Product Data Sheet

Product Name: Thrombin (MW 37kDa)
Cat. No.: GC61495

Chemical Properties

Cas. No.	9002-04-4	
SMILES	[Thrombin]	
Formula	M.Wt	37000(Average)
Solubility	Storage	4°C, protect from light
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ 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	Thrombin (MW 37kDa)	

Protocol

Cell experiment [1]:

Cell lines	N27 cells derived from E12 rat mesencephalic tissue
Preparation Method	AA and lipid hydroperoxides content was assayed from the N27 cells 24 h after thrombin (0.5 U/mL) treatment.
Reaction Conditions	24 h ,thrombin (0.5 U/ml)

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Applications	Thrombin dose-dependently induced N27 neuronal cell death, which accompanied elevated AA and lipid hydroperoxides, and smaller mitochondria with increased membrane density. Thrombin did not affect the intracellular level of ferrous iron

Animal experiment [2]:

Animal models	Male Sprague-Dawley rats, each weighing between 250 and 350 g
Preparation Method	Solutions containing 10 U of thrombin in 10 μl saline were infused into the brain over a period of 1 minute using a Harvard pump.
Dosage form	10 U of thrombin in 10 μl saline for 1 minute
Applications	AIB(a marker of BBB opening) has increased BBB permeability. Thrombin causes disruption of the BBB in the ipsilateral hemisphere

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References:

[1]. Tuo QZ, Liu Y, et,al. Thrombin induces ACSL4-dependent ferroptosis during cerebral ischemia/reperfusion. Signal Transduct Target Ther. 2022 Feb 23;7(1):59. doi: 10.1038/s41392-022-00917-z. PMID: 35197442; PMCID: PMC8866433.

[2]. Lee KR, Kawai N, et,al. Mechanisms of edema formation after intracerebral hemorrhage: effects of thrombin on cerebral blood flow, blood-brain barrier permeability, and cell survival in a rat model. J Neurosurg. 1997 Feb;86(2):272-8. doi: 10.3171/jns.1997.86.2.0272. PMID: 9010429.

Background

Thrombin (MW 37kDa) is a Na+-activated, allosteric serine protease. Thrombin induces the activation of ERK1 and ERK2[1]. Thrombin recognition sequence and can be used to digest GST-tagged proteins[2].Thrombin could activate the immune system by directly cleaving pro-interleukin-1α to the active form (IL-1α) [3]. Thrombin may be “a gas pedal” driving the innate immune system[4].

Thrombin induces neuronal ferroptosis, in N27 cells, Thrombin dose-dependently induced N27 neuronal cell death, which accompanied elevated AA and lipid hydroperoxides, and smaller mitochondria with increased membrane density. Thrombin did not affect the intracellular level of ferrous iron[5]. Thrombin elicits rapid and full activation of cPLA2 not only by promoting a rise in cytosolic free Ca^{2+} but also by inducing phosphorylation of cPLA2 thereby improving its catalytic activity[6].Low concentrations of thrombin and thrombin preconditioning yield the potential of rescuing cells and to induce survival of neurons and astrocytes exposed to various ischemic insults[7].Thrombin also has roles in brain cell death and survival as well as

neuroinflammation, predominantly via the cellular protease-activated receptor (PAR) activation and downstream signaling pathways [5]

In vivo□thrombin induces BBB disruption as well as death of parenchymal cells, whereas CBF and vasoreactivity are not altered. Cell toxicity and BBB disruption by thrombin are triggering mechanisms for the edema formation that follows intracerebral hemorrhage[9].In the primary hemostatic processis. Thrombin activates platelets, and also in the secondary hemostasis, it mediates the conversion of fibrinogen to fibrin. Thus, thrombin contributes to thrombus formation that stops bleeding and hematoma formation after blood enters the brain parenchyma [10]. Intracerebral administration of exogenous thrombin (at a dose that is non-toxic to normal brain), markedly exacerbated brain edema after transient focal cerebral ischemia. Extravascular thrombin inhibition may be a new therapeutic target for cerebral ischemia[6]

References:
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[5]. Tuo QZ, Liu Y, et,al. Thrombin induces ACSL4-dependent ferroptosis during cerebral ischemia/reperfusion. Signal Transduct Target Ther. 2022 Feb 23;7(1):59. doi: 10.1038/s41392-022-00917-z. PMID: 35197442; PMCID: PMC8866433.
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