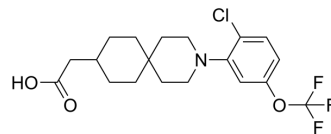


GPR120 Agonist 3

Cat. No.:	HY-101492		
CAS No.:	1599477-75-4		
Molecular Formula:	C ₁₉ H ₂₃ ClF ₃ NO ₃		
Molecular Weight:	405.84		
Target:	Free Fatty Acid Receptor		
Pathway:	GPCR/G Protein		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	2 years
		-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (123.20 mM)
 H₂O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)
 * "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent		Mass		
	Concentration		1 mg	5 mg	10 mg
	1 mM		2.4640 mL	12.3201 mL	24.6403 mL
	5 mM		0.4928 mL	2.4640 mL	4.9281 mL
	10 mM		0.2464 mL	1.2320 mL	2.4640 mL

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

In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2.5 mg/mL (6.16 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 2.5 mg/mL (6.16 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (6.16 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

GPR120 Agonist 3 is a selective Gpr120 agonist with a logEC₅₀ of -7.62.

IC₅₀ & Target

logEC₅₀: -7.62^[1]

In Vitro

GPR120 Agonist 3 is fully selective for Gpr120 (logEC₅₀=-7.62) with negligible activity towards Gpr40. GPR120 Agonist 3

produces concentration dependent increases in IP₃ production from both human and mouse Gpr120 expressing cells. GPR120 Agonist 3 leads to a concentration-dependent response to recruit β-arrestin-2 in both human and mouse Gpr120 expressing cells, with EC₅₀s of ~0.35 μM. GPR120 Agonist 3 strongly and comparably inhibits LPS-induced phosphorylation of Tak1, Ikkβ, and Jnk and blocked IκB degradation^[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

GPR120 Agonist 3 causes improved insulin sensitivity with increased glucose infusion rates, enhanced insulin stimulated-glucose disposal rate, along with a marked increase in the ability of insulin to suppress hepatic glucose production only in WT mice. GPR120 Agonist 3 treatment has beneficial effects on hepatic lipid metabolism, causing decreased hepatic steatosis, decreased liver triglycerides, and DAGs, along with reduced saturated free fatty acid content^[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration^[1]

Mice: Male C57Bl/6 WT or Gpr120 KO littermates are fed a normal chow (13.5% fat) or high-fat diet (60% fat) ad libitum for 15-20 weeks from 8 weeks of age. After 15 weeks on HFD, WT and Gpr120 KO mice are switched to an isocaloric HFD supplemented with ω3-FA concentrate or 30 mg/kg GPR120-IN-1 and fed for 5 weeks. Mice receive fresh diet every 3rd day, and food consumption and body weight are monitored^[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Acta Physiol (Oxf). 2019 May;226(1):e13215.

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REFERENCES

[1]. Oh DY, et al. A Gpr120-selective agonist improves insulin resistance and chronic inflammation in obese mice. Nat Med. 2014 Aug;20(8):942-7.

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

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