Rafoxanide

Cat. No.: HY-17598

CAS No.: 22662-39-1

Molecular Formula: $C_{19}H_{11}Cl_2I_2NO_3$

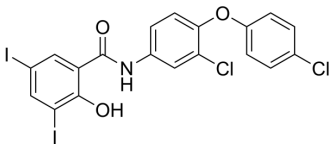
Molecular Weight: 626.01

Target: Parasite; p38 MAPK; Raf; Apoptosis; Oxidative Phosphorylation

Pathway: Anti-infection; MAPK/ERK Pathway; Apoptosis; Others

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 31 mg/mL (49.52 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.5974 mL	7.9871 mL	15.9742 mL
5 mM	0.3195 mL	1.5974 mL	3.1948 mL
10 mM	0.1597 mL	0.7987 mL	1.5974 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (3.99 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.99 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Rafoxanide is a poent, orally active halogenated salicylaniline agent with antiparasitic activity. Rafoxanide interferes with energy metabolism in trematodes by uncoupling oxidative phosphorylation. Rafoxanide is also found to be a potent inhibitor of the BRAF V600E mutant protein, which is important in colorectal cancer. Rafoxanide can be used for the control of infestation with Hemonchus species or Fasciola species in sheep and cattle as well as Oestrus ovis in sheep. Rafoxanide can also be used for cancer research[1][2][3].

IC_{50} & Target

Schistosome

In Vitro

Rafoxanide (1.25-5 μM, 24 h) inhibits ERK activation of HT-29 cells and proliferation of HT-29, HCT-116 and DLD-1 in a dose-

dependent fashion while doesn't significantly affect the proliferation of the human normal colon epithelial cell lines HCEC-1CT and NCM460[3].
Rafoxanide (1.25-5 μM, 24 h) blocks HCT-116 and DLD-1 at G0/G1 phase while down-regulating the level of cyclin D1 through inducing ERS[3].
Rafoxanide (1.25-5 μM, 48 h) activates programmed cell death of HCT-116 and DLD-1[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[3]

Cell Line:	HCT-116
Concentration:	1.25, 2.5, 5μM
Incubation Time:	24 h
Result:	Increased phosphorylate eIF2α and CHOP, which is the hallmark feature of ERS.

Apoptosis Analysis[3]

Cell Line:	HCEC-1CT, HCT-116 and DLD-1
Concentration:	1.25, 2.5, 5μM
Incubation Time:	24, 48, 60 h
Result:	Detected cell death of HCT-116 and DLD-1 from 48 h while no significant cell death was observed in HCEC-1CT. Pre-incubation of CRC cells with the pan-caspase inhibitor Q-VD-OPH totally reverted the rafoxanide-induced cell death, indicating the involvement of apoptotic pathways.

In Vivo

Rafoxanide (7.5 mg/kg, i.p., once every 2 d for 88d) reduces colonic tumorgenesis in $Apc^{min/+}$ mice[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	AOM induced $Apc^{min/+}$ mice[3]
Dosage:	7.5 mg/kg
Administration:	Intraperitoneal injection (i.p.), once every 2 d for 88d
Result:	Exerted less number and size of AOM induced lesions in the colon. No significant body weight changes were observed. Detected adenomas (28.6%), advanced adenomas (67.8%) and adenocarcinoma (3.6%) in control group as adenomas (33.3%), advanced adenomas (25%%) and no obvious lesions(41.6%) were detected in treated group. Caused increased signal for p-eIF2α, CHOP and cleaved caspase-3 in tumor tissues while these signal in non-tumor colon epithelium were barely detectable.

CUSTOMER VALIDATION

- Int J Mol Sci. 2022, 23(20), 12100.
- Exp Cell Res. 2019 Dec 15;385(2):111691.
- L'UNIVERSITE GRENOBLE ALPES. 2021 Apr 13.

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REFERENCES

[1]. El-Banna HA, et, al. Comparative pharmacokinetics of ivermectin alone and a novel formulation of ivermectin and rafoxanide in calves and sheep. Parasitol Res. 2008 May;102(6):1337-42.

[2]. van Wyk JA, et, al. Two field strains of Haemonchus contortus resistant to rafoxanide. Onderstepoort J Vet Res. 1987 Jun;54(2):143-6.

[3]. Laudisi F, et al. Induction of endoplasmic reticulum stress and inhibition of colon carcinogenesis by the anti-helmintic drug rafoxanide. Cancer Lett. 2019;462:1-11.

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