

Probenecid

Catalog No: tcsc2646



Available Sizes

Size: 1g

Size: 5g



Specifications

CAS No:

57-66-9

Formula:

$C_{13}H_{19}NO_4S$

Pathway:

Membrane Transporter/Ion Channel

Target:

TRP Channel

Purity / Grade:

>98%

Solubility:

DMSO : ≥ 100 mg/mL (350.43 mM); H₂O :

Observed Molecular Weight:

285.36

Product Description

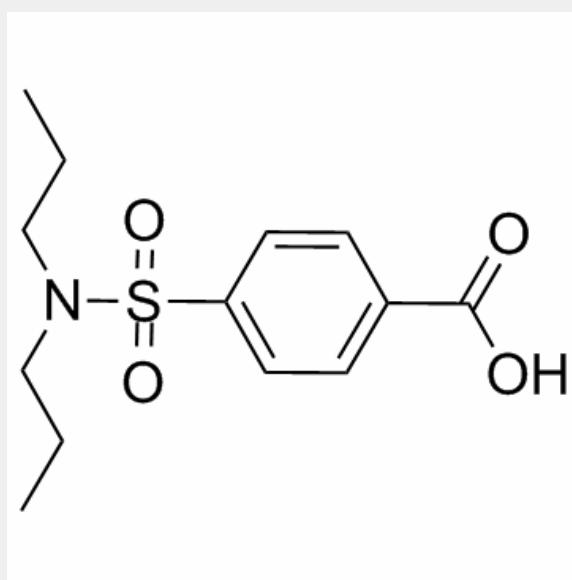
Probenecid is a potent and selective agonist of transient receptor potential vanilloid 2 (**TRPV2**) channels.

IC₅₀ & Target: TRPV2^[1]

In Vitro: Probenecid efficiently inhibits ATP-dependent active vesicular N-ethylmaleimide glutathione (NEM-GS) uptake by both MRP1 and MRP2. A significant inhibition of the MRP1-ATPase is observed at higher organic anion concentrations. In contrast, the

ATPase activity of MRP2 is strongly stimulated by both Probenecid (approximate $K_{ACT}=250\text{ }\mu\text{M}$), sulfinpyrazone ($K_{ACT}=300\text{ }\mu\text{M}$), and indomethacin ($K_{ACT}=150\text{ }\mu\text{M}$), and ATPase activation is even stronger than in the case of NEM-GS. The organic anion activation of the MRP2-ATPase followed bell-shaped curves, with maximum values obtained at about 2 mM for Probenecid, 800 μM for sulfinpyrazone, and 400 μM for indomethacin^[2]. Probenecid is an inhibitor of the hTAS2R16, hTAS2R38, and hTAS2R43 bitter taste receptors. Probenecid acts on a subset of TAS2Rs and inhibits through a novel, allosteric mechanism of action. Probenecid is also commonly used to enhance cellular signals in GPCR calcium mobilization assays. Probenecid specifically inhibits the cellular response mediated by the bitter taste receptor hTAS2R16 and provide molecular and pharmacological evidence for direct interaction with this GPCR using a non-competitive (allosteric) mechanism^[3].

In Vivo: Administration of Probenecid to WT mice results in increased contractility as measured via ejection fraction (EF) relative to EF in control mice given saline. The increased contractility is noted within 5 minutes of the bolus injection with all doses at or above 75 mg/kg (peak change of 5.26 ± 3.35 , 8.40 ± 2.80 , 7.32 ± 2.52 for 75mg/kg, 100mg/kg and 200mg/kg, respectively). The measured change in contractility as measured at 5 minute intervals (for 30 minutes total) revealed a dose dependent increase in contractility with an estimated EC_{50} of 49.33 mg/kg. The EF remained at an elevated state for at least 1 hour on subjects (n=5, dose of 200 mg/kg IV) that are evaluated for a longer period of time (average increase in EF over baseline of 8.9 ± 2.57)^[1].



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