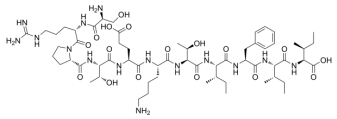
Gap 27

Cat. No.:	HY-P0139
CAS No.:	198284-64-9
Molecular Formula:	$C_{60}H_{101}N_{15}O_{17}$
Molecular Weight:	1304.53
Sequence:	Ser-Arg-Pro-Thr-Glu-Lys-Thr-Ile-Phe-Ile-Ile
Sequence Shortening:	SRPTEKTIFII
Target:	Gap Junction Protein
Pathway:	Cytoskeleton
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)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 26.32 mg/mL (20.18 mM; adjust pH to 3 with 0.1 M HCL)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	0.7666 mL	3.8328 mL	7.6656 mL
	5 mM	0.1533 mL	0.7666 mL	1.5331 mL
	10 mM	0.0767 mL	0.3833 mL	0.7666 mL

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

BIOLOGICAL ACTIVITY

Description

Gap 27, a synthetic connexin43 mimetic peptide, is a gap junction inhibitor. Gap 27 possesses conserved sequence homology to a portion of the second extracellular loop leading into the fourth transmembrane connexin segment[1][2].

In Vitro

Gap 27 causes a remarked decrease in the number of both TRAP-positive mononuclear and multinucleated rat osteoclasts cultured on bovine bone slices. The activity of the remaining osteoclasts is found to be diminished by measuring the percentage of osteoclasts with actin rings of all TRAP-positive cells. In addition, the resorbed area in the treated cultures is greatly diminished[1].

Incubation of the carotid artery with the gap junction inhibitor Gap 27 (500 μM) essentially abolishes the hyperpolarization to acetylcholine but it is without effect on that to levcromakalim. In the guinea-pig isolated internal carotid artery, Gap 27 inhibits acetylcholine-induced, endothelium-dependent hyperpolarizations[2].

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

In Vivo

Gap 27 (300 μM) inhibits relaxation by 40% in thoracic aorta and the superior mesenteric artery. Gap 27 also attenuates the

endothelium-dependent component of the relaxation induced by ATP in thoracic aorta. [3].

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

PROTOCOL

Cell Assay [1]

Bone cell cultures are cultured for 48 hours with three different treatments (control, heptanol and Gap 27). After the culture period, bone slices are fixed. The cells are stained for tartrate-resistant acid phosphatase (TRAP). To visualise the nuclei, the cells are incubated with the DNA-binding fluorochrome Hoechst 33258 (1 mg/mL stock diluted 1:800 in PBS) for 10 minutes at room temperature. The numbers of mononuclear and multinucleated TRAP-positive cells on each bone slice are counted [1].

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

CUSTOMER VALIDATION

- Cell Biol Toxicol. 2021 Jul 20.
- CNS Neurosci Ther. 2022 Jun 14.
- Front Cell Dev Biol. 2021 Jun 8;9:637233.

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REFERENCES

[1]. Ilvesaro J, et al. Connexin-mimetic peptide Gap 27 decreases osteoclastic activity. BMC Musculoskelet Disord. 2001;2:10.

[2]. Edwards G, et al. Role of gap junctions in the responses to EDHF in rat and guinea-pig small arteries. Br J Pharmacol. 1999 Dec;128(8):1788-94.

[3]. Chaytor AT,e t al. Central role of heterocellular gap junctional communication in endothelium-dependent relaxations of rabbit arteries. J Physiol. 1998 Apr 15;508 (Pt 2):561-73.

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