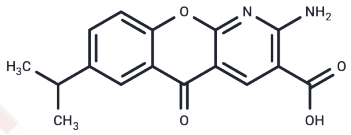
Data Sheet (Cat.No.T1639)

TargetMol

Amlexanox

Chemical Properties



CAS No. : 68302-57-8

Formula: C16H14N2O4

Molecular Weight: 298.29

Appearance: no data available

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year

Biological Description

Description	Amlexanox (AA673) is an anti-aphthous ulcer drug. Amlexanox inhibits the synthesis and release of inflammatory mediators, including leukotrienes and histamine, from mast cells, neutrophils, and mononuclear cells. Amlexanox also acts as a leukotriene D4 antagonist and a phosphodiesterase inhibitor. Amlexanox decreases the time ulcers take to heal as well as the pain associated with the ulcers.
Targets(IC50)	FGFR,IL Receptor,IκB/IKK
In vitro	AmLexanox increases phosphorylation of TBK1 on Ser172 in 3T3-L1 adipocytes, and blocks polyinosinic:polycytidylic acid (poly I:C)-stimulated phosphorylation of interferon responsive factor-3 (IRF3), a presumed substrate of IKKε and TBK1[1]. AmLexanox potently inhibits the release of histamine and leukotrienes from mast cells, basophils and neutrophils in in vitro settings, possibly through increasing intracellular cyclic AMP content in inflammatory cells, a mem-brane-stabilising effect or inhibition of calcium influx[2]. In primary bone marrow derived macrophages (BMMs), amLexanox inhibits osteoclast formation and bone resorption. At the molecular level, amLexanox suppresses RANKL-induced activation of nuclear factor-κB (NF-κB), mitogen-activated protein kinase (MAPKs), c-Fos and NFATc1. AmLexanox decreases the expression of osteoclast-specific genes, including TRAP, MMP9, Cathepsin K and NFATc1[3].
In vivo	AmLexanox (100 mg/kg, p.o.) prevents and reverses diet-induced or genetic obesity, and produces reversible weight loss in obese mice. AmLexanox also causes a significant decrease in adipose tissue mass in these mice, and an increase in circulating adiponectin. AmLexanox (25 mg/kg) significantly improves insulin sensitivity in mice with established DIO,and after four weeks of treatment, amLexanox produces marked improvements in glucose[1]. AmLexanox before the first application of the paste and at each has been shown to suppress both immediate and evaluation thereafter. A categorical scale is also delayed-type hypersensitivity reactions[2]. AmLexanox (20? mg/kg) enhances osteoblast differentiation of BMSCs. In ovariectomized (OVX) mouse model, amLexanox prevents OVX-induced bone loss by suppressing osteoclast activity [3].
Kinase Assay	The in vitro kinase assays is performed by incubating purified kinase (IKKε or TBK1) in kinase buffer containing 25 mM Tris (pH7.5), 10 mM MgCl2, 1 mM DTT, and 10 μM ATP for 30 minutes at 30°C in the presence of 0.5 μCi γ-[32P]-ATP and 1 μg MBP per sample as a substrate. The kinase reaction is stopped by adding 4x sodium dodecyl sulfate (SDS) sample buffer and boiling for 5 minutes at 95°C. Supernatants are resolved by SDS-

	polyacrylamide gel electrophoresis, transferred to nitrocellulose, and analyzed by autoradiography using a Typhoon 9410 phosphorimager.
Cell Research	To examine cell proliferation, a Cell Counting Kit-8 is used according to the manufacturer's instructions. BMMs are seeded at a density of 5×103 cells/well in 96-well plates. After 24?hours, cells are treated with different concentrations of AmLexanox (0, 1.5, 3, 6, 12, 25?μM) every 2 days in the presence of M-CSF (30?ng/mL) for 7 days. After 1, 3, 5 and 7 days, the culture medium is replaced by the medium containing 10% CCK-8 and cells are incubated at 37°C for an additional 2?h. The absorbance is then measured at a wavelength of 450?nm on an ELX800 absorbance microplate reader.

Solubility Information

Solubility	DMSO: 50 mg/mL (167.62 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	3.3524 mL	16.7622 mL	33.5244 mL
5 mM	0.6705 mL	3.3524 mL	6.7049 mL
10 mM	0.3352 mL	1.6762 mL	3.3524 mL
50 mM	0.067 mL	0.3352 mL	0.6705 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Reference

Reilly SM, et al. An inhibitor of the protein kinases TBK1 and IKK-e improves obesity-related metabolic dysfunctions in mice. Nat Med. 2013 Mar;19(3):313-21.
Cheng C, Ji Z, Sheng Y, et al. Aphthous ulcer drug inhibits prostate tumor metastasis by targeting IKK/TBK1/NF-κB signaling. Theranostics. 2018, 8(17): 4633
Bell, J. AmLexanox for the treatment of recurrent aphthous ulcers. Clin Drug Investig, 2005. 25(9): p. 555-66.
Zhang Y, et al. AmLexanox Suppresses Osteoclastogenesis and Prevents Ovariectomy-Induced Bone Loss. Sci Rep. 2015 Sep 4;5:13575.
Cheng, Chaping, et al. Aphthous ulcer drug inhibits prostate tumor metastasis by targeting IKK/TBK1/NF-κB signaling [J]. Theranostics. 2018 Sep 9;8(17):4633-4648.