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# Produktinformation

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## Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

## Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand


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# Dorsomorphin

**Cat. No.:** HY-13418A

**CAS No.:** 866405-64-3

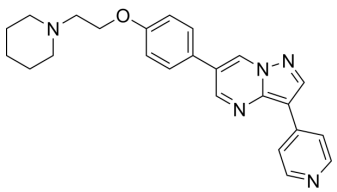
**Molecular Formula:** $C_{24}H_{25}N_5O$

**Molecular Weight:** 399.49

**Target:** AMPK; TGF-β Receptor; Autophagy; Organoid

**Pathway:** Epigenetics; PI3K/Akt/mTOR; TGF-beta/Smad; Autophagy; Stem Cell/Wnt

**Storage:** 4°C, protect from light
* In solvent : -80°C, 2 years; -20°C, 1 year (protect from light)



## SOLVENT & SOLUBILITY

**In Vitro**

1M HCl : 50 mg/mL (125.16 mM; ultrasonic and adjust pH to 1 with HCl)
DMSO : 33.33 mg/mL (83.43 mM; ultrasonic and adjust pH to 2 with 1M HCl)
$H_2O$ : < 0.1 mg/mL (insoluble)

| Preparing Stock Solutions | Mass / Solvent / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|---|
| | 1 mM | 2.5032 mL | 12.5160 mL | 25.0319 mL |
| | 5 mM | 0.5006 mL | 2.5032 mL | 5.0064 mL |
| | 10 mM | 0.2503 mL | 1.2516 mL | 2.5032 mL |

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

**In Vivo**

1. Add each solvent one by one: 0.5% CMC/saline water
   Solubility: 10 mg/mL (25.03 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 10 mg/mL (25.03 mM); Suspended solution; Need ultrasonic and warming and heat to 35°C

## BIOLOGICAL ACTIVITY

**Description**

Dorsomorphin (Compound C) is a selective and ATP-competitive AMPK inhibitor ($K_i$=109 nM in the absence of AMP). Dorsomorphin (BML-275) selectively inhibits BMP type I receptors ALK2, ALK3, and ALK6. Dorsomorphin can reverse autophagy activation and anti-inflammatory effect of Urolithin A (HY-100599)[1][2].

**$IC_{50}$ & Target**

| AMPK | ACVR1 | BMPR1A | ALK6 |
|---|---|---|---|
| 109 nM (Ki) | | | |

Autophagy

**In Vitro**

Dorsomorphin (compound C) (0-10 μM, 18 h) suppresses 2DG-induced GRP78 promoter activity in human fibrosarcoma HT1080 cells in a dose-dependent manner but has little effect on tunicamycin-induced GRP78 promoter activity. Dorsomorphin (compound C) C also suppresses GRP78 promoter activity induced by glucose withdrawal. Dorsomorphin (compound C) has no effect on 2DG-induced PERK activation and reduces the both basal and 2DG-induced AMPK phosphorylation levels in HT1080 cells[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[2]

| | |
|---|---|
| Cell Line: | Human fibrosarcoma HT1080 cells |
| Concentration: | 0-10 μM. |
| Incubation Time: | 18 hours. |
| Result: | Suppressed 2DG-induced GRP78 promoter activity in a dose-dependent manner and also suppressed GRP78 promoter activity induced by glucose withdrawal. |

**In Vivo**

Dorsomorphin (compound C: 10 mg/kg, intravenously once) treatment leads to a 60% increase in total serum iron concentrations, reduces basal levels of hepcidin expression and increasing serum iron concentrations in adult mice[3].
Dorsomorphin (compound C: 0.2 mg/kg, I.V., 30 min before LPS injection) reduces ICAM-1 and VCAM-1 expression in LPS-injected rat aorta[4].
Dorsomorphin (compound C; 25 mg/kg; i.p. injection; in male BALB/c mice) treatment before lipopolysaccharide (LPS) injection significantly reduces lethality in contrast to animals treated with LPS challenge only[5].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

| | |
|---|---|
| Animal Model: | Wild-type (WT) C57BL/6 adult mice that are fed a standard iron-replete diet express high levels of hepcidin[3]. |
| Dosage: | 10 mg/kg. |
| Administration: | Intravenously once. |
| Result: | Led to a 60% increase in total serum iron concentrations.<br>Effective in reducing basal levels of hepcidin expression and increasing serum iron concentrations in adult mice. |

| | |
|---|---|
| Animal Model: | Male Sprague-Dawley rats, 8 weeks of age (body weight 230-250 g)[4]. |
| Dosage: | 0.2 mg/kg. |
| Administration: | I.V., 30 min before LPS injection. |
| Result: | Reduced ICAM-1 and VCAM-1 expression in LPS-injected rat aorta. |

| | |
|---|---|
| Animal Model: | Male BALB/c mice at 6-7 weeks of age weighing 20-22 g[5] |
| Dosage: | 25 mg/kg |
| Administration: | Injection i.p.; 60 min before LPS challenge |
| Result: | Treatment of mice with 25 mg/kg before LPS injection significantly reduced lethality in contrast to animals treated with LPS challenge only. |

## CUSTOMER VALIDATION

- Signal Transduct Target Ther. 2022 Nov 30;7(1):384.
- Nat Nanotechnol. 2021 Jul;16(7):830-839.
- Adv Mater. 2024 Jan 17:e2304328.
- Cell Metab. 2021 Mar 2;33(3):565-580.e7.
- Cell Stem Cell. 2023 Apr 6;30(4):450-459.e9.

See more customer validations on www.MedChemExpress.com

## REFERENCES


[1]. Zhang Y, et al. Urolithin A suppresses glucolipotoxicity-induced ER stress and TXNIP/NLRP3/IL-1β inflammation signal in pancreatic β cells by regulating AMPK and autophagy. Phytomedicine. 2021 Dec;93:153741.

[2]. Zhou G, et al. Role of AMP-activated protein kinase in mechanism of action. J Clin Invest. 2001 Oct;108(8):1167-74.

[3]. Saito S, et al. Compound C prevents the unfolded protein response during glucose deprivation through a mechanism independent of AMPK and BMP signaling. PLoS One. 2012;7(9):e45845.

[4]. Yu PB, et al. Dorsomorphin inhibits BMP signals required for embryogenesis and iron metabolism. Nat Chem Biol. 2008 Jan;4(1):33-41.

[5]. Kim YM, et al. Compound C independent of AMPK inhibits ICAM-1 and VCAM-1 expression in inflammatory stimulants-activated endothelial cells in vitro and in vivo. Atherosclerosis. 2011 Nov;219(1):57-64.

[6]. Guo Y, et al. AMPK inhibition blocks ROS-NFκB signaling and attenuates endotoxemia-induced liver injury. PLoS One. 2014 Jan 24;9(1):e86881.





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