

Product Data Sheet

Product Name: Bay 11-7085

Cat. No.: GC10345

Chemical Properties

Cas. No. 196309-76-9

Chemical Name (E)-3-(4-tert-butylphenyl)sulfonylprop-2-enenitrile

SMILES CC(C)(C)C1=CC=C(C=C1)S(=O)(=O)C=CC#N

Formula $C_{13}H_{15}NO_2S$ M.Wt 249.33

Solubility $\geq 12.45\text{mg/mL}$ in DMSO Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request.

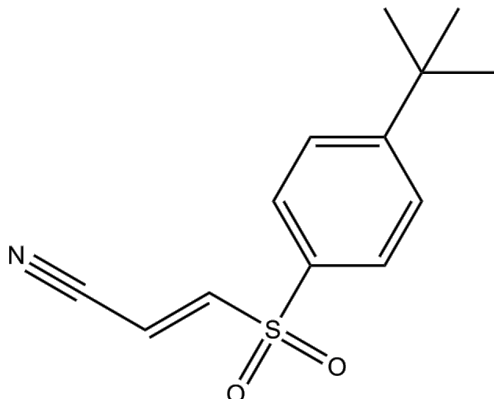
Caution: Product has not been fully validated for medical applications. For research use only.

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Structure



Protocol

Cell experiment [1]:

Cell lines Ramos B-lymphoma cells

Ramos B-lymphoma cells were grown in RPMI-1640 medium with 10% (v/v) fetal bovine serum (FBS), 0.2mM glutamine, 100U/ml penicillin, and 100µg/ml streptomycin at 37°C in 5% CO₂/atmosphere. Cells (2×10⁵ cells/ml) were

Preparation Method seeded onto 24-well plates and allowed to adhere in a 5% CO₂ incubator at 37°C overnight. Cells were treated with different concentrations of Bay 11-7085 (0, 2, 4, 6, 8, and 10µM) for 24h. Control cells were treated with DMSO at a final concentration of 0.1%. Cell viability was evaluated.

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Reaction
Conditions 0, 2, 4, 6, 8, and 10 μ M; 24h

Applications Bay 11-7085 treatment significantly reduced the cell viability of Ramos B-lymphoma cells in a dose-dependent manner.

Animal experiment [2]:

Animal models Female nu/nu athymic mice

Preparation Method Female nu/nu athymic mice (5 weeks old) were housed singly in a standard environment with food and water ad libitum. HT-29 cells were harvested in 0.25% trypsin-PBS-EDTA, washed once each in medium and PBS, and resuspended in PBS at 1×10^7 cells/200 μ l. 1×10^7 HT-29 cells were injected either s.c. in the backs or i.p. in 5-week-old female nu/nu athymic mice. For the mice with s.c. tumors, the tumors were allowed to establish themselves for 10 days after which the mice were randomized to BAY 11-7085 or DMSO. All of the mice received BAY 11-7085 (5mg/kg) or an equal volume of DMSO (200 μ l) twice weekly for 28 days. Tumor size was determined by measuring the length and width with calipers.

Dosage form 5mg/kg; twice a week for 28 days; i.p.

Applications BAY 11-7085 treatment significantly reduced the tumor volume in the HT-29 xenograft mouse model.

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References:

- [1] Berger N, Bassat H B, Klein B Y, et al. Cytotoxicity of NF- κ B inhibitors Bay 11-7085 and caffeic acid phenethyl ester to Ramos and other human B-lymphoma cell lines[J]. Experimental hematology, 2007, 35(10): 1495-1509.
- [2] Scaife C L, Kuang J, Wills J C, et al. Nuclear factor κ B inhibitors induce adhesion-dependent colon cancer apoptosis: implications for metastasis[J]. Cancer research, 2002, 62(23): 6870-6878.

Background

Bay 11-7085 is an inhibitor of NF- κ B activation and phosphorylation of I κ B α with an IC₅₀ value of 10 μ M for I κ B α [¹]. Bay 11-7085 inactivates protein tyrosine phosphatases (PTPs) by forming a covalent adduct with the active-site cysteine and inhibits the signal-induced nuclear translocation of NF κ B through inhibiting IKK activity and the phosphorylation of I κ B in response to the signal [²]. Bay 11-7085 has been widely used to inhibit and eradicate the formation of biofilms by various pathogens, and to prevent the growth of various pathogenic microorganisms[³].

In vitro, Bay 11-7085 treatment for 24 hours significantly inhibited the viability of Nalm6 cells and Raji cells, with IC₅₀ values of 0.5 μ M and 3.1 μ M, respectively[⁴]. Treatment with 10 μ M Bay 11-7085 for 2 hours induces an increase in LC3B-II in human synovial fibroblasts, phosphorylation of the glucocorticoid receptor (GR) at serine 211, and promotes autophagy of the cells[⁵]. Treatment with 4 μ M Bay 11-7085 for 24 hours promoted apoptosis in L1210 cells, accompanied by an increase in caspase-3 activity[⁶].

In vivo, Bay 11-7085 treatment via intraperitoneal injection (5mg/kg) every three days for a total of 9 days significantly reduced the tumor burden in the Su.86 xenograft mouse model [⁷]. Weekly intraperitoneal injection of Bay 11-7085 (5mg/kg) twice for 28 days significantly reduced the tumor volume and liver metastasis in the HT-29 xenograft mouse model[⁸].

References:

- [1] Pierce J W, Schoenleber R, Jesmok G, et al. Novel inhibitors of cytokine-induced I κ B α phosphorylation and endothelial cell adhesion molecule expression show anti-inflammatory effects in vivo[J]. Journal of Biological Chemistry, 1997, 272(34): 21096-21103.

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- [2] Krishnan N, Bencze G, Cohen P, et al. The anti-inflammatory compound BAY-11-7082 is a potent inhibitor of protein tyrosine phosphatases[J]. The FEBS journal, 2013, 280(12): 2830-2841.
- [3] Escobar I E, Possamai Rossatto F C, Kim S M, et al. Repurposing kinase Inhibitor Bay 11-7085 to combat Staphylococcus aureus and Candida albicans biofilms[J]. Frontiers in Pharmacology, 2021, 12: 675300.
- [4] Berger N, Bassat H B, Klein B Y, et al. Cytotoxicity of NF- κ B inhibitors Bay 11-7085 and caffeic acid phenethyl ester to Ramos and other human B-lymphoma cell lines[J]. Experimental hematology, 2007, 35(10): 1495-1509.
- [5] Relic B, Charlier E, Deroyer C, et al. BAY 11-7085 induces glucocorticoid receptor activation and autophagy that collaborate with apoptosis to induce human synovial fibroblast cell death[J]. Oncotarget, 2016, 7(17): 23370.
- [6] Cory A H, Cory J G. Induction of apoptosis in p53-deficient L1210 cells by an I- κ B- α -inhibitor (Bay 11-7085) via a NF- κ B-independent mechanism[J]. Advances in enzyme regulation, 2005, 45(1): 85-93.
- [7] Shen Y, Herde R, Doxey B W, et al. Pharmacologic downregulation of c-FLIPL restores juxtacrine death receptor-mediated apoptosis in cancer cells in a peritoneal carcinomatosis model[J]. International journal of cancer, 2012, 130(7): 1494-1503.
- [8] Scaife C L, Kuang J, Wills J C, et al. Nuclear factor κ B inhibitors induce adhesion-dependent colon cancer apoptosis: implications for metastasis[J]. Cancer research, 2002, 62(23): 6870-6878.

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