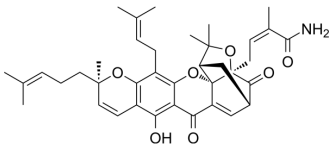
Gambogic amide

Cat. No.:	HY-121833
CAS No.:	286935-60-2
Molecular Formula:	$C_{38}H_{45}NO_7$
Molecular Weight:	627.77
Target:	Trk Receptor; Akt; ERK
Pathway:	Neuronal Signaling; Protein Tyrosine Kinase/RTK; PI3K/Akt/mTOR; MAPK/ERK Pathway; Stem Cell/Wnt



Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month

BIOLOGICAL ACTIVITY

Description

Gambogic amide is a potent and selective agonist of TrkA and also induces its tyrosine phosphorylation and activation of downstream signaling, including Akt and MAPK. Gambogic amide specifically interacts with the cytoplasmic juxtamembrane domain of the TrkA receptor and triggers its dimerization, leading to activation. Gambogic amide has neuroprotective activity preventing glutamate-induced neuronal cell death. Gambogic amide has improved efficacy in a transient middle cerebral artery occlusion model of stroke and could be used to study neurodegenerative diseases and stroke[1].

In Vitro

Gambogic amide (0.5 μM; 30 min) elicits TrkA tyrosine phosphorylation in hippocampal neurons[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	Hippocampal neurons
Concentration:	500 nM
Incubation Time:	30 min
Result:	Triggered TrkA Y490 phosphorylation.

In Vivo

Gambogic amide (2 mg/kg; sc; single dose) prevents neuronal cell death and decreases infarct volume in MCAO animal model[1].

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Animal Model:	MCAO mice model[1]
Dosage:	2 mg/kg
Administration:	s.c.; 5 min before the onset of reperfusion, after 2 h of MCAO followed by reperfusion; followed by 25 mg/kg Kainic acid (KA)
Result:	Diminished Kainic acid-triggered hippocampal neuronal cell death.

	Reduces infarct volume in MCAO rat brain.

REFERENCES

[1]. Jang SW, et al. Gambogic amide, a selective agonist for TrkA receptor that possesses robust neurotrophic activity, prevents neuronal cell death. Proc Natl Acad Sci U S A. 2007 Oct 9;104(41):16329-34.

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