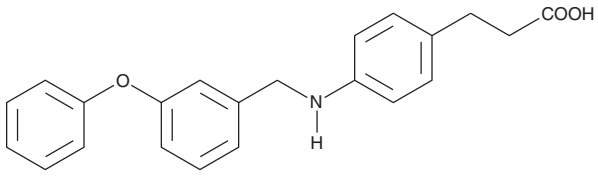
PRODUCT INFORMATION

GW 9508

Item No. 10008907

CAS Registry No.: 885101-89-3
Formal Name: 4-[[(3-phenoxyphenyl)methyl] amino]-benzenepropanoic acid
MF: $C_{22}H_{21}NO_3$
FW: 347.4
Purity: ≥98%
UV/Vis.: λ_{max}: 245, 297 nm
Supplied as: A crystalline solid
Storage: -20°C
Stability: ≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

GW 9508 is supplied as a crystalline solid. A stock solution may be made by dissolving the GW 9508 in an organic solvent purged with an inert gas. Solvents such as ethanol, DMSO, and dimethyl formamide purged with an inert gas can be used. The solubility of GW 9508 in these solvents is approximately 15 mg/ml.

GW 9508 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, GW 9508 should first be dissolved in ethanol and then diluted with the aqueous buffer of choice. GW 9508 has a solubility of approximately 0.5 mg/ml in a 1:1 solution of ethanol:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

GW 9508 is a small-molecule agonist of GPR40/FFA1 (EC_{50} = 47.8 nM for calcium mobilization in HEK293 cells).[1] It is selective for GPR40/FFA1 over GPR120/FFA4 (EC_{50} = 3,467 nM), as well as GPR43/FFA2 and GPR41/FFA3 (EC_{50}s = >50 µM).[1] GW 9508 potentiates glucose-stimulated and potassium chloride-mediated insulin secretion in MIN6 pancreatic β-cells but does not affect glucose-stimulated insulin secretion in primary rat or mouse islet cells.[1] It increases phosphorylation of AMP-activated protein kinase (AMPK) and acyl-CoA carboxylase (ACC), indicating AMPK and ACC activation, and decreases hepatic lipid accumulation in a mouse model of high-cholesterol diet-induced hepatic steatosis when administered at a dose of 100 mg/kg per day for three days.[2]

References

1. Briscoe, C.P., Peat, A.J., McKeown, S.C., *et al.* Pharmacological regulation of insulin secretion in MIN6 cells through the fatty acid receptor GPR40: Identification of agonist and antagonist small molecules. *Br. J. Pharmacol.* **148(5)**, 619-628 (2006).
2. Li, M., Meng, X., Xu, J., *et al.* GPR40 agonist ameliorates liver X receptor-induced lipid accumulation in liver by activating AMPK pathway. *Sci. Rep.* **6:25237**, (2016).

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