

# 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:**

H<sub>2</sub>O :

**Observed Molecular Weight:**

403.21

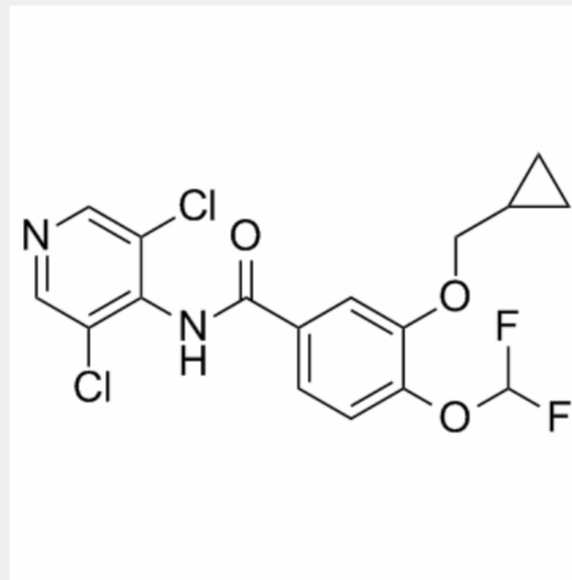
## Product Description

Roflumilast is a selective **PDE4** inhibitor with **IC<sub>50</sub>**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.

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

**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<sub>50</sub>=3 nM; 4C2, IC<sub>50</sub>=4.3 nM), which is inhibited with a slightly lower potency<sup>[2]</sup>. 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 TNF $\alpha$  synthesis in monocyte-derived dendritic cells. Roflumilast inhibits proliferation and cytokine synthesis in CD4<sup>+</sup> T cells. Proliferation is inhibited to a maximum of about 60% by Roflumilast with a potency (IC<sub>30</sub>) of 7 nM<sup>[3]</sup>.

**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<sup>[2]</sup>. Roflumilast blocks COPD progression in plgR<sup>-/-</sup> mice. For these studies, 9-month-old WT or plgR<sup>-/-</sup> mice are treated daily by oral gavage with 100  $\mu$ g of Roflumilast (5  $\mu$ g/g) or vehicle (4% methylcellulose, 1.3% PEG400) for 3 months and lungs are harvested at 12 months of age. Unlike plgR<sup>-/-</sup> mice treated with vehicle, mice treated with Roflumilast had no progression of small airway wall remodelling after starting treatment. Strikingly, 12-month-old plgR<sup>-/-</sup> mice treated with Roflumilast had reduced indices of emphysema compared with 9-month-old plgR<sup>-/-</sup> 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<sup>[4]</sup>.



All products are for RESEARCH USE ONLY. Not for diagnostic & therapeutic purposes!