# C29

**Cat. No.:** HY-100461

**CAS No.:** 363600-92-4

**Molecular Formula:** $C_{16}H_{15}NO_4$

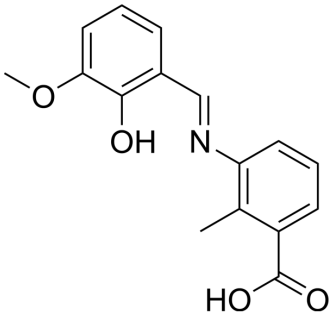
**Molecular Weight:** 285

**Target:** Toll-like Receptor (TLR)

**Pathway:** Immunology/Inflammation

**Storage:**

| | | |
|---|---|---|
| Powder | -20°C | 3 years |
| | 4°C | 2 years |
| In solvent | -80°C | 6 months |
| | -20°C | 1 month |




## SOLVENT & SOLUBILITY

**In Vitro**

DMSO : ≥ 30 mg/mL (105.26 mM)

* "≥" means soluble, but saturation unknown.

| Preparing Stock Solutions | Mass / Solvent / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|---|
| | 1 mM | 3.5088 mL | 17.5439 mL | 35.0877 mL |
| | 5 mM | 0.7018 mL | 3.5088 mL | 7.0175 mL |
| | 10 mM | 0.3509 mL | 1.7544 mL | 3.5088 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 (8.77 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.77 mM); Clear solution

## BIOLOGICAL ACTIVITY

**Description**

C29 is a Toll-like receptor 2 (TLR2) inhibitor. C29 blocks hTLR2/1 and hTLR2/6 signaling with $IC_{50}$s of 19.7 and 37.6 μM, respectively[1].

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

TLR2

**In Vitro**

C29 (10 or 50 μM; 1 hour) blocks P3C- and P2C-induced IL-8 mRNA dose-dependently in HEK-TLR2 stable transfectants. C29 (50-200 μM; 1 hour) inhibits P3C- and P2C-induced IL-1β gene expression significantly at both 1 h and 4 h following stimulation in THP-1 cells[1].

?C29 (25 or 50 μM; 1 hour) reduces P3C-induced but not P2C-induced TNF-α mRNA and IL-12 p40 protein significantly in

primary murine macrophages[1].
?C29 (50 μM; 1 hour) blocks TLR2 bacterial agonist-induced proinflammatory gene expression in HEK-TLR2 cells and murine macrophages[1].

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

Western Blot Analysis[1]

| | |
|---|---|
| Cell Line: | THP-1 cells |
| Concentration: | 150 μM |
| Incubation Time: | 1 hours |
| Result: | Diminished the interaction between endogenous TLR2 and myeloid differentiation primary response gene 88 (MyD88) at 15 and 30 min poststimulation with P3C. |

Western Blot Analysis[1]

| | |
|---|---|
| Cell Line: | Murine peritoneal macrophages |
| Concentration: | 50 μM |
| Incubation Time: | 1 hours |
| Result: | Blocked robust MAPK activation at 30 min and reduced NF-κB activation from 5 to 30 min. Prevented P3C-induced degradation of IκBα at 15 and 30 min. |

## CUSTOMER VALIDATION

- Nat Immunol. 2021 Jul;22(7):829-838.
- J Extracell Vesicles. 2023 Jun;12(6):e12335.
- Adv Sci (Weinh). 2023 Mar 22;e2300116.
- Biomaterials. 2020 May;241:119852.
- J Hazard Mater. 2023 Mar 23;452:131262

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## REFERENCES

[1]. Cai S, Zhu G, Cen X, et al. Synthesis, structure-activity relationships and preliminary mechanism study of N-benzylideneaniline derivatives as potential TLR2 inhibitors. Bioorg Med Chem. 2018;26(8):2041-2050.

[2]. Mistry P, et al. Inhibition of TLR2 signaling by small molecule inhibitors targeting a pocket within the TLR2 TIR domain. Proc Natl Acad Sci U S A. 2015 Apr 28;112(17):5455-60.

**Caution: Product has not been fully validated for medical applications. For research use only.**

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA