

Product Data Sheet

Product Name: TKL002
Cat. No.: GC79806

Chemical Properties

Cas. No. 3098518-95-4

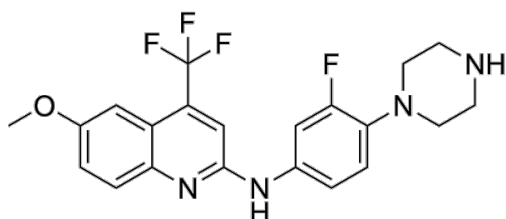
Formula $C_{21}H_{20}F_4N_4O$ M.Wt 420.4

Solubility DMSO: 10 mg/mL (23.79 mM; ultrasonic and warming and heat to 60°C) 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.

Structure



Background

TKL002 is a blood-brain barrier-permeable inhibitor of the CTH/H 2 S/NF-κB/EMT signaling axis. TKL002 induces G 2 /M phase cell cycle arrest and apoptosis in glioblastoma cells. TKL002 inhibits the migration and invasion of glioblastoma cells by upregulating E-cadherin and downregulating N-cadherin and vimentin. TKL002 is applicable to relevant research on glioblastoma [1].

In Vivo, TKL002 (5-20 mg/kg; i.p.; once every other day, 7 total administrations) inhibits the growth of

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Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA

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subcutaneous glioblastoma xenografts in a dose-dependent manner^[1]. TKL002 (10-20 mg/kg; i.v., once every three days for a total of 5 administrations) can cross the blood-brain barrier, inhibit the growth of orthotopic glioblastoma in a dose-dependent manner, reduce tumor burden, maintain the physical condition of mice, and cause no obvious organ toxicity^[1].

In Vitro, TKL002 (0.625-10 $\mu\text{mol/L}$; 24-72 h) potently inhibits the proliferation of U87MG, U118MG, and U251MG glioblastoma cells in a dose- and time-dependent manner with selectivity over normal LX2 cells, exhibiting IC_{50} values ranging from 4.141 to 6.506 $\mu\text{mol/L}$ across cell lines and time points^[1]. TKL002 (6 $\mu\text{mol/L}$; 48 h) induces late apoptosis in U87MG glioblastoma cells, mediated via upregulation of Bax and caspase-3 and downregulation of Bcl-2 and active-caspase-3^[1]. TKL002 (2-6 $\mu\text{mol/L}$; 24 h) induces G2/M phase cell cycle arrest in U87MG and U118MG glioblastoma cells in a concentration-dependent manner, with maximum G2/M fractions of 22.68% and 30.31% at 6 $\mu\text{mol/L}$ respectively^[1]. TKL002 (2-6 $\mu\text{mol/L}$; 48 h) reduces CTH, cysteine, and H₂S levels, increases GSH levels, and inhibits NF- κ B signaling and pro-inflammatory cytokine expression in U87MG and U118MG glioblastoma cells in vitro in a dose-dependent manner^[1]. TKL002 (2-6 $\mu\text{mol/L}$; 48 h) inhibits migration and invasion of U87MG and U118MG glioblastoma cells in a dose-dependent manner, mediated via upregulation of E-cadherin and downregulation of N-cadherin and vimentin to suppress epithelial-mesenchymal transition^[1].

References:

[1]. Luo Z, et al. Hijacking the Hydrogen Sulfide Axis: A Novel 4-Trifluoromethylquinoline Derivative Suppresses Glioblastoma via Cystathionine γ -Lyase Suppression. J Med Chem. 2026;69(3):3457-3476.

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