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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

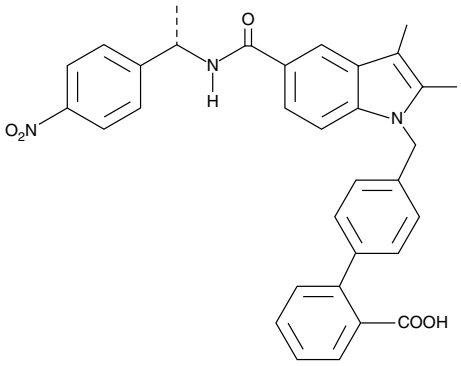
linkedin.com/company/szaboscandic

Product Information

SR 1664

Item No. 11086

CAS Registry No.: 1338259-05-4
Formal Name: 4'-[[2,3-dimethyl-5-[[[(1S)-1-(4-nitrophenyl)ethyl]amino]carbonyl]-1H-indol-1-yl]methyl]-[1,1'-biphenyl]-2-carboxylic acid
MF: $C_{33}H_{29}N_3O_5$
FW: 547.6
Purity: ≥98%
Stability: ≥2 years at -20°C
Supplied as: A crystalline solid
UV/Vis.: λ_{max}: 252 nm



Laboratory Procedures

For long term storage, we suggest that SR 1664 be stored as supplied at -20°C. It should be stable for at least two years.

SR 1664 is supplied as a crystalline solid. A stock solution may be made by dissolving the SR 1664 in the solvent of choice. SR 1664 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of SR 1664 in ethanol and DMSO is approximately 15 mg/ml and approximately 20 mg/ml in DMF.

SR 1664 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, SR 1664 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. SR 1664 has a solubility of approximately 0.5 mg/ml in a 1:1 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Apart from direct peroxisome proliferator-activated receptor γ (PPARγ) agonism, several PPARγ ligands have recently been shown to exert anti-diabetic effects through a second, distinct biochemical function: blocking the obesity-linked phosphorylation of PPARγ by cyclin-dependent kinase 5 (Cdk5) at serine 273.[1] This effect requires binding to the PPARγ ligand binding domain, which causes a conformational change that interferes with the ability of Cdk5 to phosphorylate serine 273. SR 1664 is a small molecule that blocks phosphorylation of PPARγ by Cdk5 with an IC_{50} value of 80 nM (K_i = 28.7 nM) without exhibiting agonist activity at the PPARγ receptor.[2] It demonstrates potent, dose-dependent anti-diabetic effects in obese mice without inducing fluid retention and weight gain or inhibiting bone formation.[2]

References

1. Norris, A.W. and Sigmund, C.D. A second chance for a PPARγ targeted therapy?: A commentary on "antidiabetic actions of a non-agonist PPARγ ligand blocking Cdk5-mediated phosphorylation". *Circ. Res.* **110(1)**, 8-11 (2012).
2. Choi, J.H., Banks, A.S., Kamenecka, T.M., *et al.* Anti-diabetic actions of a non-agonist PPARγ ligand blocking Cdk5-mediated phosphorylation. *Nature* **477(7365)**, 477-481 (2012).

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Cayman Chemical

Mailing address
1180 E. Ellsworth Road
Ann Arbor, MI
48108 USA

Phone
(800) 364-9897
(734) 971-3335

Fax
(734) 971-3640

E-Mail
custserv@caymanchem.com

Web
www.caymanchem.com