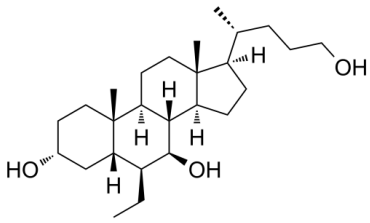
Data Sheet

Product Name:	BAR501
Cat. No.:	CS-6277
CAS No.:	1632118-69-4
Molecular Formula:	C26H46O3
Molecular Weight:	406.64
Target:	GPCR19
Pathway:	GPCR/G Protein
Solubility:	DMSO : ≥ 50 mg/mL (122.96 mM)



BIOLOGICAL ACTIVITY:

BAR501 is a potent and selective agonist of **GPBAR1** with an **EC_{50}** of 1 μM. IC50 & Target: EC50: 1 μM (GPBAR1)[1] **In Vitro:** BAR501 is a selective GPBAR1 agonist devoid of FXR agonistic activity. It effectively transactivates GPBAR1 in HEK293 cells overexpressing a CRE along with GPBAR1, with an EC_{50} of 1 μM. Exposure of GLUTAg cells to BAR501 (10 μM) increases the expression of GLP-1 mRNA by 2.5 folds[1]. **In Vivo:** Pretreating rats for 6 days with BAR501, 15 mg/kg, reduces basal portal pressure and blunts the vasoconstriction activity of norepinephrine. Pretreatment with BAR501 attenuates the hepatic vasomotor activity induced by shear stress and methoxamine. Administration of BAR501 exerts a direct vasodilatory activity in the CCl4 model. Treating mice with BAR501 at the dose of 15 mg/Kg reduces portal pressure and AST plasma levels. BAR501 attenuates endothelial dysfunction by regulating CSE expression/activity[1].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: [1]For GPBAR1 mediated transactivation, HEK-293T cells are plated at 10000 cells/well in a 24 well-plate and transfected with 200 ng of pGL4.29, a reporter vector containing a cAMP response element (CRE) that drives the transcription of the luciferase reporter gene luc2P, with 100 ng of pCMVSPORT6-human GPBAR1, and with 100 ng of pGL4.70. At 24 h post-transfection, HepG2 and HEK293T cells are incubated with 10 μM BAR501 for 18 h and luciferase activities are assayed and normalized against the Renilla activities[1]. **Animal Administration:** [1]Mouse: C57BL6 mice are administered i.p. 500 μL/Kg body weight of CCl4 in an equal volume of paraffin oil twice a week for 9 weeks. CCL4 mice are randomized to receive BAR501 (15 mg/Kg daily by gavage) or vehicle (distilled water). Serum bilirubin, albumin, aspartate aminotransferase, alanine aminotransferase and alkaline phosphatase are measured by routine biochemical clinical chemistry[1].

References:

[1]. Renga B, et al. Reversal of Endothelial Dysfunction by GPBAR1 Agonism in Portal Hypertension Involves a AKT/FOXOA1 Dependent Regulation of H2S Generation and Endothelin-1. PLoS One. 2015 Nov 5;10(11):e0141082.

CAIndexNames:

Cholane-3,7,24-triol, 6-ethyl-, (3α,5β,6β,7β)-

SMILES:

C[C@H](CCCO)[C@@]1([H])CC[C@@]2([H])[C@]3([H])[C@@H](O)[C@@H](CC)[C@]4([H])C[C@H](O)CC[C@]4(C)[C@@]3([H])CC[C@@]21C

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

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