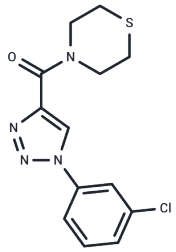
# Data Sheet (Cat.No.T8570)

TargetMol

L524-0366

## Chemical Properties



CAS No. : 951612-19-4

Formula: C13H13ClN4OS

Molecular Weight: 308.79

Storage: Keep away from direct sunlight
Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

## Biological Description

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| Description | L524-0366 is a selective Fn14 receptor antagonist that exerts its effects through competitive binding to the Fn14 receptor. L524-0366 specifically binds to the Fn14 receptor surface, with a KD of 7.12 μM. At a concentration of 10 μM, L524-0366 completely inhibits TWEAK-induced glial cell migration and exhibits no cytotoxicity at concentrations below 50 μM. L524-0366 possesses antitumor and anti-inflammatory activity and is widely used in research on diseases such as cancer. |
| Targets(IC50) | Others,TNF |
| In vitro | L524-0366 mitigated maladaptive remodeling with TAC. TWEAK induced secretion of the pro-inflammatory chemokine, monocyte chemoattractant protein 1 from WT but not Fn14-/-?fibroblasts in vitro, in part through activation of non-canonical NF-κB signaling. Finally, Fn14 expression was increased in mouse following TAC and in human failing hearts. |
| In vivo | **Methods**: Aortic stenosis surgery was performed on 8- to 12-week-old wild-type (WT) and Fn14 knockout mice to induce chronic pressure overload. Starting 3 days post-surgery, daily intraperitoneal injections of the Fn14 antagonist L524-0366 (9 mg/kg/day, dissolved in corn oil) were administered until 6 weeks post-surgery.<br>**Results**: L524-0366 treatment significantly improved TAC-induced cardiac dysfunction, reduced cardiac fibrosis, and decreased macrophage infiltration. [1] |
| Cell Research | Wild type (WT) and Fn14 knock out (Fn14-/-) mice were subjected to pressure overload [transaortic constriction (TAC)] for 1 or 6 weeks. A subset of WT TAC animals were treated with the Fn14 antagonist L524-0366. Cardiac function was measured by echocardiography. Cardiac fibrosis and macrophage infiltration were quantified using immunohistochemistry and flow cytometry, respectively. Cardiac fibroblasts were isolated for quantifying TWEAK-induced chemokine release. |

## Solubility Information

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| Solubility | DMSO: 25 mg/mL (80.96 mM),Sonication is recommended.<br>(< 1 mg/ml refers to the product slightly soluble or insoluble) |

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|---|---|
| In vivo Formulation | 5% DMSO+95% Corn oil: 1 mg/mL (3.24 mM),Suspension.<br>*Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.* |

Preparing Stock Solutions

| | 1mg | 5mg | 10mg |
|---|---|---|---|
| 1 mM | 3.2384 mL | 16.1922 mL | 32.3845 mL |
| 5 mM | 0.6477 mL | 3.2384 mL | 6.4769 mL |
| 10 mM | 0.3238 mL | 1.6192 mL | 3.2384 mL |
| 50 mM | 0.0648 mL | 0.3238 mL | 0.6477 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.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Unudurthi SD, et al. Fibroblast growth factor-inducible 14 mediates macrophage infiltration in heart to promote pressure overload-induced cardiac dysfunction. Life Sci. 2020 Apr 15;247:117440.
Li Z, Wang H, Zhu J, et al. Inhibition of TWEAK/Tnfrsf12a axis protects against acute liver failure by suppressing RIPK1-dependent apoptosis. Cell Death Discovery. 2022, 8(1): 1-11
Liu L, Wu P, Wei Y, et al.TWEAK-Fn14 signaling protects mice from pulmonary fibrosis by inhibiting fibroblast activation and recruiting pro-regenerative macrophages.Cell Reports.2025, 44(2).