

Product Data Sheet

Product Name: TTNPB (Arotinoid Acid)

Cat. No.: GC14273

Chemical Properties

Cas. No. 71441-28-6

Chemical Name 4-[(E)-2-(5,5,8,8-tetramethyl-6,7-dihydronaphthalen-2-yl)prop-1-enyl]benzoic acid

SMILES CC(=CC1=CC=C(C=C1)C(=O)O)C2=CC3=C(C=C2)C(CCC3(C)C)(C)C

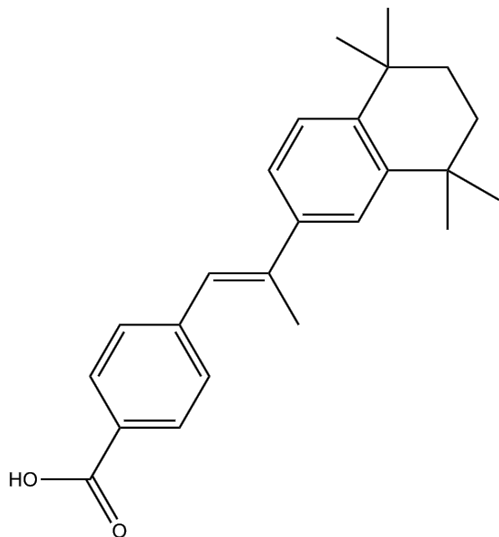
Formula $C_{24}H_{28}O_2$ M.Wt 348.48

Solubility $\geq 17.4\text{mg/mL}$ in DMSO Storage 4°C , protect from light

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C 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



Protocol

Cell experiment [1]:

Caution: Product has not been fully validated for medical applications. For research use only.

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Cell lines THP-1 cells (human monocytic leukemia cell line)

Preparation Method THP-1 cells were treated with TTNPB (5μM) for 24 or 48 hours.

Reaction
Conditions 5μM; 24h or 48h

Applications TTNPB increased CD36 mRNA and surface protein levels. Induction of CD36 by TTNPB was not dependent on PKC activation.

Animal experiment [2]:

Animal models C57B1xDBA/2f/F1 mice bearing MXT-HI and MXT-HS mammary adenocarcinomas

Preparation Method Mice were intraperitoneally administered 0.25mg/kg TTNPB from the 7th to the 21st days post-tumor graft (15 administrations total). Mice were sacrificed at the 21st day post-graft for tumor collection and analysis.

Dosage form 0.25mg/kg; i.p.; 15 administrations

Applications TTNPB significantly inhibited tumor growth in both MXT-HI and MXT-HS models. TTNPB was markedly more efficient than tamoxifen in inhibiting cell proliferation and triggering cell death (apoptosis) in both tumor models. The TTNPB-induced inhibition of macroscopic tumor growth rate relates to the triggering of apoptosis rather than to an inhibition of cell proliferation.

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References:

- [1] Han S, Sidell N. Peroxisome-proliferator-activated-receptor gamma (PPARgamma) independent induction of CD36 in THP-1 monocytes by retinoic acid. *Immunology*. 2002 May;106(1):53-9.
- [2] Darro F, Cahen P, Vianna A, et al. Growth inhibition of human in vitro and mouse in vitro and in vivo mammary tumor models by retinoids in comparison with tamoxifen and the RU-486 anti-progestagen. *Breast Cancer Res Treat*. 1998 Sep;51(1):39-55.

Background

TTNPB (Arotinoid Acid) is a selective retinoic acid receptor (RAR) agonist. TTNPB competitively binds to RAR α (IC₅₀=5.1nM), RAR β (IC₅₀=4.5nM), and RAR γ (IC₅₀=9.3nM). TTNPB can be used in research related to cancer development and stem cell differentiation and reprogramming^[1-4].

In vitro, Caco-2 cells were treated with TTNPB (0.1-10 μ M) for 48 hours. TTNPB significantly inhibited UGT2B7 mRNA expression^[5]. THP-1 cells were treated with TTNPB (5 μ M) for 24 or 48 hours. TTNPB significantly upregulated CD36 mRNA and surface protein expression independently of PKC activation^[6].

In vivo, TTNPB (0.25mg/kg) was intraperitoneally administered to mice mammary tumor models bearing MXT-HI and MXT-HS from the 7th to the 21st day post-graft (15 administrations total). TTNPB significantly inhibited tumor growth and induced apoptosis^[7]. TTNPB (0.072 μ mol/kg/day) was topically applied to the backs of female hairless mice (CrI:SKH1-hrBR) for 5 consecutive days. TTNPB caused skin desquamation, excoriation, body weight loss, and splenomegaly in mice^[8].

References:

- [1] Pignatello MA, Kauffman FC, Levin AA. Multiple factors contribute to the toxicity of the aromatic retinoid, TTNPB (Ro 13-7410): binding affinities and disposition. *Toxicol Appl Pharmacol*. 1997 Feb;142(2):319-27.
- [2] Pignatello MA, Kauffman FC, Levin AA. Multiple factors contribute to the toxicity of the aromatic retinoid TTNPB (Ro 13-7410): interactions with the retinoic acid receptors. *Toxicol Appl Pharmacol*. 1999 Sep 1;159(2):109-16.
- [3] Nath SC, Babaei-Abraki S, Meng G, et al. A retinoid analogue, TTNPB, promotes clonal expansion of human pluripotent stem cells by upregulating CLDN2 and HoxA1. *Commun Biol*. 2024

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Feb 16;7(1):190.

[4] Hammond LA, Van Krinks CH, Durham J, et al. Antagonists of retinoic acid receptors (RARs) are potent growth inhibitors of prostate carcinoma cells. Br J Cancer. 2001 Aug 3;85(3):453-62.

[5] Lu Y, Bratton S, Heydel JM, et al. Effect of retinoids on UDP-glucuronosyltransferase 2B7 mRNA expression in Caco-2 cells. Drug Metab Pharmacokinet. 2008;23(5):364-72.

[6] Han S, Sidell N. Peroxisome-proliferator-activated-receptor gamma (PPARgamma) independent induction of CD36 in THP-1 monocytes by retinoic acid. Immunology. 2002 May;106(1):53-9.

[7] Darro F, Cahen P, Vianna A, et al. Growth inhibition of human in vitro and mouse in vitro and in vivo mammary tumor models by retinoids in comparison with tamoxifen and the RU-486 anti-progestagen. Breast Cancer Res Treat. 1998 Sep;51(1):39-55.

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

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