Store at -20C

TPCA-1

5 ma



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## **Background**

TPCA-1 blocks NF- $\kappa$ B signaling and suppresses the transcription of several pro-inflammatory cytokines through inhibition of the I $\kappa$ B kinase subunit IKK $\beta$  (IKK2). Studies show that TPCA-1 specifically inhibits IKK $\beta$  (IC<sub>50</sub> of 17.9 nM) and reduces inflammation and antigen-induced T cell proliferation in murine collagen-induced arthritis (1). TPCA-1 prevents recruitment of the transcription factor STAT3 to upstream kinases through interaction with the SH2 domain of STAT3. This limits the ability of cytokines and tyrosine kinases to induce STAT3 activity, which inhibits the ability of STAT3 to regulate cell growth and apoptosis. Treatment of non-small cell lung cancer (NSCLC) cells with TPCA-1 represses cell proliferation, suggesting that TPCA-1 may be effective in treating some forms of cancer (2). Studies have examined the ability of TPCA-1 to mediate the inflammatory response through control of IKK $\beta$  and STAT3 signaling in animal models of arthritis (1), chronic periodontitis (3), and nasal epithelial inflammation (4).

Molecular FormulaC12H10FN3O2SMolecular Weight279.3 g/mol

Purity >98%

CAS 507475-17-4

**Soluble** in DMSO at 30 mg/mL.

**Storage** Store lyophilized at -20°C, desiccated. In lyophilized form, the chemical is stable for 24 months. Once in

solution, store at -20°C and use within 3 months to prevent loss of potency. *Aliquot to avoid multiple* 

freeze/thaw cycles.

**Directions for Use** TPCA-1 is supplied as a lyophilized powder. For a 15 mM stock, reconstitute 5 mg of powder in 1.19 mL

of DMSO. Working concentrations and length of treatment can vary depending on the desired effect.

Background References 1. Podolin, P.L. et al. (2005) J Pharmacol Exp Ther 312, 373-81.

2. Nan, J. et al. (2014) *Mol Cancer Ther* 13, 617-29.

3. Wang, B. et al. (2021) *Mol Oral Microbiol* 36, 192-201.

4. Sachse, F. et al. (2011) Rhinology 49, 168-73.

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