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	≥ 12.45mg/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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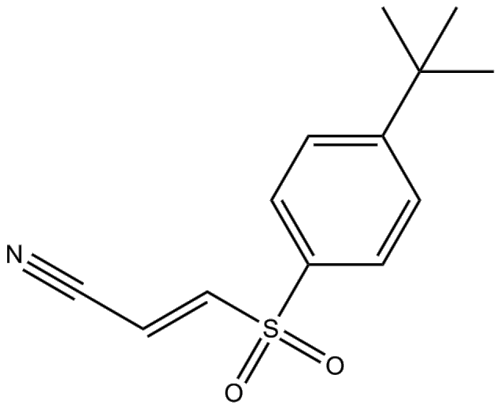
Structure



Protocol

Cell experiment [1]:

Cell lines	Ramos B-lymphoma cells
Preparation Method	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% CO_2/atmosphere. Cells (2×10^5 cells/ml) were seeded onto 24-well plates and allowed to adhere in a 5% CO_2 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 10μM; 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/200μl. 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 (200μl) 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-κB 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 κB 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-κB activation and phosphorylation of IκBα with an IC_{50} value of 10μM for IκBα[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 NFκB through inhibiting IKK activity and the phosphorylation of IκB 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 IC_{50} values of 0.5μM and 3.1μM, respectively[4]. Treatment with 10μM 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 4μM 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 IκBα 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-κB 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 κB inhibitors induce adhesion-dependent colon cancer apoptosis: implications for metastasis[J]. Cancer research, 2002, 62(23): 6870-6878.

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