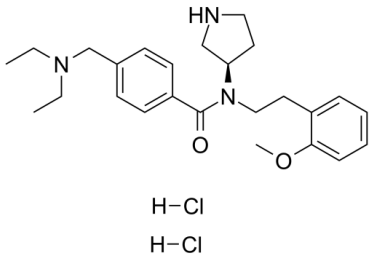
Data Sheet

Product Name:	PF429242 (dihydrochloride)
Cat. No.:	CS-6890
CAS No.:	2248666-66-0
Molecular Formula:	C25H37Cl2N3O2
Molecular Weight:	482.49
Target:	LPL Receptor; Virus Protease
Pathway:	Anti-infection; GPCR/G Protein
Solubility:	DMSO : ≥ 83.3 mg/mL (172.65 mM)



BIOLOGICAL ACTIVITY:

PF429242 dihydrochloride is a reversible and competitive **S1P** inhibitor with an **IC_{50}** of 175 nM. IC50 & Target: IC50: 175 nM (S1P)[1] **In Vitro:** 10 μM PF-429242 inhibits endogenous SREBP processing in Chinese hamster ovary cells. PF-429242 also down-regulates the signal from an SRE-luciferase reporter gene in human embryonic kidney 293 cells and the expression of endogenous SREBP target genes in cultured HepG2 cells. In HepG2 cells, PF-429242 inhibits cholesterol synthesis, with an IC_{50} of 0.5 μM[1]. The addition of PF-429242 (30 μM) shows statistically significant suppression of infectious viral titers and viral RNA copies in the cell culture fluids. PF-429242 treatment also shows suppressive effects on DENV2 yields in the cultured fluids of human-derived HEK-293, Hep G2, and non-human-primate derived LLC-MK2 cells[2]. PF-429242 efficiently prevents the processing of GPC from the prototypic arenavirus lymphocytic choriomeningitis virus (LCMV) and LASV, which correlates with the compound's potent antiviral activity against LCMV and LASV in cultured cells[3]. **In Vivo:** In mice treated with PF-429242 for 24 h, the expression of hepatic SREBP target genes is suppressed, and the hepatic rates of cholesterol and fatty acid synthesis are reduced[1].

PROTOCOL (Extracted from published papers and Only for reference)

Animal Administration: PF-429242 is prepared in saline.[1]Mice: To test the in vivo efficacy of PF-429242 in regulating SREBP target genes, male CD1 mice are dosed i.p. with 10 or 30 mg/kg PF-429242 or saline once every 6 over a 24-h period. Mice are euthanized 6 h after the final dose, and liver tissue is collected, frozen rapidly in liquid nitrogen, and stored at -80°C. For RNA isolation, 50 to 100 mg of frozen liver tissue from each sample is homogenized in 1 ml of TRIzol reagent. Total RNA is extracted following the manufacturer's instructions, and the resulting total RNA from each sample underwent DNA-free treatment[1].

References:

[1]. Hawkins JL, et al. Pharmacologic inhibition of site 1 protease activity inhibits sterol regulatory element-binding protein processing and reduces lipogenic enzyme gene expression and lipid synthesis in cultured cells and experimental animals. J Pharmacol Exp Ther. 2008 Sep;326(3):801-8.

[2]. Uchida L, et al. Suppressive Effects of the Site 1 Protease (S1P) Inhibitor, PF-429242, on Dengue Virus Propagation. Viruses. 2016 Feb 10;8(2). pii: E46. doi: 10.3390/v8020046.

[3]. Urata S, et al. Antiviral activity of a small-molecule inhibitor of arenavirus glycoprotein processing by the cellular site 1 protease. J Virol. 2011 Jan;85(2):795-803.

CAIndexNames:

Benzamide, 4-[(diethylamino)methyl]-N-[2-(2-methoxyphenyl)ethyl]-N-(3R)-3-pyrrolidinyl-,hydrochloride (1:2)

SMILES:

O=C(N(CCC1=CC=CC=C1OC)[C@H]2CNCC2)C3=CC=C(CN(CC)CC)C=C3.[H]Cl.[H]Cl

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: 732-484-9848 Fax: 888-484-5008 E-mail: sales@ChemScene.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA