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) H ₂ O : < 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 so	lubility information to select the app	propriate solvent.			
In Vivo	1. 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					
	2. 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					
	3. Add each solvent o Solubility: ≥ 2.5 m	one by one: 10% DMSO >> 90% cor g/mL (6.16 mM); Clear solution	n oil			

BIOLOGICAL ACTIVITY				
Description	GPR120 Agonist 3 is a selective Gpr120 agonist with a logEC $_{50}$ of –7.62.			
IC ₅₀ & 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			

Product Data Sheet

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	produces concentration dependent increases in IP ₃ 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 EC ₅₀ s 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 conten ^[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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