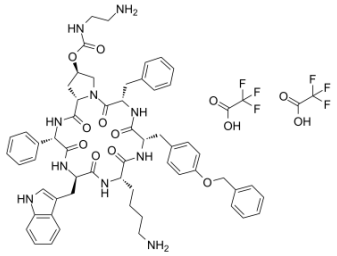
Pasireotide ditrifluoroacetate

Cat. No.:	HY-79135		
Molecular Formula:	$C_{62}H_{68}F_6N_{10}O_{13}$		
Molecular Weight:	1275.25		
Target:	Somatostatin Receptor		
Pathway:	GPCR/G Protein; Neuronal Signaling		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



BIOLOGICAL ACTIVITY

Description	Pasireotide (ditrifluoroacetate) is a stable cyclohexapeptide somatostatin mimic that exhibits unique high-affinity binding to human somatostatin receptors (subtypes sst1/2/3/4/5, pK_i=8.2/9.0/9.1/<7.0/9.9, respectively).
IC_{50} & Target	pKi: 8.2 (sst1), 9.0 (sst2), 9.1 (sst3), <7.0 (sst4), 9.9 (sst5)
In Vitro	Pasireotide effectively inhibits the growth hormone releasing hormone (GHRH) induced growth hormone (GH) release in primary cultures of rat pituitary cells with an IC_{50} of 0.4±0.1 nM[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Pasireotide potently suppressess GH secretion in rats. The ED_{50} values determined at 1 and 6 h after injection of pasireotide indicates its very long duration of action in vivo. In the rat, pasireotide strongly decreases IGF-1 plasma levels, with the efficacy being markedly enhanced compared with the effects elicited by SMS 201-995 after 7 days of treatment. Furthermore, in rats, dogs, and rhesus monkeys, pasireotide potently and dose-dependently decreases IGF-1 levels for prolonged periods of time without desensitization[1]. Pasireotide (160 mg/kg/month, s.c.) decreases serum insulin levels and increases serum glucose levels, reduces PNET tumor size, and demonstrates a reduction in tumor activity on PET/CT scan in Pdx1-Cre; Men1 floxed/floxed conditional knockout mice[2]. Pasireotide (50 μg/kg) inhibits arthritic joint swelling in a dose-dependent manner, strongly inhibits joint swelling during the acute phase of AIA. Pasireotide- and octreotide-treated mice show significantly increased mechanical thresholds on the inflamed side. Pasireotide potently decreases secondary hyperalgesia to mechanical and thermal stimuli. Mechanical thresholds in the pasireotide-treated mice are significantly higher than those in the saline-treated or octreotide-treated animals[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [1]	Mice are anesthetized using halothane and then shaved on their flank for subcutaneous injection of either phosphate buffered saline (PBS) buffer or Pasireotide at a concentration of 160 mg/Kg/month (64 mg/mL) every month for 4 months. The mice underwent a 24-h fast prior to collecting whole blood via a retro-orbital bleeding technique weekly at pre- and post-treatments. Serum glucose is measured by enzymatic colorimetric assay using a GM7 Analyzer. Serum insulin is measured by enzyme-linked immunosorbent assay (ELISA) with the UltrasensitiveMouse Insulin ELISA kit according to the manufacturer's instructions.

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CUSTOMER VALIDATION

- Hepatology. 2017 Oct;66(4):1197-1218.
- Am J Pathol. 2018 Apr;188(4):981-994.

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REFERENCES

[1]. Lewis I, et al. A novel somatostatin mimic with broad somatotropin release inhibitory factor receptor binding and superior therapeutic potential. J Med Chem. 2003 Jun 5;46(12):2334-44.

[2]. Quinn TJ, et al. Pasireotide (SOM230) is effective for the treatment of pancreatic neuroendocrine tumors (PNETs) in a multiple endocrine neoplasia type 1 (MEN1) conditional knockout mouse model. Surgery. 2012 Dec;152(6):1068-77.

[3]. Imhof AK, et al. Differential antiinflammatory and antinociceptive effects of the somatostatin analogs octreotide and pasireotide in a mouse model of immune-mediated arthritis. Arthritis Rheum. 2011 Aug;63(8):2352-62.

[4]. Lorenzo Pisarello M, et al. The combination of an HDAC6 inhibitor and a somatostatin receptor agonist synergistically reduces hepato-renal cystogenesis in an animal model of polycystic liver disease. Am J Pathol. 2018 Apr;188(4):981-994.

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