Propafenone

Cat. No.: HY-B0432

CAS No.: 54063-53-5

Molecular Formula: $C_{21}H_{27}NO_3$

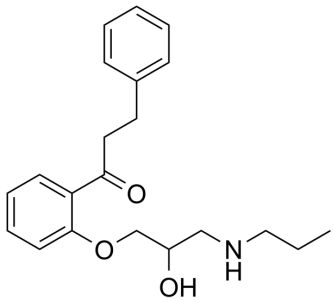
Molecular Weight: 341.44

Target: Sodium Channel; Adrenergic Receptor; Potassium Channel

Pathway: Membrane Transporter/Ion Channel; GPCR/G Protein; Neuronal Signaling

Storage: 4°C, protect from light

* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (366.10 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.9288 mL	14.6439 mL	29.2877 mL
	5 mM	0.5858 mL	2.9288 mL	5.8575 mL
	10 mM	0.2929 mL	1.4644 mL	2.9288 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

Propafenone (SA-79), a sodium-channel blocker, acts an antiarrhythmic agent. Propafenone also has high affinity for the β receptor (IC_{50}=32 nM)[1]. Propafenone blocks the transient outward current (Ito) and the sustained delayed rectifier K current (Isus) with IC_{50} values of 4.9 μm and 8.6 μm, respectively[2]. Propafenone suppresses esophageal cancer proliferation through inducing mitochondrial dysfunction and induce apoptosis[3].

In Vitro

Propafenone (5-25 μM) inhibits esophageal squamous cell carcinoma (ESCC) cell proliferation[3].
Propafenone causes mitochondrial dysfunction by a decreased mitochondrial membrane potential and reduced expression of Bcl-xL and Bcl-2[3].
Propafenone (10 and 20 μm) treatment significantly down regulates the expression levels of the anti-apoptotic proteins Bcl-xL and Bcl-2 in ESCC cells. Propafenone also reduces expression of p-ERK[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[3]

Cell Line:	The human ESCC cell lines KYSE30, KYSE150 and KYSE270
Concentration:	5, 10, 15, 20, and 25 μm

Incubation Time:	24, 48, and 72 hours
Result:	Gradually decreased cell proliferation over time and potently inhibited cell proliferation with increasing concentrations in KYSE30, KYSE150 and KYSE270 cells.

Western Blot Analysis[3]

Cell Line:	The human ESCC cell lines KYSE30, KYSE150 and KYSE270
Concentration:	0, 10, and 20 μm
Incubation Time:	72 hours
Result:	Significant downregulation of Bcl-xL and Bcl-2 expression levels was observed.

In Vivo

Propafenone (20 mg/kg; intraperitoneal injection every other day) markedly suppresses the tumor burden with a decrease of 69.2%[3].

Propafenone also significantly inhibits tumor cell proliferation (mean index decreased from 56.3±6.7% in the DMSO-treated group to 20.7±5.1% in the 10 mg/kg propafenone-treated group and 11.3±4.0% in the 20 mg/kg propafenone-treated group) [3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Female BALB/c nude mice bearing KYSE270-derived xenografts (6-8 weeks)[3]
Dosage:	10 mg/kg or 20 mg/kg
Administration:	Intraperitoneally injected
Result:	Exerted a significantly inhibitory effect on the growth of tumor xenografts.

REFERENCES

[1]. J T Lee, et al. The role of genetically determined polymorphic drug metabolism in the beta-blockade produced by propafenone. N Engl J Med. 1990 Jun 21;322(25):1764-8.

[2]. A Seki, et al. Effects of propafenone on K currents in human atrial myocytes. Br J Pharmacol. 1999 Mar;126(5):1153-62.

[3]. Wei-Bin Zheng, et al. Propafenone suppresses esophageal cancer proliferation through inducing mitochondrial dysfunction. Am J Cancer Res. 2017 Nov 1;7(11):2245-2256.

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