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## Psalmotoxin 1 TFA

Cat. No.:	HY-P1411A	
Molecular Formula:	C <sub>200</sub> H <sub>312</sub> N <sub>62</sub> O <sub>57</sub> S <sub>6</sub> .xC <sub>2</sub> HF <sub>3</sub> O <sub>2</sub>	Glu-Asp-Cys-Ile-Pro-Lys-Trp-Lys-Gly-
Sequence:	Glu-Asp-Cys-Ile-Pro-Lys-Trp-Lys-Gly-Cys-Val-Asn-Arg-His-Gly-Asp-Cys-Cys-Glu-Gly-Leu Cys-Val-Asn-Arg-His-Gly-Asp-Cys-Cys- -Glu-Cys-Trp-Lys-Arg-Arg-Ser-Phe-Glu-Val-Cys-Val-Pro-Lys-Thr-Pro-Lys-Thr (Disulfide bridge: Cys <sub>3</sub> -Cys <sub>18</sub> , Cys <sub>10</sub> -Cys <sub>23</sub> , Cys <sub>17</sub> -Cys <sub>33</sub> )	Glu-Gly-Leu-Glu-Cys-Trp-Lys-Arg-Arg-Ser-Phe-Glu-Val-Cys-Val-Pro-Lys-Thr-Pro-Lys-Thr (Disulfide bridge: Cys <sub>3</sub> -Cys <sub>18</sub> , Cys <sub>10</sub> -Cys <sub>23</sub> , Cys <sub>17</sub> -Cys <sub>33</sub> ) (TFA salt)
Sequence Shortening:	EDCIPWKKGCVNRHGDCCLEGWCRRRSFEVCVPKTPKT	(Disulfide bridge: Cys <sub>3</sub> -Cys <sub>18</sub> , Cys <sub>10</sub> -Cys <sub>23</sub> , Cys <sub>17</sub> -Cys <sub>33</sub> )
Target:	Sodium Channel; Apoptosis	
Pathway:	Membrane Transporter/Ion Channel; Apoptosis	
Storage:	Sealed storage, away from moisture Powder      -80°C      2 years -20°C      1 year	

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

### BIOLOGICAL ACTIVITY

Description	Psalmotoxin 1 (PcTx1) TFA is a protein toxin that can bind at subunit-subunit interfaces of acid-sensing ion channel 1a (ASIC1a). Psalmotoxin 1 TFA is a potent and selective ASIC1a inhibitor ( $IC_{50}$ : 0.9 nM) by increasing the apparent affinity for H <sup>+</sup> of ASIC1a. Psalmotoxin 1 TFA can induce cell apoptosis, also inhibits cell migration, proliferation and invasion of cancer cells. Psalmotoxin 1 TFA can be used in the research of cancers, or neurological disease <sup>[1][3][4][6]</sup> .														
In Vitro	<p>Psalmotoxin 1 (20 nM, 125 s) TFA inhibits ASIC1a currents by drastically shifting the steady-state desensitization curve to lower H<sup>+</sup> concentrations<sup>[1]</sup>.</p> <p>Psalmotoxin 1 (30 nM) TFA competes with Ca<sup>2+</sup> in binding to ASIC1a channels<sup>[1]</sup>.</p> <p>Psalmotoxin 1 (100 or 200 ng, 24-72 h) TFA significantly weakens the migration, proliferation and invasion of MCF-7 and MDA-MB-231 cells<sup>[4]</sup>.</p> <p>Psalmotoxin 1 (100 ng/mL, 24 h) TFA significantly inhibits acid-induced increases in intracellular calcium and LDH release, induces cell apoptosis and cell cycle arrest in nucleus pulposus cells (NPCs)<sup>[5]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p><b>Cell Proliferation Assay<sup>[4]</sup></b></p> <table border="1"> <tr> <td>Cell Line:</td> <td>MCF-7 and MDA-MB-231 cells</td> </tr> <tr> <td>Concentration:</td> <td>100 or 200 ng</td> </tr> <tr> <td>Incubation Time:</td> <td>24, 48, 72 h</td> </tr> <tr> <td>Result:</td> <td>Inhibited the cell migration, proliferation and invasion of breast cancer cells.</td> </tr> </table> <p><b>Western Blot Analysis<sup>[5]</sup></b></p> <table border="1"> <tr> <td>Cell Line:</td> <td>Nucleus pulposus cells (NPCs)</td> </tr> <tr> <td>Concentration:</td> <td>100 ng/mL</td> </tr> <tr> <td>Incubation Time:</td> <td>24 h</td> </tr> </table>	Cell Line:	MCF-7 and MDA-MB-231 cells	Concentration:	100 or 200 ng	Incubation Time:	24, 48, 72 h	Result:	Inhibited the cell migration, proliferation and invasion of breast cancer cells.	Cell Line:	Nucleus pulposus cells (NPCs)	Concentration:	100 ng/mL	Incubation Time:	24 h
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Cell Line:	Nucleus pulposus cells (NPCs)														
Concentration:	100 ng/mL														
Incubation Time:	24 h														

