



AM580

Catalog No: tcsc3251

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Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg



Specifications

CAS No:

102121-60-8

Formula:

 $C_{22}H_{25}NO_3$

Pathway:

Metabolic Enzyme/Protease; Autophagy

Target:

RAR/RXR; Autophagy

Purity / Grade:

>98%

Solubility:

DMSO : \geq 45 mg/mL (128.04 mM); H2O :

Alternative Names:

CD336;NSC608001;Ro 40-6055

Observed Molecular Weight:

351.44

Product Description





AM580 is a stable retinobenzoic derivative, and originally synthesized as a $RAR\alpha$ agonist.

In Vitro: In the presence of G-CSF, AM580 (at 10^{-8} M) produces a remarkable induction in LAP mRNA of NB4 cells. At a concentration of 10^{-5} M, AM580 and ATRA, in combination with G-CSF, induce almost the same level of LAP transcript. AM580 (at 10^{-8} M) leads to an approximately sixfold increase in the steady-state levels of the transcript coding for the G-CSF receptor in NB4 cells^[1]. AM580 (50 nM) increases caspase-3 expression in all of the colonies, and in 30% of the colonies induce acinar-like cavitation^[2]. Knockdown of *RARγ1* in primary Myc cells using shRARγ1 followed by Am580 treatment results in an even higher level of *CRBP1* expression, showing that in these cells RARγ has a repressive effect on the RARα target gene *CRBP1*. Am580 (200 nM) enhances the anti-proliferative effect exhibited by *RARγ* knockdown in the MCF-10A and MCF-7 cell lines but not in the MDA-MB-231 cells^[3].

In Vivo: Am580 (0.3 mg/kg/day) treatment has a more profound effect on tumor-free survival of MMTV-wnt1 mice, the effect being noticeable even in early appearing tumors, and no overt toxicity is found in liver, lungs, kidney, and spleen. Am580 treatment reduces substantially and equally the level of hyperplasia in both transgenic glands^[2]. Treatment of MMTV-Myc mice with the RAR α -selective agonist Am580 leads to significant inhibition of mammary tumor growth, lung metastasis and extends tumor latency in 63% of mice^[3].

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