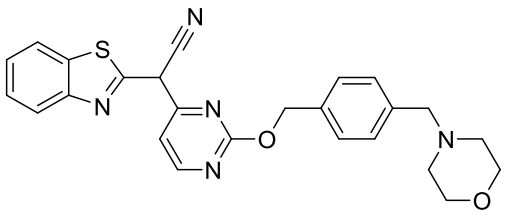
Product data sheet

MedKoo Cat#: 319883 Name: Bentamapimod CAS#: 848344-36-5 Chemical Formula: $C_{25}H_{23}N_5O_2S$ Exact Mass: 457.1572 Molecular Weight: 457.552	
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:

Bentamapimod, also known as AS602801 and PGL5001, is a JNK Inhibitor. AS602801 induces regression of endometriotic lesions in animal models. AS602801 interrupts immune pathways. Bentamapimod induced regression of endometriotic lesions in endometriosis rodent animal models without suppressing ER action. Endometriosis is an estrogen (ER)-dependent gynecological disease caused by the growth of endometrial tissue at extrauterine sites.

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	13.43	29.35
DMF	2.0	4.37

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.19 mL	10.93 mL	21.86 mL
5 mM	0.44 mL	2.19 mL	4.37 mL
10 mM	0.22 mL	1.09 mL	2.19 mL
50 mM	0.04 mL	0.22 mL	0.44 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. Kuramoto K, Yamamoto M, Suzuki S, Sanomachi T, Togashi K, Seino S, Kitanaka C, Okada M. AS602801, an Anti-Cancer Stem Cell Drug Candidate, Suppresses Gap-junction Communication Between Lung Cancer Stem Cells and Astrocytes. Anticancer Res. 2018 Sep;38(9):5093-5099. doi: 10.21873/anticanres.12829. PMID: 30194154.
2. Okada M, Kuramoto K, Takeda H, Watarai H, Sakaki H, Seino S, Seino M, Suzuki S, Kitanaka C. The novel JNK inhibitor AS602801 inhibits cancer stem cells in vitro and in vivo. Oncotarget. 2016 May 10;7(19):27021-32. doi: 10.18632/oncotarget.8395. PMID: 27027242; PMCID: PMC5053629.

In vivo study

1. Chen M, Sun J, Lu C, Chen X, Ba H, Lin Q, Cai J, Dai J. The impact of neuronal Notch-1/JNK pathway on intracerebral hemorrhage-induced neuronal injury of rat model. Oncotarget. 2016 Nov 8;7(45):73903-73911. doi: 10.18632/oncotarget.12094. PMID: 27655677; PMCID: PMC5342022.
2. Palmer SS, Altan M, Denis D, Tos EG, Gotteland JP, Osteen KG, Bruner-Tran KL, Nataraja SG. Bentamapimod (JNK Inhibitor AS602801) Induces Regression of Endometriotic Lesions in Animal Models. Reprod Sci. 2016 Jan;23(1):11-23. doi: 10.1177/1933719115600553. Epub 2015 Sep 2. PMID: 26335175; PMCID: PMC5933194.

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7. Bioactivity

Biological target:

Bentamapimod (AS 602801) is an ATP-competitive JNK inhibitor with IC50 of 80 nM, 90 nM, and 230 nM for JNK1, JNK2, and JNK3, respectively.

In vitro activity

AS602801 treatment induced cell death and accordingly decreased the number of viable cells in all three cell lines in a dose-dependent manner, suggesting that AS602801 may have selective cytotoxic activity against neoplastic cells (Figure 1A and 1B). This study next investigated whether cancer stem cells derived from these cell lines (PANC-1 CSLCs, A549 CSLCs, and A2780 CSLCs) were resistant to AS602801-induced cell death. AS602801 induced cell death in these cells as efficiently as in the original cell lines, suggesting that the cancer stem cell and non-cancer stem cell subpopulations within a cell line are equally sensitive to AS602801 (Figure 2A and 2B). GS-Y01 cells, which are patient-derived glioma stem cells, were also tested to examine whether AS602801 has cytotoxic activity against cells established directly from patient tumor tissues. AS602801 also had cytotoxic activity against GS-Y01 cells (Figure 2A and 2B).

Reference: Oncotarget. 2016 May 10; 7(19): 27021–27032. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5053629/

In vivo activity

Compared to control or Antide-treated rats, both total c-Jun (Figure 6A-C) and phospho-c-Jun staining (Figure 6D-F) were decreased with bentamapimod treatment. The reduction in phospho-c-Jun was more dramatic (Figure 6E) in mice treated with bentamapimod than the change in total c-Jun (Figure 6B). As expected, signs of apoptosis as measured by TUNEL staining were evident in sections from bentamapimod-treated uteri and Antide-treated uteri, relative to control (Figure 6G-I). Lastly, CD45 staining, reflecting a broad marker for T and B lymphocytes, was suppressed by AS602801, whereas the presence of CD45 was unaffected by Antide treatment (Figure 6L-N). These results suggest that bentamapimod has potential to reduce the number of CD45+ lymphocytes recruited to lesions (perhaps through inhibition of phosphorylated c-Jun) or alternatively to reduce the presence of T or B cells present within lesions, through processes that could include apoptosis. These results confirm an effect of JNK inhibition on the inflammatory and/or immune responses to endometriosis and demonstrate that this effect occurs through a different mechanism than endocrine regulators such as Antide.

Reference: Reprod Sci. 2016 Jan; 23(1): 11–23. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5933194/

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.