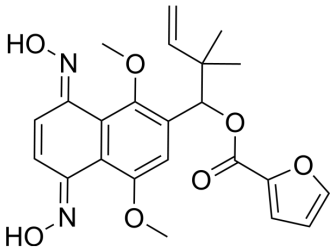
DSO-5a

Cat. No.:	HY-154985
CAS No.:	2195411-63-1
Molecular Formula:	$C_{23}H_{24}N_2O_7$
Molecular Weight:	440.45
Target:	PPAR; Bombesin Receptor; ERK
Pathway:	Cell Cycle/DNA Damage; Vitamin D Related/Nuclear Receptor; GPCR/G Protein; MAPK/ERK Pathway; Stem Cell/Wnt
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	DSO-5a is a potent, selective, orally active BB_3 agonist. DSO-5a is a representative DMAKO-00 derivative compound. DSO-5a upregulates ppar-γ activity through BB_3 and activates ERK1/2 phosphorylation. DSO-5a can be used in diabetes-related research[1].
IC_{50} & Target	PPARγ

In Vitro

DSO-5a (50 nM; 60min) induces IP-1 accumulation in hBB_3-HEK cells with a pEC_{50} of 8.485 and a strong calcium response with a pEC_{50} of 7.964[1].
DSO-5a (500 nM; 60min) induces IP-1 accumulation in mBB_3-HEK cells with a pEC_{50} of 7.262 and a strong calcium response with a pEC_{50} of 7.174[1].
DSO-5a (0-100 nM; 8min) causes a dose-dependent activation of ERK1/2 in hBB_3-H1299 and mBB_3-HEK cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	hBB_3-H1299 cells
Concentration:	0, 1,10,100 nM
Incubation Time:	8 min
Result:	Caused a dose-dependent activation of ERK1/2 in hBB_3-H1299 cells, which was completely blocked by Bantag-1.

In Vivo

DSO-5a (3-30 mg/kg; P.O.; 30 min) reduces blood glucose excursions in a dose-dependent manner in C57BL/6 mice[1].
DSO-5a (10 mg/kg/day; P.O.; 2-4 weeks) reduces the blood glucose concentration of diabetic db/db mice[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	C57BL/6 mice[1]
Dosage:	3 mg/kg; 10 mg/kg; 30 mg/kg
Administration:	Oral administration;30 min before glucose challenge (3 g/kg)

Result:	Showed that the change rates of AUC at 3, 10 and 30 mg/kg were 5.03, 16.42 and 28.30%, respectively. In BB_3 knockout mice, DSO-5a failed to inhibit blood glucose drift.

Animal Model:	Diabetic db/db mice[1]
Dosage:	10 mg/kg/day
Administration:	Oral administration; 2-4 weeks
Result:	After two weeks of treatment, the blood glucose excursion of db/db mice was significantly reduced. After four weeks, fasting blood glucose levels, glycosylated serum protein (GSP), and HOMA-IR were significantly decreased in the DSO-5a treatment group. Increased the protein expression of PPAR-gamma in white adipose tissue of db/db mice.

REFERENCES

[1]. Wu L, et al. Discovery of Dimethyl Shikonin Oxime 5a, a Potent, Selective Bombesin Receptor Subtype-3 Agonist for the Treatment of Type 2 Diabetes Mellitus. J Med Chem. 2023 Jun 22;66(12):8011-8029.

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