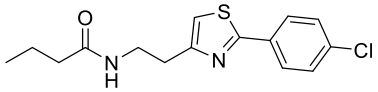
Product data sheet

MedKoo Cat#: 329524 Name: Azoramide CAS#: 932986-18-0 Chemical Formula: $C_{15}H_{17}ClN_2OS$ Exact Mass: 308.075 Molecular Weight: 308.824	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:

Azoramide is a small-molecule modulator of the unfolded protein response with antidiabetic activity. Azoramide mproves ER protein-folding ability and activates ER chaperone capacity to protect cells against ER stress in multiple systems. Azoramide also exhibited potent antidiabetic efficacy in two independent mouse models of obesity by improving insulin sensitivity and pancreatic β cell function. Azoramide can be potential drug candidate for type 2 diabetes.

2. CoA, QC data, SDS, and handling instruction

SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	37.0	119.81
DMF	20.0	64.76
Ethanol	40.5	131.14
Ethanol:PBS (pH 7.2) (1:1)	0.5	1.62

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	3.24 mL	16.19 mL	32.38 mL
5 mM	0.65 mL	3.24 mL	6.48 mL
10 mM	0.32 mL	1.62 mL	3.24 mL
50 mM	0.06 mL	0.32 mL	0.65 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator

Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study

In vitro study

1. Ke M, Chong CM, Zeng H, Huang M, Huang Z, Zhang K, Cen X, Lu JH, Yao X, Qin D, Su H. Azoramide protects iPSC-derived dopaminergic neurons with PLA2G6 D331Y mutation through restoring ER function and CREB signaling. Cell Death Dis. 2020 Feb 18;11(2):130. doi: 10.1038/s41419-020-2312-8. PMID: 32071291; PMCID: PMC7028918.
2. Walenna NF, Kurihara Y, Chou B, Ishii K, Soejima T, Hiromatsu K. Chlamydia pneumoniae infection-induced endoplasmic reticulum stress causes fatty acid-binding protein 4 secretion in murine adipocytes. J Biol Chem. 2020 Feb 28;295(9):2713-2723. doi: 10.1074/jbc.RA119.010683. Epub 2020 Jan 28. PMID: 31992597; PMCID: PMC7049972.

In vivo study

1. Ruan B, Zhu Z, Yan Z, Yang W, Zhai D, Wang L, Ye Z, Lu H, Xiang A, Liang J, Jiang Y, Xu C, Wang Z, Wei M, Lei X, Cao X, Lu Z. Azoramide, a novel regulator, favors adipogenesis against osteogenesis through inhibiting the GLP-1 receptor-PKA-β-catenin pathway. Stem Cell Res Ther. 2018 Mar 9;9(1):57. doi: 10.1186/s13287-018-0771-y. PMID: 29523188; PMCID: PMC5845182.

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2. Fu S, Yalcin A, Lee GY, Li P, Fan J, Arruda AP, Pers BM, Yilmaz M, Eguchi K, Hotamisligil GS. Phenotypic assays identify azoramide as a small-molecule modulator of the unfolded protein response with antidiabetic activity. Sci Transl Med. 2015 Jun 17;7(292):292ra98. doi: 10.1126/scitranslmed.aaa9134. PMID: 26084805; PMCID: PMC5063051.

7. Bioactivity

Biological target:

Azoramide is a dual-function endoplasmic reticulum (ER) modulator.

In vitro activity

CCK-8 assay showed that 3 and 10 μM azoramide significantly enhanced cell viability (27 and 39%, respectively) (Fig. 2e). Therefore, 10 μM was used as a working concentration of azoramide for neuroprotection. Western blotting demonstrated that 10 μM azoramide dramatically inhibited the release of cytochrome c from mitochondria and decreased the cleaved level of caspase 3 and the ratio of Bax/Bcl2 in PLA2G6 mutant neurons (Fig. 2g, h). Azoramide significantly enhanced expression of CREB in PLA2G6 mutant neurons (Fig. 2g, h).

Reference: Cell Death Dis. 2020 Feb; 11(2): 130. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7028918/

In vivo activity

Briefly, 14-day-old C57BL/6 mice were injected with recombinant BMP2 three times a day for 5 days into the periosteal tissue overlying the right parietal bone with vehicle injected to the left parietal bone as a negative control. Meanwhile, azoramide and DMSO control were administered via intraperitoneal injection once a day for 14 consecutive days (Fig. 1a). The azoramide treatment group, induced by BMP2, showed remarkable reductions in new bone formations compared with the control group (Fig. 1b); the volume of newly formed bone was approximately 68% smaller in the groups injected with azoramide (Fig. 1b, c). These data indicated that azoramide treatment could impair the BMP2-induced formation of new bone. Histological analysis of the local new bone tissues in responses to azoramide showed an obvious decrease in the number of osteoblasts and a significant increase in the number of adipocytes in the newly formed bone marrow spaces after azoramide treatment (Fig. 1d, e). Taken together, the results suggested that azoramide inhibited the bone forming ability of mesenchymal progenitor cells under BMP2 induction. More likely, it preferentially induces MSCs towards adipocytes rather than osteoblasts.

Reference: Stem Cell Res Ther. 2018; 9: 57. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5845182/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.