Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

# Conivaptan (hydrochloride)

## Catalog No: tcsc2015

### Available Sizes

**Size:** 10mg

**Size:** 50mg

**Size:** 100mg

### Specifications

**CAS No:**
168626-94-6

**Formula:**
$C_{32}H_{27}ClN_4O_2$

**Pathway:**
GPCR/G Protein

**Target:**
Vasopressin Receptor

**Purity / Grade:**
>98%

**Solubility:**
DMSO : ≥ 100 mg/mL (186.90 mM)

**Alternative Names:**
YM 087

**Observed Molecular Weight:**
535.04

## Product Description

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Conivaptan (hydrochloride) is a non-peptide antagonist of **vasopressin receptor**, with $\mathbf{K_i}$ values of 0.48 and 3.04 nM for rat liver V1A receptor and rat kidney V2 receptor respectively.

IC50 & Target: Ki: 0.48 nM (V1A receptor), 3.04 nM (V2 receptor)

***In Vivo:*** Conivaptan (0.03, 0.1 and 0.3 mg/kg, i.v.) dose-dependently increases urine volume and reduces urine osmolality in both myocardial infarction and sham-operated rats. Conivaptan (0.3 mg/kg i.v.) significantly reduces right ventricular systolic pressure, left ventricular end-diastolic pressure, lung/body weight and right atrial pressure in myocardial infarction rats. Conivaptan (0.3 mg/kg i.v.) significantly increases dP/dt(max)/left ventricular pressure in myocardial infarction rats[1]. Conivaptan produces an acute increase in urine volume (UV), a reduction in osmolality (UOsm) and, at the end of the investigation, cirrhotic rats receiving the V(1a)/V(2)-AVP receptor antagonist does not show hyponatremia or hypoosmolality. Conivaptan also normalizes U(Na)V without affecting creatinine clearance and arterial pressure[2]. Conivaptan (0.01 to 0.1 mg/kg, i.v.) exerts a dose-dependent diuretic effect in dogs without an increase in the urinary excretion of electrolytes, inhibits the pressor effect of exogenous vasopressin in a dose-dependent manner (0.003 to 0.1 mg/kg i.v.) and, at the highest dose (0.1 mg/kg i.v.), almost completely blocks vasoconstriction caused by exogenous vasopressin. Conivaptan (0.1 mg/kg, i.v.) improves cardiac function, as evidenced by significant increases in left ventricular dP/dtmax, cardiac output and stroke volume, and reduces preload and afterload, as evidenced by significant decreases in left ventricular end-diastolic pressure and total peripheral vascular resistance in dogs with congestive heart failure[3].