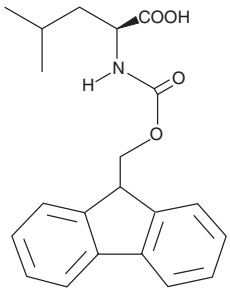
PRODUCT INFORMATION

Fmoc-L-Leucine

Item No. 10004888

CAS Registry No.:	35661-60-0
Formal Name:	N-[(9H-fluoren-9-ylmethoxy)carbonyl]-L-leucine
Synonyms:	Fmoc-Leu, NPC 15199, NSC 334290
MF:	$C_{21}H_{23}NO_4$
FW:	353.4
Purity:	≥98%
UV/Vis.:	λ_{max}: 206, 265, 289, 300 nm
Supplied as:	A crystalline solid
Storage:	Room Temperature
Stability:	≥4 years



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

Laboratory Procedures

Fmoc-L-leucine is supplied as a crystalline solid. A stock solution may be made by dissolving the Fmoc-L-leucine in the solvent of choice, which should be purged with an inert gas. Fmoc-L-leucine is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide. The solubility of Fmoc-L-leucine in these solvents is approximately 30 mg/ml.

Fmoc-L-leucine is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, Fmoc-L-leucine should first be dissolved in ethanol and then diluted with the aqueous buffer of choice. Fmoc-L-leucine 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

Peroxisome proliferator-activated receptor γ (PPARγ) isoforms heterodimerize with retinoic X receptors to modulate gene expression related to adipocyte differentiation, fatty acid uptake and storage, and glucose metabolism.[1] Natural agonists of PPARγ include fatty acids (*e.g.*, linoleic acid and 15-deoxy-$\Delta^{12,14}$-prostaglandin J_2), while thiazolidinediones (*e.g.*, rosiglitazone and pioglitazone) are potent synthetic agonists.[2,3] Fmoc-L-leucine is a partial agonist of PPARγ.[2,4] It activates PPARγ with a lower potency (K_i = 15 *versus* 0.035 µM) but a similar maximal efficacy compared to rosiglitazone.[4] Fmoc-L-leucine improves insulin resistance in normal, diet-induced glucose-intolerant and in diabetic *db/db* mice, yet has reduced adipogenic activity.[4] As a result, it is classified as a selective PPARγ modulator (SPPARM), capable of producing insulin-sensitizing effects while minimizing side effects associated with full agonists.[2]

References

1. Heikkinen, S., Auwerx, J., and Argmann, C.A. PPARγ in human and mouse physiology. *Biochim. Biophys. Acta* **1771(8)**, 999-1013 (2007).
2. Villacorta, L., Schopfer, F.J., Zhang, J., *et al.* PPARγ and its ligands: Therapeutic implications in cardiovascular disease. *Clin. Sci.* **116(3)**, 205-218 (2009).
3. Zieleniak, A., Wójcik, M., and Wozniak, L.A. Structure and physiological functions of the human peroxisome proliferator-activated receptor γ. *Arch. Immunol. Ther. Exp.* **56(5)**, 331-345 (2008).
4. Rocchi, S., Picard, F., Vamecq, J., *et al.* A unique PPARγ ligand with potent insulin-sensitizing yet weak adipogenic activity. *Mol. Cell* **8(4)**, 737-747 (2001).

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