# GGTI298 Trifluoroacetate

**Cat. No.:** HY-15871

**CAS No.:** 1217457-86-7

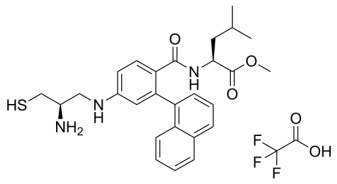
**Molecular Formula:** $C_{29}H_{34}F_3N_3O_5S$

**Molecular Weight:** 593.66

**Target:** Ras; Apoptosis

**Pathway:** GPCR/G Protein; Apoptosis

**Storage:** 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



## SOLVENT & SOLUBILITY

**In Vitro**

DMSO : 100 mg/mL (168.45 mM; Need ultrasonic)

| Preparing Stock Solutions | Concentration / Solvent / Mass | 1 mg | 5 mg | 10 mg |
|---|---|---|---|---|
| | 1 mM | 1.6845 mL | 8.4223 mL | 16.8447 mL |
| | 5 mM | 0.3369 mL | 1.6845 mL | 3.3689 mL |
| | 10 mM | 0.1684 mL | 0.8422 mL | 1.6845 mL |

Please refer to the solubility information to select the appropriate solvent.

**In Vivo**

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: 2.5 mg/mL (4.21 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (4.21 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.21 mM); Clear solution

## BIOLOGICAL ACTIVITY

**Description**

GGTI298 Trifluoroacetate is a CAAZ peptidomimetic geranylgeranyltransferase I (GGTase I) inhibitor, which can inhibit Rap1A with $IC_{50}$ of 3 μM; little effect on Ha-Ras with $IC_{50}$ of >20 μM.

**$IC_{50}$ & Target**

IC50: 3 μM (Rap1A, in vivo), > 20 μM (Ha-Ras, in vivo)[3]

**In Vitro**

RhoA inhibitor (GGTI298 Trifluoroacetate) significantly reduces cAMP agonist-stimulated apical K+ conductance[1]. Knockdown of DR4 abolishes NF-κB activation, leading to sensitization of DR5-dependent apoptosis induced by the combination of GGTI298 Trifluoroacetate and TRAIL. GGTI298 Trifluoroacetate/TRAIL activates NF-κB and inhibits Akt. Knockdown of DR5, prevents GGTI298/TRAIL-induced IκBα and p-Akt reduction, suggesting that DR5 mediates reduction of I

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|---|---|
| | κBα and p-Akt induced by GGTI298/TRAIL. In contrast, DR4 knockdown further facilitates GGTI298/TRAIL-induced p-Akt reduction[2].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **In Vivo** | The vivo mouse ileal loop experiments show fluid accumulation is reduced in a dose-dependent manner by TRAM-34, GGTI298 Trifluoroacetate, or H1152 when inject together with cholera toxin into the loop[1].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

## PROTOCOL

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|---|---|
| **Kinase Assay** [2] | The given cells are lysed with reporter lysis buffer and subjected to luciferase activity assay using luciferase assay system in a luminometer. Relative luciferase activity is normalized to protein content[2].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **Cell Assay** [2] | Cells are seeded in 96-well cell culture plates and treated the next day with the agents (including GGTI298 Trifluoroacetate). The viable cell number is determined using the sulforhodamine B assay[2].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **Animal Administration** [1] | The ileal loop experiment is performed in 6-8-week-old mice by a modifing rabbit ileal loop assay. Following gut sterilization, the animals are kept fasted for 24 h prior to surgery and fed only water ad libitum. Anesthesia is induced by a mixture of ketamine (35 mg/kg of body weight) and xylazine (5 mg/kg of body weight). A laparotomy is performed, and the experimental loops of 5-cm length are constricted at the terminal ileum by tying with non-absorbable silk. The following fluids are instilled in each loop by means of a tuberculin syringe fitting with a disposable needle through the ligated end of the loop: pure CT (1 μg; positive control), saline (negative control), CT (1 μg)+TRAM-34 (different concentrations in μM), CT (1 μg)+ H1152 (1 μM), and CT (1 μg)+GGTI298 Trifluoroacetate (different concentrations in μM), a specific inhibitor of Rap1A. The intestine is returned to the peritoneum, and the mice are sutured and returned to their cages. After 6 h, these animals are sacrificed by cervical dislocation, and the loops are excised[1].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

## CUSTOMER VALIDATION

- Mol Cell Proteomics. 2023 Jun 14;100593.
- Int Immunopharmacol. 2023 Mar 15;117:110014.

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## REFERENCES


[1]. Sheikh IA, et al. The Epac1 signaling pathway regulates Cl- secretion via modulation of apical KCNN4c channels in diarrhea. J Biol Chem. 2013 Jul 12;288(28):20404-15.

[2]. Chen S, et al. Dissecting the roles of DR4, DR5 and c-FLIP in the regulation of geranylgeranyltransferase I inhibition-mediated augmentation of TRAIL-induced apoptosis. Mol Cancer. 2010 Jan 29;9:23.

[3]. McGuire TF, et al. Platelet-derived growth factor receptor tyrosine phosphorylation requires protein geranylgeranylation but not farnesylation. J Biol Chem. 1996 Nov 1;271(44):27402-7.

**Caution: Product has not been fully validated for medical applications. For research use only.**

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