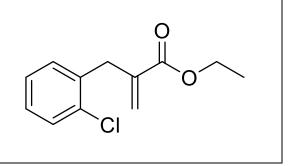
Product data sheet



| MedKoo Cat#: 530591 | | | | |
|--|--|--|--|--|
| | | | | |
| Name: INF39 | | | | |
| CAS: 866028-26-4 | | | | |
| Chemical Formula: C ₁₂ H ₁₃ ClO ₂ | | | | |
| Exact Mass: 224.0604 | | | | |
| Molecular Weight: 224.684 | | | | |
| Product supplied as: | Powder | | | |
| Purity (by HPLC): | $\geq 98\%$ | | | |
| Shipping conditions | Ambient temperature | | | |
| Storage conditions: | Powder: -20°C 3 years; 4°C 2 years. | | | |
| | In solvent: -80°C 3 months; -20°C 2 weeks. | | | |



1. Product description:

INF39 is a nontoxic, irreversible NLRP3 inhibitor able to decrease interleukin-1 β release from macrophages. INF39 Targets the NLRP3 Inflammasome, and may be useful for the Treatment of Inflammatory Bowel Disease. Bioluminescence resonance energy transfer experiments proved that INF39 was able to directly interfere with NLRP3 activation in cells.

2. CoA, QC data, SDS, and handling instruction

SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

| Solvent | Max Conc. mg/mL | Max Conc. mM |
|---------|-----------------|--------------|
| DMF | 30.0 | 133.52 |
| DMSO | 66.0 | 293.75 |
| Ethanol | 30.0 | 133.52 |

4. Stock solution preparation table:

| Concentration / Solvent Volume / Mass | 1 mg | 5 mg | 10 mg |
|---------------------------------------|---------|----------|----------|
| 1 mM | 4.46 mL | 22.25 mL | 44.51 mL |
| 5 mM | 0.89 mL | 4.45 mL | 8.90 mL |
| 10 mM | 0.45 mL | 2.23 mL | 4.45 mL |
| 50 mM | 0.09 mL | 0.45 mL | 0.89 mL |

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator

Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study

In vitro study

1. Pu Z, Han C, Zhang W, Xu M, Wu Z, Liu Y, Wu M, Sun H, Xie H. Systematic understanding of the mechanism and effects of Arctigenin attenuates inflammation in dextran sulfate sodium-induced acute colitis through suppression of NLRP3 inflammasome by SIRT1. Am J Transl Res. 2019 Jul 15;11(7):3992-4009. PMID: 31396314; PMCID: PMC6684881.

In vivo study

1. Zhang T, Huang W, Ma Y. Down-regulation of TRPM2 attenuates hepatic ischemia/reperfusion injury through activation of autophagy and inhibition of NLRP3 inflammasome pathway. Int Immunopharmacol. 2022 Mar;104:108443. doi: 10.1016/j.intimp.2021.108443. Epub 2022 Jan 10. PMID: 35021129.

2. Wang Y, Song B, Chen J, Cao J, Li X, Sun C. Polymethoxyflavones in Citrus Regulate Lipopolysaccharide-Induced Oscillating Decay of Circadian Rhythm Genes by Inhibiting Nlrp3 Expression. Oxid Med Cell Longev. 2021 Sep 14;2021:8419415. doi: 10.1155/2021/8419415. PMID: 34567414; PMCID: PMC8457985.

7. Bioactivity

Biological target:

Product data sheet

INF39 is an irreversible and noncytotoxic NLRP3 inhibitor.

In vitro activity

To further explore the role of NLRP3 in the effects of arctigenin on inflammation in DSS-induced acute colitis, NLRP3 inhibitor, 12.5 mg/kg of INF39 was used to DSS-induced acute colitis by arctigenin. As showed in Figure 8A-E, NLRP3 inhibitor suppressed the protein expression of IL-1 β , IL-18, caspase-1 and NLRP3 in DSS-induced acute colitis by arctigenin, compared with treatment with arctigenin group. Next, NLRP3 inhibitor also increased the effects of arctigenin on the weight, colon length, histochemical score and MPO activity levels in DSS-induced acute colitis, compared with treatment with arctigenin group (Figure 8F-K).

Reference: Am J Transl Res. 2019 Jul 15;11(7):3992-4009. https://pubmed.ncbi.nlm.nih.gov/31396314/

In vivo activity

Additionally, this study detected the expression of NLRP3 inflammasome pathway in the OGD/R-induced hepatocytes which had been treated with the autophagy agonist and inhibitor, and found that autophagy negatively regulated the NLRP3 inflammasome pathway. Moreover, this study discovered that the administration of NLRP3-inhibitor INF39 increased cell viability and caused a decline in cell death in the OGD/R-treated hepatocytes.

Reference: Int Immunopharmacol. 2022 Mar;104:108443. https://pubmed.ncbi.nlm.nih.gov/35021129/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.

