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

Quiflapon sodium

Catalog No: tcsc0936

Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg

Specifications

CAS No:
147030-01-1

Formula:
$C_{34}H_{34}ClN_2NaO_3S$

Pathway:
Immunology/Inflammation

Target:
FLAP

Purity / Grade:
>98%

Solubility:
10 mM in DMSO

Alternative Names:
MK591

Observed Molecular Weight:
609.15

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Product Description

MK591 is a selective and specific 5-Lipoxygenase-activating protein (**FLAP**) inhibitor.

In Vitro: MK591 and SB203580 are able to block SEB-induced human PBMC cell proliferation. MK591 down regulates three genes [for cathepsin L, IL-17 and guanylate binding protein (GBP)-2] that are up regulated by SEB[1]. MK591 undergoes apoptosis within hours of treatment. MK591 also induces rapid activation of the stress kinase, c-Jun N-terminal kinase (JNK), which plays an important role in the apoptosis process. MK591 triggers apoptosis in prostate cancer cells without inhibition of PI3K-Akt, or ERK. Moreover, MK591 and LY294002 (an inhibitor of PI3K) exert synergistic effect in inducing apoptosis in prostate cancer cells[2]. MK-591 influences cAMP response element-binding protein but not Sp1[4].

In Vivo: Hyperoxia groups of mice treated with MK-0591 (20, 40 mg/kg) show alveolarization that resembles that of room air controls while untreated hyperoxia groups show definite evidence of aberrant alveolarization but no inflammation[3]. Comparison of the Aβ-immunopositive areas between the placebo and MK-591 (320 mg/kg)-treated group reveals a statistically significant reduction of the amyloid burden in the treated mice. MK-591 also has a significant reduction in brain levels of IL-1β. Mice treated with MK-591 show a statistically significant decrease in the steady-state levels of total CREB and its phosphorylated form at Ser133[4]
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