DASA-58

Cat. No.: HY-19330

CAS No.: 1203494-49-8

Molecular Formula: $C_{19}H_{23}N_3O_6S_2$

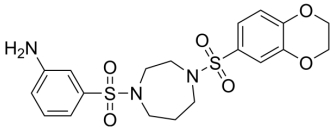
Molecular Weight: 453.53

Target: Pyruvate Kinase

Pathway: Metabolic Enzyme/Protease

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 : ≥ 35 mg/mL (77.17 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.2049 mL	11.0246 mL	22.0493 mL
5 mM	0.4410 mL	2.2049 mL	4.4099 mL
10 mM	0.2205 mL	1.1025 mL	2.2049 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: ≥ 2.08 mg/mL (4.59 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.59 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.59 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

DASA-58 is a potential pyruvate kinase isozyme (PKM2) allosteric activator. DASA-58 can be used for the research of metabolism and kinds of cancer[1].

In Vitro

DASA-58 (15 μM; 2 h) potentiates the antitumor effects of other metabolic stressors[1].
?DASA-58 (15 μM; 24 h, 72 h) enhances pyruvate kinase activity in breast cancer cells without a clear effect on proliferation[1].
?DASA-58 (30 μM, 60 μM; 0-72 h) enhances extracellular acidification and lactate levels in BCa cell lines and induce

extracellular acidification levels in prostate cancer cell lines[1].
?DASA-58 (15 μM, 30 μM; 0-72 h) affects respiration levels in BCa cells without an indication of mitochondrial damage[1].
?DASA-58 (15 μM; 0-72 h) not rescues TXNIP levels in any combintion and mitochondrial inhibitors enhance PKM2 effects on activating AMPK signaling (T172 phosphorylation of AMPK) [1].
?DASA-58 (15 μM; 0-72 h) leads to depletion in TXNIP levels independent of? AMPK and ER signaling, and not through enhanced? proteasomal degradation but rather depleted upstream glycolytic intermediates[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	Five breast cancer cell lines (BCa cells) (MDA MB 231, MDA MB 468, HCC 1443, T47-D, MCF7, LnCap, PC3, and DU145)
Concentration:	15 μM
Incubation Time:	2 h
Result:	Could be exploited by other metabolic stressors.

Western Blot Analysis[1]

Cell Line:	BCa cells
Concentration:	15 μM
Incubation Time:	24 h, 72 h
Result:	Showed comparable PKM2 protein levels in five breast cancer cell lines, except HCC1443 cells and MDA MB 468 that showed the highest and lowest PKM2 protein levels, respectively. Not changed PKM2 levels in five breast cancer cell lines but seemingly reduced TXNIP levels in cells expressing detectable TXNIP levels.

CUSTOMER VALIDATION

- Nature. 2021 Sep;597(7875):263-267.
- Nat Immunol. 2018 Mar;19(3):267-278.
- Nat Commun. 2020 Jun 22;11(1):3162.
- Nat Commun. 2020 Feb 18;11(1):941.
- Cancer Res. 2022 Jul 12;can.21.4222.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Fadi Almouhanna, et al. Pharmacological activation of pyruvate kinase M2 reprograms glycolysis leading to TXNIP depletion and AMPK activation in breast cancer cells. Cancer Metab. 2021 Jan 22;9(1):5.

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