Web: www.taiclone.com
Tel: +886-2-2735-9682
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Roflumilast

Catalog No: tcsc0963

Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Specifications

CAS No:
162401-32-3

Formula:
$C_{17}H_{14}Cl_2F_2N_2O_3$

Pathway:
Metabolic Enzyme/Protease

Target:
Phosphodiesterase (PDE)

Purity / Grade:
>98%

Solubility:
H2O :

Observed Molecular Weight:
403.21

Product Description

Roflumilast is a selective **PDE4** inhibitor with **IC_{50}**s of 0.7, 0.9, 0.7, and 0.2 nM for **PDE4A1**, **PDEA4**, **PDEB1**, and **PDEB2**, respectively, without affecting PDE1, PDE2, PDE3 or PDE5 isoenzymes from various cells.

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IC50 & Target: IC50: 0.7 nM (PDE4A1), 0.9 nM (PDE4A4), 0.7 nM (PDE4B1), 0.2 nM (PDE4B2)[1]

In Vitro: Roflumilast does not affect PDE enzymes apart from PDE4, and is a subnanomolar inhibitor of most PDE4 splicing variants tested. It showed no PDE4 subtype selectivity apart from PDE4C (4C1, IC_{50}=3 nM; 4C2, IC_{50}=4.3 nM), which is inhibited with a slightly lower potency[2]. Roflumilast is a potent and selective PDE4 inhibitor. Roflumilast is a monoselective PDE4 inhibitor since it does not affect other PDE isoenzymes, including PDE1, PDE2, PDE3, and PDE5 up to 10,000-fold higher concentrations. Roflumilast inhibits human neutrophil functions. Roflumilast inhibits TNFα synthesis in monocyte-derived dendritic cells. Rolfumilast inhibits proliferation and cytokine synthesis in $CD4^+$ T cells. Proliferation is inhibited to a maximum of about 60% by Roflumilast with a potency (IC_{30}) of 7 nM[3].

In Vivo: Animal studies with Roflumilast demonstrated that it reduced the accumulation of neutrophils in bronchoalveolar lavage fluid following short-term exposure of guinea pigs, mice or rats to tobacco smoke, and following exposure of rats to a combination of tobacco smoke and bacterial lipopolysaccharide, and abolished the lung parenchymal influx of inflammatory cells seen in rats exposed to tobacco smoke for 7 months[2]. Roflumilast blocks COPD progression in $pIgR^{-/-}$ mice. For these studies, 9-month-old WT or $pIgR^{-/-}$ mice are treated daily by oral gavage with 100 µg of Roflumilast (5 µg/g) or vehicle (4% methylcellulose, 1.3% PEG400) for 3 months and lungs are harvested at 12 months of age. Unlike $pIgR^{-/-}$ mice treated with vehicle, mice treated with Roflumilast had no progression of small airway wall remodelling after starting treatment. Strikingly, 12-month-old $pIgR^{-/-}$ mice treated with Roflumilast had reduced indices of emphysema compared with 9-month-old $pIgR^{-/-}$ mice, indicating that Roflumilast not only blocks progression of emphysema in this model but apparently facilitates some resolution of the emphysematous destruction of lung parenchyma[4].