

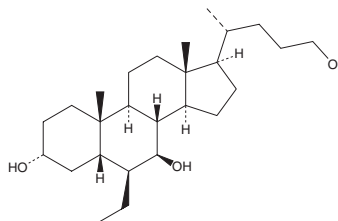
# PRODUCT INFORMATION



## BAR501

Item No. 22460

**CAS Registry No.:** 1632118-69-4  
**Formal Name:** (3 $\alpha$ ,5 $\beta$ ,6 $\beta$ ,7 $\beta$ )-6-ethyl-cholane-3,7,24-triol  
**MF:** C<sub>26</sub>H<sub>46</sub>O<sub>3</sub>  
**FW:** 406.6  
**Purity:**  $\geq 95\%$   
**Supplied as:** A crystalline solid  
**Storage:** -20°C  
**Stability:**  $\geq 4$  years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

### Laboratory Procedures

BAR501 is supplied as a crystalline solid. A stock solution may be made by dissolving the BAR501 in the solvent of choice. BAR501 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide, which should be purged with an inert gas. The solubility of BAR501 in these solvents is approximately 2, .1, and 20 mg/ml, respectively.

BAR501 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, BAR501 should first be dissolved in ethanol and then diluted with the aqueous buffer of choice. BAR501 has a solubility of approximately .5 mg/ml in a 1:2 solution of ethanol:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

### Description

BAR501 is a selective agonist of the G protein-coupled bile acid-activated receptor (GP-BAR1; EC<sub>50</sub> = 1  $\mu$ M) that lacks activity at the farnesoid X receptor (FXR).<sup>1</sup> *In vivo*, BAR501 reduces hepatic perfusion pressure and counteracts norepinephrine-induced vasoconstriction in naïve rats. It protects against development of endothelial dysfunction by increasing liver cystathione- $\gamma$ -liase (CSE) expression and activity, while reducing endothelin 1 (ET-1) gene expression in a rat model of cirrhosis. *In vitro*, BAR501 increases Akt-dependent phosphorylation of CSE and endothelial nitric oxide synthase (eNOS) and inhibits ET-1 transcription in human liver sinusoidal cells. BAR501 also shifts macrophages from the inflammatory (M1) phenotype to the anti-inflammatory (M2) phenotype and reduces trinitrobenzenesulfonic acid-induced colitis in mice in a dose-dependent manner.<sup>2</sup>

### References

1. Renga, B., Cipriani, C., Carino, A., *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* **10(11)** (2015).
2. Biagioli, M., Carino, A., Cipriani, S., *et al.* The bile acid receptor GPBAR1 regulates the M1/M2 phenotype of intestinal macrophages and activation of GPBAR1 rescues mice from murine colitis. *J. Immunol.* **199(2)**, 718-733 (2017).

#### WARNING

THIS PRODUCT IS FOR RESEARCH ONLY - NOT FOR HUMAN OR VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

#### SAFETY DATA

This material should be considered hazardous until further information becomes available. Do not ingest, inhale, get in eyes, on skin, or on clothing. Wash thoroughly after handling. Before use, the user must review the complete Safety Data Sheet, which has been sent via email to your institution.

#### WARRANTY AND LIMITATION OF REMEDY

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#### CAYMAN CHEMICAL

1180 EAST ELLSWORTH RD  
ANN ARBOR, MI 48108 · USA

**PHONE:** [800] 364-9897  
[734] 971-3335

**FAX:** [734] 971-3640

CUSTSERV@CAYMANCHEM.COM  
WWW.CAYMANCHEM.COM