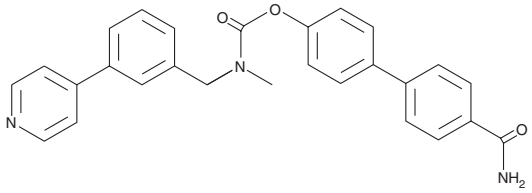
PRODUCT INFORMATION

WWL70

Item No. 10011213

CAS Registry No.: 947669-91-2
Formal Name: N-methyl-N-[[3-(4-pyridinyl)phenyl] methyl]-carbamic acid, 4'-(aminocarbonyl) [1,1'-biphenyl]-4-yl ester
MF: $C_{27}H_{23}N_3O_3$
FW: 437.5
Purity: ≥97%
UV/Vis.: λ_{max}: 203, 266 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

WWL70 is supplied as a crystalline solid. A stock solution may be made by dissolving the WWL70 in the solvent of choice, which should be purged with an inert gas. WWL70 is soluble in organic solvents such as DMSO and dimethyl formamide (DMF). The solubility of WWL70 in these solvents is approximately 1 and 2 mg/ml, respectively.

WWL70 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, WWL70 should first be dissolved in DMF and then diluted with the aqueous buffer of choice. WWL70 has a solubility of approximately 0.2 mg/ml in a 1:3 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

WWL70 is an inhibitor of α/β-hydrolase domain-containing protein 6 (ABHD6; IC_{50} = 70 nM).[1] It increases the expression of the adipose browning-related gene *Ucp1* in differentiated 3T3-L1 mouse adipocytes and increases the oxygen consumption rate (OCR), an effect that can be blocked by the PPARα antagonist GW 6471 (Item No. 11697), when used at a concentration of 10 µM.[2] WWL70 (10 mg/kg per day) also increases the expression of the adipose browning-related genes *Ucp1*, *Prdm16*, *Tmem26*, and *Tbx1* in visceral adipose tissue in mice fed a high-fat diet. WWL70 reduces adipose tissue mass and prevents glucose-intolerance and increases in body weight in mice fed a high-fat diet but does not reduce hepatic triacylglycerol levels.[3] It increases brain levels of 2-arachidonoyl glycerol (2-AG; Item No. 62160) and decreases the severity of experimental autoimmune encephalomyelitis (EAE) in wild-type, but not cannabinoid (CB) receptor 2 (CB_2) knockout, mice when administered at a dose of 10 mg/kg per day starting at disease onset.[4]

References

1. Li, W., Blankman, J.L., and Cravatt, B.F. A functional proteomic strategy to discover inhibitors for uncharacterized hydrolases. *J. Am. Chem. Soc.* **129(31)**, 9594-9595 (2007).
2. Zhao, S., Mugabo, Y., Ballentine, G., *et al.* α/β-Hydrolase domain 6 deletion induces adipose browning and prevents obesity and type 2 diabetes. *Cell Rep.* **14(12)**, 2872-28888 (2016).
3. Thomas, G., Betters, J.L., Lord, C.C., *et al.* The serine hydrolase ABHD6 Is a critical regulator of the metabolic syndrome. *Cell Rep.* **5(2)**, 508-520 (2013).
4. Wen, J., Ribeiro, R., Tanaka, M., *et al.* Activation of CB2 receptor is required for the therapeutic effect of ABHD6 inhibition in experimental autoimmune encephalomyelitis. *Neuropharmacology* **99**, 196-209 (2015).

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