SZABO SCANDIC

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Produktinformation

- Forschungsprodukte & Biochemikalien
- Zellkultur & Verbrauchsmaterial
- Diagnostik & molekulare Diagnostik
- Laborgeräte & Service

Weitere Information auf den folgenden Seiten!
See the following pages for more information!

Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

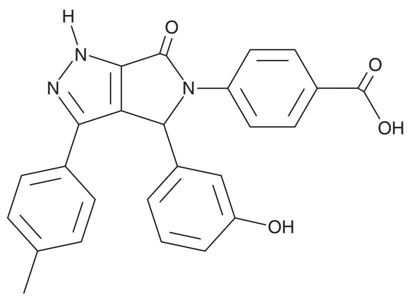
SZABO-SCANDIC HandelsgmbH
Quellenstraße 110, A-1100 Wien
T. +43(0)1 489 3961-0
F. +43(0)1 489 3961-7
mail@szabo-scandic.com
www.szabo-scandic.com

linkedin.com/company/szaboscandic

PRODUCT INFORMATION

CID16020046

Item No. 15247



CAS Registry No.: 834903-43-4
Formal Name: 4-[4,6-dihydro-4-(3-hydroxyphenyl)-3-(4-methylphenyl)-6-oxopyrrolo[3,4-c]pyrazol-5(1H)-yl]-benzoic acid
MF: $C_{25}H_{19}N_3O_4$
FW: 425.4
Purity: ≥98%
UV/Vis.: λ_{max}: 280 nm
Supplied as: A crystalline solid
Storage: -20°C
Stability: ≥2 years

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

Laboratory Procedures

CID16020046 is supplied as a crystalline solid. A stock solution may be made by dissolving the CID16020046 in the solvent of choice, which should be purged with an inert gas. CID16020046 is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of CID16020046 in these solvents is approximately 25 mg/ml.

CID16020046 is sparingly soluble in aqueous solutions. To enhance aqueous solubility, dilute the organic solvent solution into aqueous buffers or isotonic saline. If performing biological experiments, ensure the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. We do not recommend storing the aqueous solution for more than one day.

Description

CID16020046 is a GPR55 inverse agonist that antagonizes GPR55 constitutive activity with an IC_{50} value of 15 µM.[1] It inhibits GPR55-mediated ERK1/2 phosphorylation, LPI-induced Ca^{2+} signaling (IC_{50} = 0.21 µM in HEK-GPR55 cells), and GPR55-mediated transcription factor activation.[1] It does not affect ERK1/2 phosphorylation or transcription factor activation in CB receptor expressing cells and demonstrates weak activity against a broad spectrum of other GPCRs, ion channels, kinases, and nuclear receptors.[1] This compound has been shown to block GPR55-mediated endothelial wound healing and reverse LPI-inhibited platelet aggregation.[1]

Reference

1. Kargl, J., Brown, A.J., Andersen, L., *et al*. A selective antagonist reveals a potential role of G protein-coupled receptor 55 in platelet and endothelial cell function. *J. Pharmacol. Exp. Ther*. **346**, 54-66 (2013).

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