

Inolitazone

Catalog No: tcsc0778



Available Sizes

Size: 5mg



Specifications

CAS No:

223132-37-4

Formula:

$C_{27}H_{26}N_4O_4S$

Pathway:

Cell Cycle/DNA Damage

Target:

PPAR

Purity / Grade:

>98%

Solubility:

10 mM in DMSO

Alternative Names:

Efatutazone;CS-7017;RS5444

Observed Molecular Weight:

502.58

Product Description

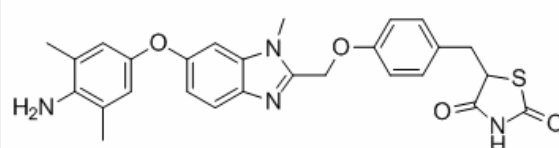
Inolitazone a novel high-affinity **PPAR γ** agonist that is dependent upon PPAR γ for its biological activity with **IC₅₀** of 0.8 nM for growth inhibition.

IC50 & Target: PPAR γ ^[1]

In Vitro:

Inolitazone (RS5444) upregulates the cell cycle kinase inhibitor, p21^{WAF1/CIP1}. Silencing p21^{WAF1/CIP1} rendered cells insensitive to Inolitazone. A 10 nM dose of Inolitazone activates PPAR γ :RXR α -dependent transcription as demonstrated in a transient transfection assay utilizing a PPRE response element fused to a luciferase reporter gene (PPRE3-tk-luc). DRO cells are treated in culture with Inolitazone, Rosiglitazone, or Troglitazone at the indicated concentrations. DRO cells are transiently transfected with PPRE3-tk-luc to examine effective concentrations at which EC₅₀ occurs. The EC₅₀s are 1 nM (Inolitazone), 65 nM (Rosiglitazone) and 631 nM (Troglitazone). Similarly, the calculated inhibitory concentration at IC₅₀ is 0.8 nM for Inolitazone, 75 nM for Rosiglitazone, and 1412 nM for Troglitazone. Inolitazone specifically activates PPAR γ , but not PPAR α or PPAR δ . Exposure of 10 nM Inolitazone following transient transfection with the appropriate PPAR isoform (γ , α , or δ) and PPAR response element linked to a luciferase reporter in RIE rat small intestinal cell line, which does not express PPARs, yields increased luciferase activity only in the presence of PPAR γ and PPRE3-tk-luc^[1]. DRO cells are growth inhibited by 10 nM Inolitazone (RS5444) through a PPAR γ -dependent mechanism^[2].

In Vivo: Inolitazone (RS5444) plus Paclitaxel demonstrate additive antiproliferative activity in cell culture and minimal ATC tumor growth. When Inolitazone is administered in the diet to athymic nude mice prior to DRO tumor cell implantation, tumor growth is inhibited in a dose responsive fashion. At the highest dose, 0.025% Inolitazone inhibits growth on day 32 by 94.4% as compared to that of control. In this treatment group, five of 10 animals do not develop demonstrable tumors. In the 0.0025% treatment group, tumor growth is inhibited by 62.3% compared to that of control on day 32 while the 0.00025% dose demonstrated no growth inhibitory activity as compared to control. Tumors is nest allowed to establish in the mouse and began 0.025% Inolitazone treatment of mice 1 week after DRO or ARO tumor cell implantation. Inolitazone treated animals demonstrate tumor growth inhibition of 68.9% in DRO tumors and 48.3% in ARO tumors as compared to that of their respective controls on day 35^[1].



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