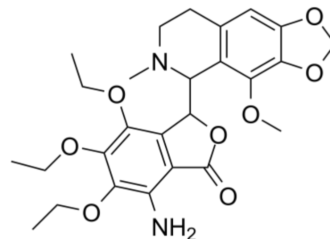
Tritoqualine

Cat. No.:	HY-U00065
CAS No.:	14504-73-5
Molecular Formula:	$C_{26}H_{32}N_2O_8$
Molecular Weight:	500.54
Target:	Others
Pathway:	Others
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Tritoqualine is used as a histidine decarboxylase inhibitor.
IC_{50} & Target	histidine decarboxylase[1]
In Vitro	The effect of Tritoqualine (TRQ) on the CCl_4-induced enzyme leakage is investigated by pretreatment of rats with Tritoqualine (200 mg/kg/day, p.o., 7 days). The ratio of lactate dehydrogenase (LDH) release from the cells of Tritoqualine-pretreated rats is significantly less than that in control rats. The rate of malondialdehyde (MDA) production is accelerated after the addition of 7.8 mM CCl_4. Tritoqualine reduces its rate in a dose dependent manner, and it completely prevents CCl_4 -stimulated lipid peroxidation at a concentration of 33 μM[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	A single administration of CCl_4 (0.75 mL/kg, p.o.) causes a five-fold increase of in vivo lipid peroxidation in the liver. In contrast, a reduction of 37% in the lipid peroxidation is obtained by Tritoqualine (100 mg/kg) pretreatment for 14 days prior to CCl_4 treatment. A 63% reduction is observed in vitamin E (25 mg/kg) pretreated rats[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [2]	Tritoqualine (TRQ) is suspended in 0.2% Tween 80 solution[2]. Male Wistar rats weighing about 150 g are used. Tritoqualine (TRQ) is given to the rats (200 mg/kg, p.o.) daily for 7 days. Hepatocytes are isolated from a perfused liver by a modified Seglen's procedure. Briefly, perfusion of the liver is started with 150 mL of a 10 mM HEPES-buffered Ca^{2+} free Hanks' solution (pH 7.4) containing 0.5 mM EGTA. After 5 min, the liver is excised, and the perfusate is changed to 100 mL of a recirculating HEPES-buffered Hanks' solution (pH 7.4) containing 5 mM $CaCl_2$, 0.05% collagenase and 0.005% soybean trypsin inhibitor instead of EGTA. The process continues for 15-20 min at 37°C with aeration of carbogen (95% O_2, 5% CO_2), until the liver began to disintegrate visibly. Cells are dispersed in a HEPES-buffered Hanks' solution (pH 7.4), filtered through a nylon stocking, and sedimented by triple centrifugation for 1 min at 50×g. Finally, the hepatocytes are suspended at 5×10^5 cells/mL in the same solution supplemented with 2% BSA. Two mL of the cell suspension is placed on 35 mm diameter culture dishes and incubated in an incubator at 37 °C. CCl_4 is diluted with ethanol (10%, v/v) and added to the dishes at a concentration of 2.6 or 5.2 mM (0.25 or 0.5%, v/v, respectively). As a marker of cell membrane damage, lactate dehydrogenase (LDH) leaked out from the cells is determined using the supernatant of centrifuged samples. Total LDH activity is measured after lysis of the cells by sonication. The results are expressed as a ratio

	of LDH release induced by CCl_4 to total LDH in the cells[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Rats[2] Tritoqualine (TRQ) is given p.o. to rats weighing about 200 g daily for 14 days. The same amount of 0.2% Tween 80 solution is given to the other rats. CCl_4 diluted with olive oil (37.5%, v/v) is given to the rats (0.75 mL/kg, i.p.) 4 hr after the last administration of these compounds. The animals are killed 20 hr after the injection of CCl_4. Serum is obtained from arterial blood. The liver is washed with 1.15% KCl solution through the portal vein, excised and homogenized in ten volumes of the same solution. Lipid peroxidation in the liver homogenates is quantified by the 1 % phosphoric acid method. All manipulations are done rapidly on ice to avoid further peroxidation. Glutamic oxaloacetic transaminase (GOT) and glutamic pyruvic transaminase (GPT) in the serum are measured using commercial test kits. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Ishii K, et al. Therapeutic effect of histidine decarboxylase inhibitor on chronic active hepatitis. Gastroenterol Jpn. 1978;13(2):105-10.

[2]. Yuasa S, et al. Suppressive effect of tritoqualine on lipid peroxidation and enzyme leakage induced by carbon tetrachloride in rat hepatocytes. Jpn J Pharmacol. 1986 Jun;41(2):205-10.

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

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