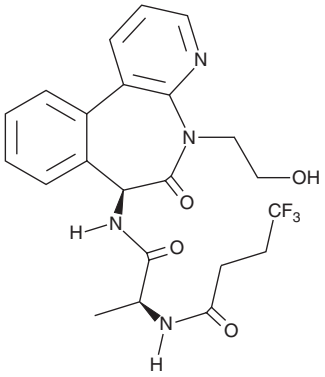
PRODUCT INFORMATION

Crenigacestat

Item No. 27919

CAS Registry No.:	1421438-81-4
Formal Name:	N-[(1S)-2-[[(7S)-6,7-dihydro-5-(2-hydroxyethyl)-6-oxo-5H-pyrido[3,2-a][3]benzazepin-7-yl]amino]-1-methyl-2-oxoethyl]-4,4,4-trifluoro-butanamide
Synonym:	LY3039478
MF:	$C_{22}H_{23}F_3N_4O_4$
FW:	464.4
Purity:	≥95%
UV/Vis.:	λ_{max}: 227 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

Crenigacestat is supplied as a crystalline solid. A stock solution may be made by dissolving the crenigacestat in the solvent of choice, which should be purged with an inert gas. Crenigacestat is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of crenigacestat in ethanol is approximately 10 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 crenigacestat can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of crenigacestat in PBS, pH 7.2, is approximately 0.5 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Crenigacestat is an orally bioavailable Notch inhibitor.[1] It induces cell cycle arrest at the G_0/G_1 phase and decreases expression of the oncogenes *MYC* and *CCNA1* in Caki human renal cancer cells.[2] Crenigacestat (8 mg/kg per day) reduces tumor growth and increases survival in a 769-P mouse xenograft model.

References

1. Yuen, E., Posada, M., Smith, C., *et al.* Evaluation of the effects of an oral notch inhibitor, crenigacestat (LY3039478), on QT interval, and bioavailability studies conducted in healthy subjects. *Cancer Chemother. Pharmacol.* **83(3)**, 483-492 (2019).
2. Bhagat, T.D., Zou, Y., Huang, S., *et al.* Notch pathway is activated via genetic and epigenetic alterations and is a therapeutic target in clear cell renal cancer. *J. Biol. Chem.* **292(3)**, 837-846 (2017).

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