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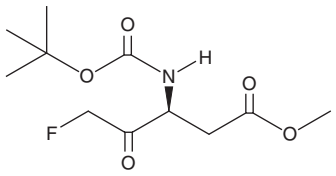
PRODUCT INFORMATION

Boc-D-FMK

Item No. 16118

CAS Registry No.: 187389-53-3
Formal Name: 3S-[[(1,1-dimethylethoxy)carbonyl]amino]-5-fluoro-4-oxo-pentanoic acid, methyl ester
Synonyms: BAF, Boc-Asp(OMe)-FMK, Boc-D-Fluoromethyl Ketone, Boc-D(OMe)-FMK
MF: $C_{11}H_{18}FNO_5$
FW: 263.3
Purity: ≥95%
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

Boc-D-FMK is supplied as a crystalline solid. A stock solution may be made by dissolving the Boc-D-FMK in the solvent of choice. Boc-D-FMK is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of Boc-D-FMK in ethanol is approximately 50 mg/ml and approximately 30 mg/ml in DMSO and DMF.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. Organic solvent-free aqueous solutions of Boc-D-FMK can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of Boc-D-FMK in PBS, pH 7.2, is approximately 0.3 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Boc-D-FMK is a cell-permeable, irreversible pan-caspase inhibitor.[1-2] Also known as Boc-D(OMe)-FMK, this compound contains a methyl ester group, which facilitates uptake by cells and subsequently is removed by cytoplasmic esterases, leaving the functional inhibitor. Boc-D-FMK blocks apoptosis stimulated by TNF-α in neutrophils (IC_{50} = 39 µM), by bile duct ligation in hepatocytes, and by TNF-α in renal endothelial cells.[3-5] The FMK pharmacophore is known to interact with non-caspase cysteine proteases, presumably explaining why Boc-D-FMK also inhibits cathepsins H and L.[1]

References

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2. Gregoli, P.A. and Bondurant, M.C. *J. Cell Physiol.* **178(2)**, 133-143 (1999).
3. Cowburn, A.S., White, J.F., Deighton, J., *et al*. *Blood* **105(7)**, 2970-2972 (2005).
4. Sheen-Chen, S.M., Hung, K.S., and Eng, H.L. *J. Gastroenterol. Hepatol.* **23(8 Pt 1)**, 1276-1279 (2008).
5. Wu, X., Guo, R., Chen, P., *et al*. *Am. J. Physiol. Renal. Physiol.* **297(2)**, F316-F326 (2009).

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