N-Formyl-Met-Leu-Phe

Cat. No.: HY-P0224

CAS No.: 59880-97-6

Molecular Formula: $C_{21}H_{31}N_3O_5S$

Molecular Weight: 437.55

Sequence: Formyl-Met-Leu-Phe

Sequence Shortening: Formyl-MLF

Target: TNF Receptor

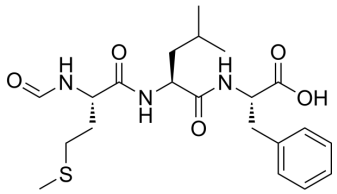
Pathway: Apoptosis

Storage: Sealed storage, away from moisture

Powder -80°C 2 years

-20°C 1 year

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 82.5 mg/mL (188.55 mM)

H_2O : < 0.1 mg/mL (ultrasonic) (insoluble)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.2855 mL	11.4273 mL	22.8545 mL
5 mM	0.4571 mL	2.2855 mL	4.5709 mL
10 mM	0.2285 mL	1.1427 mL	2.2855 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: ≥ 2.08 mg/mL (4.75 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.75 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

N-Formyl-Met-Leu-Phe (fMLP; N-Formyl-MLF) is a chemotactic peptide and a specific ligand of N-formyl peptide receptor (FPR). N-Formyl-Met-Leu-Ph is reported to inhibit TNF-alpha secretion.

IC_{50} & Target

TNF-alpha[1]

In Vitro	Binding of N-Formyl-Met-Leu-Phe to its specific cell surface receptor, N-formyl peptide receptor (FPR), triggers different cascades of biochemical events, eventually leading to cellular activation. FPR is a chemoattractant receptor belonging to the G protein-coupled receptor family. N-Formyl-Met-Leu-Phe promotes osteoblastic commitment and suppresses adipogenic commitment under osteoblastic differentiation conditions. N-Formyl-Met-Leu-Phe stimulates osteogenesis is associated with increased expression of osteogenic markers and mineralization. N-Formyl-Met-Leu-Phe inhibits expression of peroxisome proliferator-activated receptor-γ1. N-Formyl-Met-Leu-Phe-stimulated osteogenic differentiation is mediated via FPR1-phospholipase C/phospholipase D-Ca^{2+}-calmodulin-dependent kinase II-ERK-CREB signaling pathways[1]. N-Formyl-Met-Leu-Phe, a bacterial-derived peptide, induced proinflammatory cytokine gene expression in human peripheral blood monocytes. Bacterial products LPS and N-Formyl-Met-Leu-Phe synergistically induce inflammatory response via multiple signaling pathways. TLR4, IKKβ-IκBα, and NF-κB signaling pathways are involved in the synergistic induction of TNF-α via p65 nuclear translocation-dependent mechanisms[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	N-Formyl-Met-Leu-Phe promotes bone formation in zebrafish and rabbits. Extensive skeletal development is evident at 5 dpf in over 80% of N-Formyl-Met-Leu-Phe-treated zebrafish. Treatment with N-Formyl-Met-Leu-Phe results in increased expression of Runx2. Bone marrow spaces are widely formed, and connective tissue covering bone is dense, like periosteum, in N-Formyl-Met-Leu-Phe-treated calvaria[1]. N-Formyl-Met-Leu-Phe mediate release of calprotectin from PMN in vitro. It induces release of calprotectin from PMN in a dose dependent manner. A minimum of 10% of total PMN calprotectin is retained at concentrations of 0.1-10.0 nM of N-Formyl-Met-Leu-Phe[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	Cells are cotransfected with either a dominant negative form of IκBα or a dominant negative form of IKKβ together with the NF-κB-dependent luciferase reporter plasmid. The plasmid pCMVβ is used as a control for transfection efficiency and this is monitored via the expression of β-galactosidase. Cells are transiently transfected with plasmids using DEAE-dextran. The transfected cells are cultivated for 48 h before a 6-h incubation in medium ±N-Formyl-Met-Leu-Phe, LPS, or N-Formyl-Met-Leu-Phe/LPS. Luciferase activity is determined by using the luciferase assay kit and a Monolight 3010 luminometer[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice: N-Formyl-Met-Leu-Phe is prepared in sterile PBS. Under the anesthesia, mice are intranasally treated with LPS (0.3 mg/kg) or N-Formyl-Met-Leu-Phe (0.5 mg/kg) or N-Formyl-Met-Leu-Phe and LPS in 50 μL of sterile PBS (control), BAL is performed by cannulating the trachea with sterilized PBS, and cells from BAL fluid are stained with Wright-Giemsa stain after cytocentrifuge. For TNF-α protein release, BAL fluid is collected and secreted TNF-α is measured by ELISA as described above[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Mol Immunol. 2022 Jan 25;1-17.
- Biomaterials. 2021, 120784.
- Nano Res. 2021 Mar 27.
- Pharmacol Res. 2023 May 6;106791.
- Research Square Print. 2022.

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REFERENCES

[1]. Shin MK, et al. N-formyl-methionyl-leucyl-phenylalanine (fMLP) promotes osteoblast differentiation via the N-formyl peptide receptor 1-mediated signaling pathway in human mesenchymal stem cells from bone marrow. J Biol Chem. 2011 May 13;286(19):17133-43.

[2]. Chen LY, et al. Synergistic induction of inflammation by bacterial products lipopolysaccharide and fMLP: an important microbial pathogenic mechanism. J Immunol. 2009 Feb 15;182(4):2518-24.

[3]. Hetland G, et al. Chemotaxins C5a and fMLP induce release of calprotectin (leucocyte L1 protein) from polymorphonuclear cells in vitro. Mol Pathol. 1998 Jun;51(3):143-8.

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