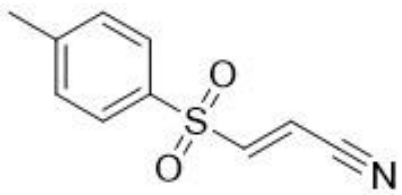
Product data sheet

MedKoo Cat#: 522392 Name: BAY 11-7082 CAS#: 19542-67-7 Chemical Formula: $C_{10}H_9NO_2S$ Exact Mass: 207.0354 Molecular Weight: 207.25	
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:

BAY 11-7082 is an inhibitor of κB kinase (IKK) that has pharmacological activities that include anticancer, neuroprotective, and anti-inflammatory effects. BAY 11-7082 strongly suppressed the production of nitric oxide, prostaglandin E(2), and tumor necrosis factor-α and reduced the translocation of p65, major subunit of nuclear factor-κB, and its upstream signaling events such as phosphorylation of IκBα, IKK, and Akt. In addition. Bay 11-7082 induces cell death independent from inhibition of activation of NFκB transcription factors.BAY 11-7082 suppresses the MyD88-dependent signalling network by targeting the ubiquitin system. Bay 11-7082 induces cell death independent from inhibition of activation of NFκB transcription factors.

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	55.0	267.0

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	4.83	24.13	48.25
5 mM	0.97	4.83	9.65
10 mM	0.48	2.41	4.83
50 mM	0.10	0.48	0.97

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. Strickson S, Campbell DG, Emmerich CH, Knebel A, Plater L, Ritorto MS, Shpiro N, Cohen P. The anti-inflammatory drug BAY 11-7082 suppresses the MyD88-dependent signalling network by targeting the ubiquitin system. Biochem J. 2013 May 1;451(3):427-37. doi: 10.1042/BJ20121651. PMID: 23441730; PMCID: PMC3685219.
2. Wang Y, Zhang XL, Sun CM. BAY-11-7082 induces apoptosis of multiple myeloma U266 cells through inhibiting NF-κB pathway. Eur Rev Med Pharmacol Sci. 2018 May;22(9):2564-2571. doi: 10.26355/eurrev_201805_14949. PMID: 29771406.

In vivo study

1. Sasaki CT, Doukas SG, Vageli DP. In Vivo Short-Term Topical Application of BAY 11-7082 Prevents the Acidic Bile-Induced mRNA and miRNA Oncogenic Phenotypes in Exposed Murine Hypopharyngeal Mucosa. Neoplasia. 2018 Apr;20(4):374-386. doi: 10.1016/j.neo.2018.02.001. Epub 2018 Mar 9. PMID: 29529473; PMCID: PMC5909679.
2. Vageli DP, Kasle D, Doukas SG, Doukas PG, Sasaki CT. The temporal effects of topical NF-κB inhibition, in the in vivo prevention of bile-related oncogenic mRNA and miRNA phenotypes in murine hypopharyngeal mucosa: a preclinical model. Oncotarget. 2020 Sep 1;11(35):3303-3314. doi: 10.18632/oncotarget.27706. PMID: 32934775; PMCID: PMC7476734.

Product data sheet

7. Bioactivity

Biological target:

BAY 11-7082 is an IκBα phosphorylation and NF-κB inhibitor that selectively and irreversibly inhibits the TNF-α-induced phosphorylation of IκB-α and also inhibits ubiquitin-specific protease USP7 and USP21 (IC50=0.19, 0.96 μM, respectively).

In vitro activity

It was found that BAY 11-7082 did not inhibit IKKα, IKKβ and the IKK-related kinases TBK1 and IKKϵ when assayed at 10 μM in vitro (Supplementary Table S1 at http://www.biochemj.org/bj/451/bj4510427add.htm). Nevertheless, BAY 11-7082 completely suppressed the LPS-stimulated (Figure 2A) and IL-1-stimulated (Figure 2B) phosphorylation of the activation loop of IKKβ. As a consequence, the phosphorylation of its substrate p105/NF-κB1 and the degradation of IκBα (which is triggered by the IKK-catalysed phosphorylation of IκBα) were also prevented. The protein kinase TAK1 was partially inhibited by BAY 11-7082 in vitro (Supplementary Table S1), but BAY 11-7082 also suppressed the IL-1-stimulated activation of TBK1 in IL-1R cells (Figure 2C), which is dependent upon the expression of TRAF6, but independent of the expression or catalytic activity of TAK1 [22]. BAY 11-7082 additionally prevented the LPS- or IL-1-stimulated activation of JNK in RAW or IL-1R cells. BAY 11-7082 did not inhibit IRAK4 or IRAK1 in vitro (Supplementary Table S1), which are the most ‘upstream’ protein kinases in the MyD88 signalling network, and nor did it prevent the autophosphorylation of IRAK4 induced by LPS in RAW macrophages (Figure 3A) or IL-1 in IL-1R cells (Figure 3B). The suppression of K63-pUb chains and/or linear-pUb chains presumably explains how BAY 11-7082 prevents the activation of the IKK subfamily of protein kinases by LPS and IL-1.

Biochem J. 2013 May 1; 451(Pt 3): 427–437. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3685219/

In vivo activity

It is hypothesized that short-term in vivo topical application of NF-κB inhibitor BAY 11-7082 can prevent acidic bile-induced early preneoplastic molecular events, suggesting its potential role in disease prevention. Murine hypoharyngeal (HM) (C57Bl/6j wild-type) were topically exposed to a mixture of bile acids at pH 3.0 with and without BAY 11-7082 3 times/day for 7 days. Immunofluorescence, Western blotting, immunohistochemistry, quantitative polymerase chain reaction, and polymerase chain reaction microarrays were used to identify NF-κB activation and its associated oncogenic mRNA and miRNA phenotypes, in murine hypopharyngeal cells in vitro and in murine HM in vivo. Short-term exposure of HM to acidic bile is a potent stimulus accelerating the expression of NF-κB signaling (70 out of 84 genes) and oncogenic molecules. Topical application of BAY 11-7082 sufficiently blocks the effect of acidic bile. BAY 11-7082 eliminates NF-κB activation in regenerating basal cells of acidic bile-treated HM and prevents overexpression of molecules central to head and neck cancer, including bcl-2, STAT3, EGFR, TNF-α, and WNT5A. NF-κB inhibitor reverses the upregulated "oncomirs" miR-155 and miR-192 and the downregulated "tumor suppressors" miR-451a and miR-375 phenotypes in HM affected by acidic bile. There is novel evidence that acidic bile-induced NF-κB-related oncogenic mRNA and miRNA phenotypes are generated after short-term 7-day mucosal exposure and that topical mucosal application of BAY 11-7082 can prevent the acidic bile-induced molecular alterations associated with unregulated cell growth and proliferation of hypopharyngeal cells.

Neoplasia. 2018 Apr;20(4):374-386. https://pubmed.ncbi.nlm.nih.gov/29529473/

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.