Tirofiban hydrochloride monohydrate

Cat. No.: HY-17369

CAS No.: 150915-40-5

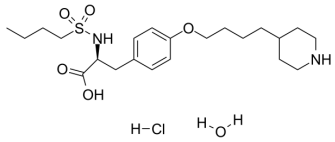
Molecular Formula: $C_{22}H_{39}ClN_2O_6S$

Molecular Weight: 495.07

Target: Integrin

Pathway: Cytoskeleton

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 : 100 mg/mL (201.99 mM; Need ultrasonic)
H_2O : 1 mg/mL (2.02 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.0199 mL	10.0996 mL	20.1992 mL
5 mM	0.4040 mL	2.0199 mL	4.0398 mL
10 mM	0.2020 mL	1.0100 mL	2.0199 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 (5.05 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.05 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.05 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Tirofiban (L700462) hydrochloride monohydrate is a selective and reversible platelet integrin receptor (Gp IIb/IIIa) antagonist that inhibits fibrinogen binding to this receptor and has antithrombotic activity. Tirofiban hydrochloride monohydrate induces proliferation and migration on endothelial cell by inducing production of VEGF. Tirofiban hydrochloride monohydrate can significantly reduces myocardial no-reflow and ischemia-reperfusion injury by alleviating myocardial microvascular structural and endothelial dysfunction in the ischemic area[1][2][3].

IC_{50} & Target

Gp IIb/IIIa Receptor[1]

In Vitro

Tirofiban hydrochloride monohydrate (0.25, 1, 3 μg/mL; 72 hours) increases proliferation of HAEC cells[1].
Tirofiban hydrochloride monohydrate (24 hours) closes the scratch of HUVECs migration within 18 hours[1].
Tirofiban hydrochloride monohydrate (0.25, 1 μg/mL; 1 hour) induces production of VEGF after 30 minutes which can stimulates proliferation of endothelial cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	HAEC cells
Concentration:	0.25, 1, 3 μg/mL
Incubation Time:	72 hours
Result:	Increased proliferation of HAEC cells.

Cell Migration Assay [1]

Cell Line:	HUVEC cells
Concentration:	
Incubation Time:	24 hours
Result:	Stimulated the migratory capacity of endothelial cells.

Western Blot Analysis[1]

Cell Line:	HAEC cells
Concentration:	0.05, 0.12, 0.25, 1 μg/mL
Incubation Time:	1 hour
Result:	Induced production of VEGF which stimulated proliferation of endothelial cells.

In Vivo

Tirofiban hydrochloride monohydrate (60 μg/kg; i.v.; once) shows activity of increasing contraction force, ventricular compliance, and improving heart function by increasing HR, LVESP, dp/dtmax, and reducing LVEDP[2].
Tirofiban hydrochloride monohydrate (60 μg/kg; i.v.; once) enhances eNOS activity, decreases iNOS activity and reduces area of no-reflow after reperfusion following AMI[2].Tirofiban hydrochloride monohydrate (50 μg/per; irrigate; once) shows anticoagulant effect with patency rates of 59% at 24 hours after microvascular anastomosis in the crush model[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Sprague-Dawley rats (10 to 15-week-age; 270-330 g)[2].
Dosage:	60 μg/kg
Administration:	Intravenous injection; once.
Result:	Increased contraction force, ventricular compliance, and improved heart function. Reduced the size of no-reflow and infarct.

Animal Model:	Sprague-Dawley rats (350-400 g; crush injury model)[3]
Dosage:	50 μg/per (50 μg/mL, 1 mL for each)
Administration:	Irrigate 1 mL within the vessel lumen (before placement of the last suture); once.

Result:	Showed anticoagulant effect with patency rates of 59%.

REFERENCES

[1]. Giordano A, et al. Tirofiban induces VEGF production and stimulates migration and proliferation of endothelial cells. Vascul Pharmacol. 2014 May-Jun;61(2-3):63-71.

[2]. Liu X, et al. Effects of tirofiban on the reperfusion-related no-reflow in rats with acute myocardial infarction. J Geriatr Cardiol. 2013 Mar;10(1):52-8.

[3]. Yates YJ, et al. The effect of tirofiban on microvascular thrombosis: crush model. Plast Reconstr Surg. 2005 Jul;116(1):205-8

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