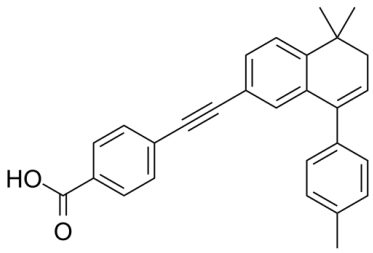
Data Sheet

Product Name:	AGN 193109
Cat. No.:	CS-0035405
CAS No.:	171746-21-7
Molecular Formula:	C28H24O2
Molecular Weight:	392.49
Target:	Autophagy; RAR/RXR
Pathway:	Autophagy; Metabolic Enzyme/Protease
Solubility:	DMSO : 2 mg/mL (5.10 mM; Need warming)



BIOLOGICAL ACTIVITY:

AGN 193109 is a retinoid analog, and acts as a specific and highly effective antagonist of **retinoic acid receptors (RARs)**, with **K_d**s of 2 nM, 2 nM, and 3 nM for **RARα**, **RARβ**, and **RARγ**, respectively. IC50 & Target: Kd: 2 nM (RARα), 2 nM (RARβ), 3 nM (RARγ)[1] **In Vitro:** AGN 193109 is a highly effective antagonist of retinoic acid receptors, with K_ds of 2 nM, 2 nM, and 3 nM for RARα, RARβ, and RARγ, respectively. AGN 193109 is completely RAR specific, because it does not bind to or transactivate through any of the RXRs[1]. AGN 193109 (100 nM) inhibits the TTNPB (a retinoic acid receptor agonist)-dependent morphological change in ECE16-1 cells. AGN193109 half-reverses retinoid-dependent growth suppression at 10 nM, and completely shows this effect at 100 nM in ECE16-1 cells. AGN193109 (100 nM) also eliminates TTNPB-induced decrease in levels of K5, K6, K14, K16, and K17 and increase in levels of K7, K8, and K19[2]. **In Vivo:** AGN 193109 (1.15 μmol/kg) does not causes overt toxicity and has no effect on spleen weight on the mice, but it suppresses TTNPB-induced increase in spleen weight of the mice. AGN 193109 also significantly reduces the cutaneous toxicity induced by ATRA. AGN 193109 (0.30 or 1.20 μmol/kg) by topical treatment significantly reduces both weight loss and cutaneous toxicity caused by oral TTNPB cotreatment[3].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: AGN 193109 is dissolved in DMSO.[2]**Cells (10,000/cm^2)** are seeded in complete medium and allowed to attach overnight. The cells are then shifted to **defined medium (DM)**, allowed to equilibrate for **24 h**, and treatment is initiated by addition of fresh DM or DM containing epidermal growth factor (EGF) or retinoid. After 3 days of daily treatment with retinoid, the cells are harvested with 0.025% trypsin, 1 mM EDTA, fixed in isotonic buffer containing 4% formaldehyde, and counted using a counter[2]. **Animal Administration:** [3]**Mice** (n=6) are treated topically on the dorsal skin with vehicle (**92.5% acetone/7.5% DMSO**), 0.072 μmol/kg of TTNPB, **1.15 μmol/kg of AGN 193109**, or 0.072 μmol/kg of TTNPB plus **0.072, 0.288, or 1.15 μmol/kg of AGN 193109** for 5 days. Mice are euthanized on Day 8[3].

References:

[1]. Johnson AT, et al. Synthesis and characterization of a highly potent and effective antagonist of retinoic acid receptors. J Med Chem. 1995 Nov 24;38(24):4764-7.

[2]. Agarwal C, et al. AGN193109 is a highly effective antagonist of retinoid action in human ectocervical epithelial cells. J Biol Chem. 1996 May 24;271(21):12209-12.

[3]. Standeven AM, et al. Specific antagonist of retinoid toxicity in mice. Toxicol Appl Pharmacol. 1996 May;138(1):169-75.

CAIndexNames:

Benzoic acid, 4-[2-[5,6-dihydro-5,5-dimethyl-8-(4-methylphenyl)-2-naphthalenyl]ethynyl]-

SMILES:

O=C(O)C1=CC=C(C#CC2=CC=C3C(C)(C)CC=C(C4=CC=C(C)C=C4)C3=C2)C=C1

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