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

Bestatin (trifluoroacetate)

Catalog No: tcsc1912

Available Sizes

Size: 10mg

Size: 50mg

Size: 100mg

Specifications

CAS No:
223763-80-2

Formula:
$C_{18}H_{25}F_3N_2O_6$

Pathway:
Metabolic Enzyme/Protease

Target:
Aminopeptidase

Purity / Grade:
>98%

Solubility:
10 mM in DMSO

Alternative Names:
Ubenimex trifluoroacetate

Observed Molecular Weight:
422.4

Product Description

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Bestatin trifluoroacetate is an inhibitor of **CD13 (Aminopeptidase N)/APN** and **leukotriene A4 hydrolase**, used for cancer treatment.

In Vitro: Bestatin enhances ATRA-induced differentiation and inhibits ATRA-driven phosphorylation of p38 MAPK in ATRA-sensitive APL NB4 cells. Bestatin can not reverse the differentiation block in ATRA-resistant APL MR2 cells. CD13 ligation with anti-CD13 antibody WM-15 results in phosphorylation of p38 MAPK, reduces the inhibition of Bestatin on the phosphorylation of p38 MAPK, and completely abolishes the enhancement of Bestatin on ATRA-inducing differentiation in NB4 cells[2]. Bestatin (600 μM)-treated cells progress slower through the cell cycle due to decreased rate of cell growth and the frequency of cell division. Bestatin inhibits the frequency of mitosis and the inherent multinuclearity in D. discoideum, and is not cytotoxic to D. discoideum cells at 0-600 μM. Bestatin inhibits aminopeptidase activity in lysates of PsaA-GFP- and GFP-expressing cells by 69.39% ± 10.5% and 39.93% ± 18.7% of control, respectively[4].

In Vivo: Bestatin (20 μM) significantly reduces CD13 expression in diabetic mice and results a significant inhibition of MMP-9 specific gelationolytic band densities compared to diabetic vehicle-treated mice. Bestatin treatment significantly inhibits the expression of VEGF and heparanase in diabetic mice. Intravitreal bestatin treatment significantly downregulates the expression of both HIF-1α and VEGF in diabetic mice retinas. Furthermore, the upregulated expression of heparanase in diabetic mice retinas is significantly inhibited by intravitreal bestatin treatment[1]. Bestatin (10, 1, and 0.1mg/kg, i.p.) treatment before the antigen-potentiated humoral response to SRBC results in an increased number of splenocytes producing hemolytic anti-SRBC antibodies (PFC) and the 2-ME-resistant serum hemagglutinin titer (at a dose of 0.1 mg/kg). Bestatin (1 and 0.1 mg/kg) administered to mice five times on alternate days after cyclophosphamide injection does not change the suppressive effect of the drug regarding the number of PFC, and even causes the further decrease of the total anti-SRBC hemagglutinins at dose of 1 mg/kg on day 7 after antigen stimulation[3].