
Product Data Sheet

Preparation Method	HaCaT cells were maintained in high-glucose Dulbecco's Modified Eagle Medium (DMEM) supplemented with 10% fetal bovine serum (FBS), 2mM L-glutamine, and antibiotics (50U/mL penicillin, 50µg/mL streptomycin) at 37°C, 5% CO ₂ . Cells were pretreated with PBP 10 (2-10µg/mL) for 20 min, followed by stimulation with bacterial lipopolysaccharide (LPS; 1µg/mL) or lipoteichoic acid (LTA; 1µg/mL) for 24h in serum-free conditions.
Reaction Conditions	2-10µg/mL; pretreatment for 20min
Applications	PBP 10 significantly suppressed LPS/LTA-induced production of inflammatory mediators, including nitric oxide (NO), reactive oxygen species (ROS), and interleukin-8 (IL-8), in a dose-dependent manner. PBP 10 also reversed LPS-induced softening of cell stiffness, as measured by atomic force microscopy (AFM), indicating restoration of cytoskeletal integrity.
References:	
[1] Korimová A, Dubový P. N-Formylated Peptide Induces Increased Expression of Both Formyl Peptide Receptor 2 (Fpr2) and Toll-Like Receptor 9 (TLR9) in Schwannoma Cells-An In Vitro Model for Early Inflammatory Profiling of Schwann Cells. Cells. 2020 Dec 11;9(12):2661.	

Background

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

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PBP 10 is a cell-permeable formyl peptide receptor 2 (FPR2) antagonist^[1-2]. PBP 10 exhibits multiple biological activities, including antibacterial, antiviral, and cancer-inhibitory effects^[3-4].

In vitro, pretreatment of human keratinocytes (HaCaT) with PBP 10 (2-10µg/mL) for 20 minutes significantly suppresses the release of pro-inflammatory mediators such as nitric oxide (NO), reactive oxygen species (ROS), and interleukin-8 (IL-8) induced by stimulation with lipopolysaccharide (LPS; 1µg/mL) or lipoteichoic acid (LTA; 1µg/mL), while also reversing inflammation-related nanomechanical changes, such as the reduction in cell stiffness caused by LPS^[5]. Pretreatment of RT4 Schwann cells with PBP 10 (1 µM) for 20 minutes, followed by stimulation with fMLF (10nM-100nM) for 6 hours, significantly inhibits fMLF-induced upregulation of Fpr2 protein expression and the decrease in p-NFκB levels, while also reversing changes in the expression of CCR2, CXCR4, and PKCβ^[6].

References:

- [1] Forsman H, Andréasson E, Karlsson J, et al. Structural characterization and inhibitory profile of formyl peptide receptor 2 selective peptides descending from a PIP2-binding domain of gelsolin. *J Immunol*. 2012 Jul 15;189(2):629-37.
- [2] Holdfeldt A, Winther M, Gabl M, et al. Data on human neutrophil activation induced by pepducins with amino acid sequences derived from β2AR and CXCR4. *Data Brief*. 2016 Jun 1;8:411-4.
- [3] Courtin N, Fotso AF, Fautrad P, et al. Antiviral activity of formyl peptide receptor 2 antagonists against influenza viruses. *Antiviral Res*. 2017 Jul;143:252-261.
- [4] Jia G, Wang X, Wu W, et al. LXA4 enhances prostate cancer progression by facilitating M2 macrophage polarization via inhibition of METTL3. *Int Immunopharmacol*. 2022 Jun;107:108586.
- [5] Korimová A, Dubový P. N-Formylated Peptide Induces Increased Expression of Both Formyl Peptide Receptor 2 (Fpr2) and Toll-Like Receptor 9 (TLR9) in Schwannoma Cells-An In Vitro Model for Early Inflammatory Profiling of Schwann Cells. *Cells*. 2020 Dec 11;9(12):2661.
- [6] Xu J, Su Z, Cheng X, et al. High PPT1 expression predicts poor clinical outcome and PPT1 inhibitor DC661 enhances sorafenib sensitivity in hepatocellular carcinoma. *Cancer Cell Int*. 2022 Mar 11;22(1):115.

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