hours
Result:	Reduced viability of all AML cell lines. Displayed substantial cell death activity within 6 hours.

Western Blot Analysis[1]

Cell Line:	NB4 cells
Concentration:	2.5 μM, 5 μM, 10 μM
Incubation Time:	6 hours
Result:	Significant BAX mitochondrial translocation was induced in a dose-dependent manner.

Apoptosis Analysis[1]

Cell Line:	OCI-AML3 cells
Concentration:	0.15625 μM, 0.3125 μM, 0.625 μM, 1.25 μM, 2.5 μM, 5 μM, 10 μM
Incubation Time:	4 hours, 6 hours, 8 hours, 12 hours, 24 hours
Result:	Induced dose-dependent caspase-3/7 activation in OCI-AML3 cells. Caspase-3/7 activation was monitored within 4-24 hr and maximal caspase-3/7 activation was detected in 4 hr.

In Vivo

BTSA1 (10 mg/kg; intraperitoneal injection; every two days; NOD-SCID IL2Rγ null (NSG) mice) treatment significantly increases survival when compared to vehicle-treated mice. BTSA1 treatment induces significant suppression of leukemia growth[1].

Animal Model:	NOD-SCID IL2Rγ null (NSG) mice (6-8 weeks old) with THP-1 cells[1]
Dosage:	10 mg/kg
Administration:	Intraperitoneal injection; every two days
Result:	Significantly increased survival when compared to vehicle-treated mice.

REFERENCES

[1]. Reyna DE, et al. Direct Activation of BAX by BTSA1 Overcomes Apoptosis Resistance in Acute Myeloid Leukemia. Cancer Cell. 2017 Oct 9;32(4):490-505.e10.

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

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