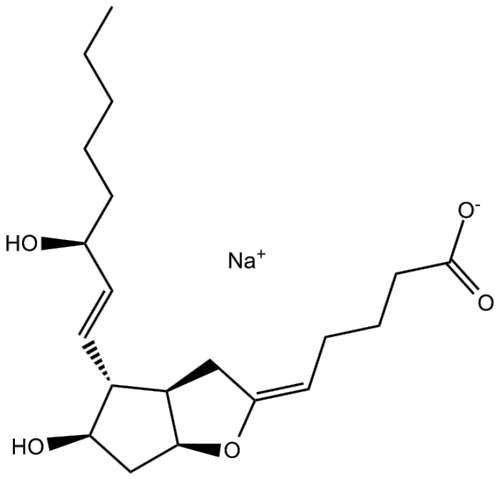
Product Data Sheet

Product Name: Epoprostenol
Cat. No.: GC12101

Chemical Properties

Cas. No.	61849-14-7		
Chemical Name	sodium (E)-5-((3aR,4R,5R,6aS)-5-hydroxy-4-((S,E)-3-hydroxyoct-1-en-1-yl)hexahydro-2H-cyclopenta[b]furan-2-ylidene)pentanoate		
SMILES	O[C@H]1[C@H](/C=C/[C@H](CCCCC)O)[C@@H](C/2)[C@H](C1)OC2=C\CCCC([O-])=O.[Na+]		
Formula	$C_{20}H_{31}NaO_5$	M.Wt	374.45
Solubility	DMF: 5 mg/ml,DMSO: 5 mg/ml,Ethanol: 10 mg/ml*,PBS (pH >10.2): 10 mg/ml,Water: 10 mg/ml	Storage	Desiccate at -80°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure



Protocol

Cell experiment [1]:

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Cell lines	Pulmonary artery smooth muscle cells (PASMCs)
Preparation Method	PASMCs were seeded in 6-well plates at a density of 5×10^5 cells/well. After incubation for 24h in DMEM medium supplemented with 10% Fetal bovine serum (FBS), Epoprostenol (0.1, 1, 5, and 10ng/ml) was added to the culture media. After incubation for 15minutes, cAMP was measured.
Reaction Conditions	0.1, 1, 5, and 10ng/ml; 15min
Applications	Epoprostenol treatment enhanced the production of cAMP in PASMCs.

References:
[1] Akagi S, Nakamura K, Matsubara H, et al. Prostaglandin I2 induces apoptosis via upregulation of Fas ligand in pulmonary artery smooth muscle cells from patients with idiopathic pulmonary arterial hypertension[J]. International journal of cardiology, 2013, 165(3): 499-505.

Background

Epoprostenol is a synthetic analogue of the naturally occurring eicosanoid prostacyclin [1]. The inhibitory effect of epoprostenol on platelet aggregation is mediated by the stimulation of adenylate cyclase and the subsequent increase of cyclic adenosine monophosphate-3', 5' (cAMP) in platelets, leading to a rise in the intracellular levels of cyclic AMP [2]. Epoprostenol is widely used in studies on pulmonary arterial hypertension (PAH) to promote vascular dilation[3].

In vitro, Epoprostenol treatment for 24h at 1.0ng/ml induced apoptosis in PAH-pulmonary artery smooth muscle cells (PASMCs), resulting in increased cAMP levels and upregulated FasL expression[4].

References:
[1] Provencher S, Paruchuru P, Spezzi A, et al. Quality of life, safety and efficacy profile of

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thermostable flolan in pulmonary arterial hypertension[J]. PLoS One, 2015, 10(3): e0120657.
[2] Mohammadi A, Matos W F, Intriago C, et al. Use of Epoprostenol in the treatment of pulmonary arterial hypertension[J]. Cureus, 2021, 13(9).
[3] Sitbon O, Noordegraaf A V. Epoprostenol and pulmonary arterial hypertension: 20 years of clinical experience[J]. European Respiratory Review, 2017, 26(143).
[4] Akagi S, Nakamura K, Matsubara H, et al. Prostaglandin I2 induces apoptosis via upregulation of Fas ligand in pulmonary artery smooth muscle cells from patients with idiopathic pulmonary arterial hypertension[J]. International journal of cardiology, 2013, 165(3): 499-505.

Caution: Product has not been fully validated for medical applications. For research use only.
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