	Result:	Decreased Bax and cleaved caspase-3 expression, and increased Bcl-2 expression.
In Vivo	<p>Psalmotoxin 1 (i.c.v., 1 ng/kg, a single dose) TFA is neuroprotective in a conscious model of stroke via direct inhibition of ASIC1a<sup>[2]</sup>.</p> <p>Psalmotoxin 1 (tail vein injection, 10 ng/kg, daily for 7 days) TFA inhibits tumor growth in breast cancer mice model<sup>[4]</sup>. MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>	
	Animal Model:	Male spontaneously hypertensive rats (SHR) <sup>[2]</sup>
	Dosage:	1 ng/kg, a single dose.
	Administration:	Intracerebroventricular (i.c.v.) injection
	Result:	<p>Reduced cortical and striatal infarct volumes measured 72 h post-stroke.</p> <p>Reduced the severity of motor deficit at 1 and 3 days after stroke compared to control rats.</p> <p>Displayed an anti-apoptotic effect in the occluded hemisphere (reduced stroke-induced caspase-3 positive cells).</p>
	Animal Model:	Female nude BALB/C mice (orthotopic implantation, MCF-7 and MDA-MB-231 cells) <sup>[3]</sup>
	Dosage:	10 ng/kg, daily for 7 days.
	Administration:	Tail vein injection
	Result:	Inhibited breast tumor growth.

## CUSTOMER VALIDATION

- Aging. 2021 Apr 6;13(7):10703-10723.

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## REFERENCES

- [1]. Chen X, et al. The tarantula toxin psalmotoxin 1 inhibits acid-sensing ion channel (ASIC) 1a by increasing its apparent H<sup>+</sup> affinity. J Gen Physiol. 2005 Jul;126(1):71-9.
- [2]. Claudia A McCarthy, et al. PCTx1 affords neuroprotection in a conscious model of stroke in hypertensive rats via selective inhibition of ASIC1a. Neuropharmacology. 2015 Dec;99:650-7.
- [3]. Niko Joeres, et al. Functional and pharmacological characterization of two different ASIC1a/2a heteromers reveals their sensitivity to the spider toxin PCTx1. Sci Rep. 2016 Jun 9;6:27647. doi: 10.1038/srep27647.
- [4]. Chao Yang, et al. Overexpression of acid-sensing ion channel 1a (ASIC1a) promotes breast cancer cell proliferation, migration and invasion. Transl Cancer Res. 2020 Dec;9(12):7519-7530.
- [5]. Feng Cai, et al. Acid-sensing ion channel 1a regulates the survival of nucleus pulposus cells in the acidic environment of degenerated intervertebral discs. Iran J Basic Med Sci. 2016 Aug;19(8):812-820.
- [6]. P Escoubas, et al. Isolation of a tarantula toxin specific for a class of proton-gated Na<sup>+</sup> channels. J Biol Chem. 2000 Aug 18;275(33):25116-21.

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

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