Product Data Sheet

Product Name: G3335
Cat. No.: GC15085

Chemical Properties

Cas. No.	36099-95-3		
Chemical Name	L-tryptophyl-L-glutamic acid		
SMILES	OC(CC[C@@H](C(O)=O)NC([C@@H](N)CC1=CNC2=C1C=CC=C2)=O)=O		
Formula	$C_{16}H_{19}N_3O_5$	M.Wt	333.3
Solubility	≥ 14.35mg/mL in DMSO	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ 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	G3335		

Background

Kd = 8.34 μM

G3335 is a PPARγ antagonist.

The peroxisome proliferator-activated receptor gamma (PPARgamma) is a key therapeutic drug target for several conditions, such as inflammation, diabetes, hypertension, dyslipidemia, and cancer.

In vitro: Biacore 3000 study based on the surface plasmon resonance technique found that G3335 exhibited a highly specific binding affinity against PPARgamma and was able to block rosiglitazone, a potent PPARgamma agonist, in the stimulation of the interaction between the PPARgamma ligand-binding domain (LBD) and RXRalpha-LBD. Moreover, the yeast two-hybrid assays indicated that G3335 had strong antagonistic activity in perturbing rosiglitazone in the promotion of the PPARgamma-LBD-CBP interaction. In addition, G3335 could competitively bind to PPARgamma against 0.1 microM

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA

rosiglitazone to repress reporter-gene expression [1].

In vivo: In a previous study, the effect of rosiglitazone was examined on spinal cord injury (SCI) in rats. The animals were randomly divided into vehicle group, rosiglitazone treated group, and G3335 treated group. Locomotor function recovery was evaluated. Results showed that compared with the vehicle groups, the rosiglitazone could significantly ameliorate locomotor recovery, reduce NF-κB expression, and increase the proliferation of endogenous NPCs. In addition, when the PPAR-γ antagonist G3335 was applied, such effects were abolished [2].

Clinical trial: So far, no clinical study has been conducted.

References:
[1] Ye, F. ,Zhang, Z.S.,Luo, H.B., et al. The dipeptide H-Trp-Glu-OH shows highly antagonistic activity against PPARγ: Bioassay with molecular modeling simulation. ChemBioChem 7, 74-82 (2006).
[2] Meng, Q. Q.,Liang, X.J.,Wang, P., et al. Rosiglitazone enhances the proliferation of neural progenitor cells and inhibits inflammation response after spinal cord injury. Neuroscience Letters 503, 191-195 (2011).

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