Cyclo(his-pro)

Cat. No.: HY-101402

CAS No.: 53109-32-3

Molecular Formula: $C_{11}H_{14}N_4O_2$

Molecular Weight: 234.25

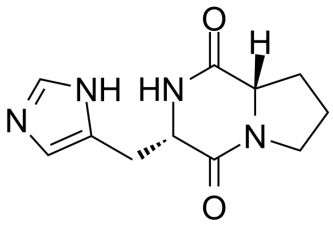
Sequence Shortening: Cyclo(HP)

Target: NF-κB; Endogenous Metabolite

Pathway: NF-κB; Metabolic Enzyme/Protease

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 30 mg/mL (128.07 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.2689 mL	21.3447 mL	42.6894 mL
5 mM	0.8538 mL	4.2689 mL	8.5379 mL
10 mM	0.4269 mL	2.1345 mL	4.2689 mL

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

BIOLOGICAL ACTIVITY

Description

Cyclo(his-pro) (Cyclo(histidyl-proline)) is an orally active cyclic dipeptide structurally related to tyreotropin-releasing hormone[1]. Cyclo(his-pro) could inhibit NF-κB nuclear accumulation. Cyclo(his-pro) can cross the brain-blood-barrier and affect diverse inflammatory and stress responses[2].

IC_{50} & Target		
	NF-κB	Human Endogenous Metabolite

In Vitro

cyclo(his-pro) (Cyclo(histidyl-proline); 50 μM; 1-48 hours) increases the nuclear level of Nrf2 and inhibits NF-κB nuclear translocation. Cyclo(His-Pro) alone has no effect on nuclear translocation of these transcription factors[2].

cyclo(his-pro) (50 μM; prior to PQ exposure for 48 hours) abolishes protein nitration that followed paraquat (PQ) exposure and lessenes its functional consequences, as shown by decrease in cell apoptosis, detected by caspase 3 activity and by cytochrome c release[2].

Cyclo(his-pro) inhibits NF-κB nuclear accumulation induced by paraquat in rat pheochromocytoma PC12 cells via the Nrf2/heme oxygenase-1 pathway[2].

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

Western Blot Analysis[1]

Cell Line:	PC12 cells
Concentration:	50 μM
Incubation Time:	1, 2, 4, 8, 24, 48 hours
Result:	Increased the nuclear level of Nrf2 and inhibited NF-κB nuclear translocation.

In Vivo

Cyclo(his-pro) (Cyclo(histidyl-proline); 1.8 mg/ear; topical application on the right ear; 30 min prior to TPA) reduces TPA-induced ear oedema confirming that it can exert anti-inflammatory effect[2].

Cyclo(his-pro) exerts in vivo anti-inflammatory effects in the central nervous system by down-regulating hepatic and cerebral TNFα expression thereby counteracting LPS-induced gliosis. Moreover, by up-regulating Bip, Cyclo(his-pro) increases the ER stress sensitivity andtriggers the unfolded protein response to alleviate the ER stress[3].

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

Animal Model:	Sixty two/three month-old male C57BL/6 mice (25-30 g)[2]
Dosage:	1.8 mg/ear
Administration:	Topical application on the right ear; 30 min prior to TPA
Result:	Reduced TPA-induced ear oedema.

REFERENCES

[1]. Grottelli S, et al. The Role of Cyclo(His-Pro) in Neurodegeneration. Int J Mol Sci. 2016 Aug 12;17(8). pii: E1332.

[2]. Minelli A, et al. Cyclo(His-Pro) exerts anti-inflammatory effects by modulating NF-κB and Nrf2 signalling. Int J Biochem Cell Biol. 2012 Mar;44(3):525-35.

[3]. Bellezza I, et al. Neuroinflammation and endoplasmic reticulum stress are coregulated by cyclo(His-Pro) to prevent LPS neurotoxicity. Int J Biochem Cell Biol. 2014 Jun;51:159-69.

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