

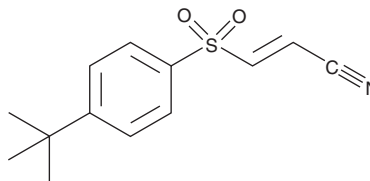
# PRODUCT INFORMATION



## BAY 11-7085

Item No. 14795

CAS Registry No.: 196309-76-9  
Formal Name: 3-[[4-(1,1-dimethylethyl)phenyl]sulfonyl]-2E-propenenitrile  
MF:  $C_{13}H_{15}NO_2S$   
FW: 249.3  
Purity:  $\geq 98\%$   
UV/Vis.:  $\lambda_{max}$ : 205, 252 nm  
Supplied as: A crystalline solid  
Storage:  $-20^{\circ}C$   
Stability:  $\geq 4$  years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

### Laboratory Procedures

BAY 11-7085 is supplied as a crystalline solid. A stock solution may be made by dissolving the BAY 11-7085 in the solvent of choice. BAY 11-7085 is soluble in organic solvents such as ethanol and DMSO, which should be purged with an inert gas. The solubility of BAY 11-7085 in these solvents is approximately 20 mg/ml.

BAY 11-7085 is sparingly soluble in aqueous solutions. To enhance aqueous solubility, dilute the organic solvent solution into aqueous buffers or isotonic saline. If performing biological experiments, ensure the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. We do not recommend storing the aqueous solution for more than one day.

### Description

In the classical pathway of NF- $\kappa$ B activation, phosphorylation of the inhibitor of NF- $\kappa$ B (I $\kappa$ B $\alpha$ ) releases the inhibitor from NF- $\kappa$ B, allowing I $\kappa$ B $\alpha$  degradation and NF- $\kappa$ B activation and nuclear import.<sup>1</sup> BAY 11-7085 is an irreversible inhibitor of I $\kappa$ B $\alpha$  phosphorylation, preventing activation of NF- $\kappa$ B by cytokines and lipopolysaccharide ( $IC_{50} = 10 \mu M$ ).<sup>2</sup> It blocks gene expression that is regulated through the classical pathway of NF- $\kappa$ B activation and in this way blocks apoptosis, cell adhesion, and inflammation.<sup>2-4</sup> BAY 11-7085 is also used to study I $\kappa$ B $\alpha$  actions that are independent of NF- $\kappa$ B signaling.<sup>5</sup>

### References

1. Wajant, H. and Scheurich, P. TNFR1-induced activation of the classical NF- $\kappa$ B pathway. *FEBS J.* **278**(6), 862-876 (2011).
2. Pierce, J.W., Schoenleber, R., Jesmok, G., *et al.* Novel inhibitors of cytokine-induced I $\kappa$ B $\alpha$  phosphorylation and endothelial cell adhesion molecule expression show anti-inflammatory effects *in vivo*. *J. Biol. Chem.* **272**(34), 21096-21103 (1997).
3. Scaife, C.L., Kuang, J., Wills, J.C., *et al.* Nuclear factor  $\kappa$ B inhibitors induce adhesion-dependent colon cancer apoptosis: Implications for metastasis. *Cancer Res.* **62**(23), 6870-6878 (2002).
4. Di Giuseppe, M., Gambelli, F., Hoyle, G.W., *et al.* Systemic inhibition of NF- $\kappa$ B activation protects from silicosis. *PLoS One* **4**(5), e5689 (2009).
5. Hu, X., Janssen, W.E., Moscinski, L.C., *et al.* An I $\kappa$ B $\alpha$  inhibitor causes leukemia cell death through a p38 MAP kinase-dependent, NF- $\kappa$ B-independent mechanism. *Cancer Res.* **61**(16), 6290-6296 (2001).

#### WARNING

THIS PRODUCT IS FOR RESEARCH ONLY - NOT FOR HUMAN OR VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

#### SAFETY DATA

This material should be considered hazardous until further information becomes available. Do not ingest, inhale, get in eyes, on skin, or on clothing. Wash thoroughly after handling. Before use, the user must review the complete Safety Data Sheet, which has been sent via email to your institution.

#### WARRANTY AND LIMITATION OF REMEDY

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