

Product data sheet



MedKoo Cat#: 563409 Name: Thiamet G CAS#: 1009816-48-1 Chemical Formula: C ₉ H ₁₆ N ₂ O ₄ S Exact Mass: 248.0831 Molecular Weight: 248.29		
Product supplied as:	Powder	
Purity (by HPLC):	≥ 98%	
Shipping conditions	Ambient temperature	
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years. In solvent: -80°C 3 months; -20°C 2 weeks.	

1. Product description:

Thiamet G is a potent and selective inhibitor of O-GlcNAcase.

2. CoA, QC data, SDS, and handling instruction

SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under “QC And Documents” section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	32.0	128.99
H2O	50.0	201.55

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	4.03 mL	20.14 mL	40.28 mL
5 mM	0.81 mL	4.03 mL	8.06 mL
10 mM	0.40 mL	2.01 mL	4.03 mL
50 mM	0.08 mL	0.40 mL	0.81 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator

Please refer the product web page under section of “Calculator”

6. Recommended literature which reported protocols for in vitro and in vivo study

In vitro study

1. Takeuchi T, Horimoto Y, Oyama M, Nakatani S, Kobata K, Tamura M, Arata Y, Hatanaka T. Osteoclast Differentiation Is Suppressed by Increased O-GlcNAcylation Due to Thiamet G Treatment. *Biol Pharm Bull.* 2020;43(10):1501-1505. doi: 10.1248/bpb.b20-00221. PMID: 32999159.
2. He Y, Ma X, Li D, Hao J. Thiamet G mediates neuroprotection in experimental stroke by modulating microglia/macrophage polarization and inhibiting NF-κB p65 signaling. *J Cereb Blood Flow Metab.* 2017 Aug;37(8):2938-2951. doi: 10.1177/0271678X16679671. Epub 2016 Jan 1. PMID: 27864466; PMCID: PMC5536801.

In vivo study

1. He Y, Ma X, Li D, Hao J. Thiamet G mediates neuroprotection in experimental stroke by modulating microglia/macrophage polarization and inhibiting NF-κB p65 signaling. *J Cereb Blood Flow Metab.* 2017 Aug;37(8):2938-2951. doi: 10.1177/0271678X16679671. Epub 2016 Jan 1. PMID: 27864466; PMCID: PMC5536801.
2. Jiang M, Yu S, Yu Z, Sheng H, Li Y, Liu S, Warner DS, Paschen W, Yang W. XBP1 (X-Box-Binding Protein-1)-Dependent O-GlcNAcylation Is Neuroprotective in Ischemic Stroke in Young Mice and Its Impairment in Aged Mice Is Rescued by Thiamet-G. *Stroke.* 2017 Jun;48(6):1646-1654. doi: 10.1161/STROKEAHA.117.016579. Epub 2017 May 9. PMID: 28487326; PMCID: PMC5493893.

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7. Bioactivity

Biological target:

Thiamet G is a potent and selective inhibitor of O-GlcNAcase (OGA), which acts to remove O-GlcNAc from modified proteins, with K_i of 20 nM for human OGA.

In vitro activity

To further confirm the effect of TMG (Thiamet G) on polarization of microglia, a polarization experiment was carried out in BV2 cells with or without TMG. BV2 cells were cultivated in medium containing M1 or M2 polarization cytokines in the presence of TMG. After 12 h, no significant change of M2 was observed in TMG-treated groups compared with the MCAO group, although M1 polarization was suppressed by TMG (Figure 5(a) to (d)). These results provide further evidence to assert that TMG influences microglial polarization. The transcriptional activity of NF- κ B was also examined during polarization. The data showed that NF- κ B was suppressed along with the decreased M1 phenotype triggered by TMG treatment (Figure 5(e) to (h)). After intervention with TMG, less p65 translocated into nuclei even when the amount of p65 in the cytoplasm did not change dramatically, which indicated that the transcriptional activity of p65 was suppressed by TMG (Figure 5(e) and (g), cytoplasmic: 0.52 ± 0.02 in the TMG group vs. 0.45 ± 0.01 in the Control group, ns: not significant, $n = 9$ per group; Figure 5(f) and (h), nuclear: 0.81 ± 0.06 in TMG group vs. 1.33 ± 0.01 in the Control group, $P < 0.05$, $n = 9$ per group). The implication was that TMG may shift the polarization of microglia/macrophages by inhibiting NF- κ B activation.

Reference: J Cereb Blood Flow Metab. 2017 Aug; 37(8): 2938–2951. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5536801/>

In vivo activity

To assess the effect of TMG (Thiamet G) on the brain injury after focal ischemia in mice, infarct volumes and neurological deficit scores were quantified at several doses and multiple time points after MCAO. Higher doses (10–40 mg/kg) significantly reduced infarct volumes (Figure 1(a)). These decreases of infarct volume reached significance at 72 h after I/R injury, although the decreased mNSS reached significance at 24 h (Supplemental Figure 1). The following experiments were performed at a dose of 20 mg/kg TMG, and infarct volume was evaluated at 72 h after MCAO. To evaluate the effect of TMG on neuronal function of mice after MCAO, a variety of behavior function tests were executed. All these tests were performed at 24 h after MCAO. Results from mNSS testing exemplified a dramatic change in the MCAO group compared to both TMG-treated groups: 9 (6.5–10.5) in the preventative treatment group vs. 13 (10.5–13.5) in the MCAO group, $P < 0.05$, $n = 9$ per group; 6 (4.5–8) in the therapeutic treatment group vs. 13 (10.5–13.5) in MCAOs, $P < 0.01$, $n = 9$ per group (Figure 1(d)). Further functional analyses with the foot-fault test, adhesion-removal test, and inclined plane test also indicated markedly improved outcomes after treatment with TMG in MCAO mice (Figure 1(e) to (g)). Together, these results support the presumption that treatment with TMG is neuroprotective in MCAO mice. Results shown in Figure 1(b) and (c) indicate that treatment with TMG before ischemic injury significantly reduced the infarct size (37.00 ± 0.60 mm³ in the preventative treatment group vs. 52.44 ± 1.00 mm³ in the MCAO group, $P < 0.05$, $n = 9$ per group). The therapeutic treatment group yielded virtually the same result (38.11 ± 1.60 mm³ vs. 52.44 ± 1.00 mm³ in the MCAO group, $P < 0.05$, $n = 9$ per group).

Reference: J Cereb Blood Flow Metab. 2017 Aug; 37(8): 2938–2951. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5536801/>

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.