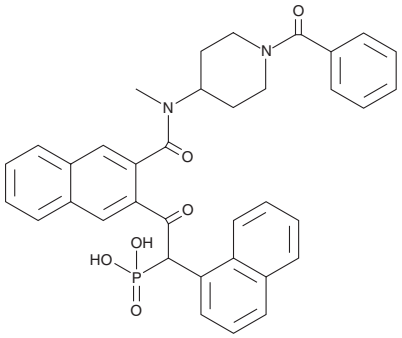
PRODUCT INFORMATION

Cathepsin G Inhibitor I

Item No. 14928

CAS Registry No.:	429676-93-7
Formal Name:	P-[2-[3-[[(1-benzoyl-4-piperidinyl)methylamino] carbonyl]-2-naphthalenyl]-1-(1-naphthalenyl)-2-oxoethyl]-phosphonic acid
MF:	$C_{36}H_{33}N_2O_6P$
FW:	620.6
Purity:	≥95%
UV/Vis.:	λ_{max}: 224, 248 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

Cathepsin G inhibitor I is supplied as a crystalline solid. A stock solution may be made by dissolving the cathepsin G inhibitor I in the solvent of choice, which should be purged with an inert gas. Cathepsin G inhibitor I is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of cathepsin G inhibitor I in these solvents is approximately 10 and 5 mg/ml, respectively.

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

Description

Cathepsin G inhibitor I is an inhibitor of cathepsin G (IC_{50} = 53 nM).[1] It is selective for cathepsin G over various serine proteases, including thrombin, Factor Xa, Factor IXa, plasmin, trypsin, and leukocyte proteinase 3 (PR3; IC_{50}s = >100 µM for all), but also weakly inhibits chymotrypsin. It inhibits cathepsin G-induced activation of IL-36 in neutrophil degranulates when used at a concentration of 10 µM.[2] Cathepsin G inhibitor I (10 µM) reduces $CD4^+$ T cell-induced secretion of IFN-γ and IL-17 in B cells incubated with tetanus toxin C-fragments (TTC) and also reduces TTC presentation in B cells.[3] It decreases the size of neuromyelitis optica lesions and reduces the number of perivascular neutrophils, indicating decreased neutrophil brain entry, when used in combination with the neutrophil elastase inhibitor sivelestat (Item No. 17779) in a mouse model of IgG-induced brain injury when administered intracerebrally at a dose of 5 µg/animal.[4]

Reference

1. Greco, M.N., Hawkins, M.J., Powell, E.T., *et al.* Nonpeptide inhibitors of cathepsin G: Optimization of a novel β-ketophosphonic acid lead by structure-based drug design. *J. Am. Chem. Soc.* **124(15)**, 3810-3811 (2002).
2. Henry, C.M., Sullivan, G.P., Clancy, D.M., *et al.* Neutrophil-derived proteases escalate inflammation through Aactivation of IL-36 family cytokines. *Cell Rep.* **14(4)**, 708-722 (2016).
3. Reich, M., Lesner, A., Legowska, A., *et al.* Application of specific cell permeable cathepsin G inhibitors resulted in reduced antigen processing in primary dendritic cells. *Mol. Immunol.* **46(15)**, 2994-2999 (2009).
4. Saadoun, S., Waters, P., MacDonald, C., *et al.* Neutrophil protease inhibition reduces neuromyelitis optica-immunoglobulin G-induced damage in mouse brain. *Ann. Neurol.* **71(3)**, 323-333 (2012).

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