Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

Talabostat (mesylate)

Catalog No: tcsc3187

Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Specifications

CAS No:
150080-09-4

Formula:
$C_{10}H_{23}BN_2O_6S$

Pathway:
Metabolic Enzyme/Protease

Target:
Dipeptidyl Peptidase

Purity / Grade:
>98%

Solubility:
DMSO : ≥ 40 mg/mL (128.96 mM)

Alternative Names:
Val-boroPro;PT100

Observed Molecular Weight:
310.18

Product Description

Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

Talabostat mesylate is a potent, nonselective and orally available **dipeptidyl peptidase IV** (DPP-IV) inhibitor with a $\mathbf{K_i}$ of 0.18 nM.

IC50 & Target: Ki: 0.18 nM (DPP-IV), 1.5 nM (DPP8), 0.76 nM (DPP9)[1]

In Vitro: Talabostat mesylate is a nonselective DPP-IV inhibitor, inhibiting DPP8/9, FAP, DPP2 and some other DASH family enzymes essentially as potently as it inhibits DPP-IV[1]. Talabostat stimulates the immune system by triggering a proinflammatory form of cell death in monocytes and macrophages known as pyroptosis. The inhibition of two serine proteases, DPP8 and DPP9, activates the proprotein form of caspase-1 independent of the inflammasome adaptor ASC[2]. Talabostat competitively inhibits the dipeptidyl peptidase (DPP) activity of FAP and CD26/DPP-IV, and there is a high-affinity interaction with the catalytic site due to the formation of a complex between $Ser^{630/624}$ and the boron of talabostat[3].

In Vivo: Talabostat can stimulate immune responses against tumors involving both the innate and adaptive branches of the immune system. In WEHI 164 fibrosarcoma and EL4 and A20/2J lymphoma models, PT-100 causes regression and rejection of tumors. The antitumor effect appears to involve tumor-specific CTL and protective immunological memory. Talabostat treatment of WEHI 164-inoculated mice increases mRNA expression of cytokines and chemokines known to promote T-cell priming and chemoattraction of T cells and innate effector cells[3]. Talabostat treated mice show significant less fibrosis and FAP expression is reduced. Upon PT100 treatment, significant differences in the MMP-12, MIP-1α, and MCP-3 mRNA expression levels in the lungs are also observed. Treatment with PT100 in this murine model of pulmonary fibrosis has an anti-fibro-proliferative effect and increases macrophage activation[4].