Product Data Sheet

Product Name: Rho-Kinase-IN-2
Cat. No.: GC66050

Chemical Properties

Cas. No.	2573071-18-6		
Formula	$C_{20}H_{25}FN_4O_2$	M.Wt	372.44
Solubility	DMSO : 50 mg/mL (134.25 mM; Need ultrasonic)	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure	Rho-Kinase-IN-2		

Background

Rho-Kinase-IN-2 (Compound 23) is an orally active, selective, and central nervous system (CNS)-penetrant Rho Kinase (**ROCK**) inhibitor (ROCK2 $\mathbf{IC_{50}}$=3 nM). Rho-Kinase-IN-2 can be used in Huntington's research[1].

Rho-Kinase-IN-2 (0-10 mM, 1 hour) treatment shows an increase in AKT phosphorylation and a decrease in MYPT1 phosphorylation[1].

Western Blot Analysis[1]

Cell Line:	A7r5 and PANC1 cells
Concentration:	0-10 mM
Incubation Time:	1 hour

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Result:	Showed concentration-dependent effects, leading to an increase in AKT phosphorylation (EC_{50}=28 nM) and a decrease in MYPT1 phosphorylation (IC_{50}=14 nM).

Rho-Kinase-IN-2 (oral adiministration; 10 mg/kg; 6 times; 0.5, 1, 2, 4, 8, and 12 h) treatment shows dose- and time-dependent ROCK1 and ROCK2 target engagement[1].
Rho-Kinase-IN-2 (oral adiministration; 10 or 20 mg/kg; QD or BID; 2 weeks) treatment shows excellent tolerability assessment[1].
Rho-Kinase-IN-2 (oral adiministration; 1-20 mg/kg; once) treatment shows a direct dose- and time-dependent relationship between brain exposure and MYPT1 phosphorylation status[1].
Rho-Kinase-IN-2 (oral adiministration; 10 or 20 mg/kg; once) treatment decreases in the mean arterial, systolic, diastolic blood pressure, and heart rate[1].
Rho-Kinase-IN-2 (oral adiministration; 10 mg/kg; twice a day; 90 days) treatment leads to lower-than-expected brain concentrations[1].

Animal Model:	Male C57BL/6 mice[1]
Dosage:	10 mg/kg
Administration:	Oral adiministration; 10 mg/kg; 6 times; 0.5, 1, 2, 4, 8, and 12 h
Result:	Observed dose- and time-dependent ROCK1 and ROCK2 TE, with a free brain KiNativ ROCK1 and ROCK2 IC_{50}=~6 nM.
Animal Model:	3-4 months old heterozygote Q175DN KI and wild-type littermate mice[1]
Dosage:	10 or 20 mg/kg
Administration:	Oral adiministration; 10 or 20 mg/kg; once a day or twice a day; 2 weeks
Result:	Scored neurological index normally at all doses although a slight loss in bodyweight (~2%) in the 20 mg/kg treatment group.
Animal Model:	Heterozygote HTT zQ175DN knock-in mice[1]

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Dosage:	1-20 mg/kg
Administration:	Oral adiministration; 1-20 mg/kg; once
Result:	Remained over MYPT1 IC_{50} for over 2 h of the free brain at 10 mg/kg, and observed the dose- and time-dependent inhibition of MYPT1 phosphorylation in the striatum following acute in vivo dosing.
Animal Model:	CD1 mice[1]
Dosage:	10 and 20 mg/kg
Administration:	Oral adiministration; 10 or 20 mg/kg; once
Result:	Observed the decreases in the mean arterial (maximum change of 61.0 ± 8.5 mmHg from baseline), systolic (maximum change of 59.5 ± 8.4 mmHg from baseline), diastolic blood pressure (maximum change of 56.4 ± 9.0 mmHg from baseline), and heart rate (maximum change from predose of 107 bpm) when compared to the control group from ~0.5 to 2 h post dose.
Animal Model:	Heterozygote Q175DN KI mouse model of HD[1]
Dosage:	10 mg/kg
Administration:	Oral adiministration; 10 mg/kg; twice a day; 90 days
Result:	Led to lower-than-expected brain concentrations compared to single dosing.

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