

# Doxapram (hydrochloride hydrate)

Catalog No: tcsc2655



## Available Sizes

**Size:** 50mg

**Size:** 100mg



## Specifications

**CAS No:**

7081-53-0

**Formula:**

$C_{24}H_{33}ClN_2O_3$

**Pathway:**

Membrane Transporter/Ion Channel

**Target:**

Potassium Channel

**Purity / Grade:**

>98%

**Solubility:**

DMSO :  $\geq 55$  mg/mL (127.03 mM)

**Observed Molecular Weight:**

432.98

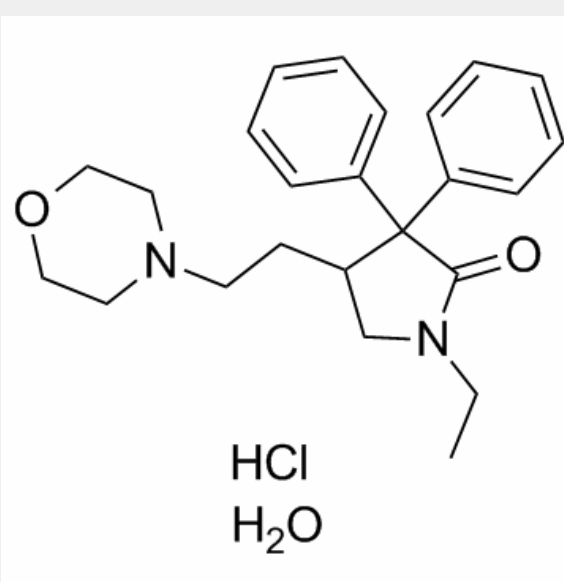
## Product Description

Doxapram hydrochloride hydrate inhibits TASK-1, TASK-3, TASK-1/TASK-3 heterodimeric channel function with EC50 of 410 nM, 37  $\mu$ M, 9  $\mu$ M, respectively.

Target: Potassium Channel

Doxapram 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 Ca<sup>2+</sup> 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<sup>+</sup> currents recorded in type I cells (IC<sub>50</sub> approximately 13 microM). doxapram was also seen to directly inhibit Ca(2+)-independent K<sup>+</sup> currents. Doxapram was a more potent inhibitor of the Ca(2+)-activated K<sup>+</sup> currents recorded under control conditions. Doxapram (10 microM) was without effect on L-type Ca<sup>2+</sup> channel currents recorded under conditions where K<sup>+</sup> channel activity was minimized and was also without significant effect on K<sup>+</sup> 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].



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