Doxapram hydrochloride hydrate

Cat. No.: HY-B0551A

CAS No.: 7081-53-0

Molecular Formula: $C_{24}H_{33}ClN_2O_3$

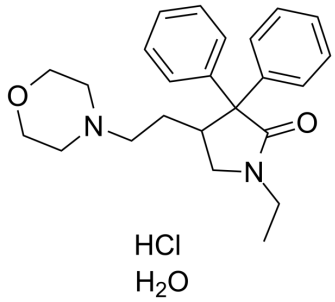
Molecular Weight: 432.98

Target: Potassium Channel

Pathway: Membrane Transporter/Ion Channel

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 55 mg/mL (127.03 mM)

H_2O : 25 mg/mL (57.74 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3096 mL	11.5479 mL	23.0958 mL
	5 mM	0.4619 mL	2.3096 mL	4.6192 mL
	10 mM	0.2310 mL	1.1548 mL	2.3096 mL

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

BIOLOGICAL ACTIVITY

Description

Doxapram hydrochloride hydrate inhibits TASK-1, TASK-3, TASK-1/TASK-3 heterodimeric channel function with EC50 of 410 nM, 37 μM, 9 μM, respectively.Target: Potassium ChannelDoxapram is a respiratory stimulant. Doxapram (15-150 microM) also evoked 3H overflow in a concentration dependent manner, and doxapram-evoked release was inhibited by the Ca2+ channel blocker nifedipine (5 microM). Analysis of released tritiated compounds suggested that doxapram preferentially stimulated the release of dopamine. Our results indicate that the mechanism of action of doxapram shares similarities with that of hypoxia in the carotid body [1]. Doxapram (1-100 microM) caused rapid, reversible and dose-dependent inhibitions of K+ currents recorded in type I cells (IC50 approximately 13 microM). doxapram was also seen to directly inhibit Ca(2+)-independent K+ currents. Doxapram was a more potent inhibitor of the Ca(2+)-activated K+ currents recorded under control conditions. Doxapram (10 microM) was without effect on L-type Ca2+ channel currents recorded under conditions where K+ channel activity was minimized and was also without significant effect on K+ currents recorded in the neuronal cell line NG-108 15, suggesting a selective effect on carotid body type I cells. The effects of doxapram on type I cells show similarities to those of the physiological stimuli of the carotid body, suggesting that doxapram may share a similar mechanism of action in stimulating the intact organ [2].

REFERENCES

[1]. Cotten JF, et al. The ventilatory stimulant doxapram inhibits TASK tandem pore (K2P) potassium channel function but does not affect minimum alveolar anesthetic concentration. Anesth Analg, 2006, 102(3), 779-785.

[2]. Anderson-Beck, R., et al., Doxapram stimulates dopamine release from the intact rat carotid body in vitro. Neurosci Lett, 1995. 187(1): p. 25-8.

[3]. Peers, C., Effects of doxapram on ionic currents recorded in isolated type I cells of the neonatal rat carotid body. Brain Res, 1991. 568(1-2): p. 116-22.

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