

Product Data Sheet

Product Name: BP79
Cat. No.: GC79427

Chemical Properties

Cas. No. 3104965-33-2

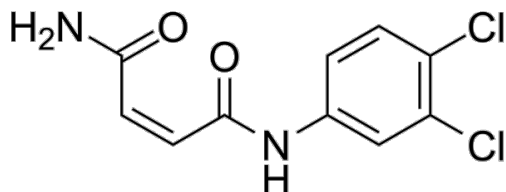
Formula $C_{10}H_8Cl_2N_2O_2$ M.Wt 259.09

Solubility DMSO: 100 mg/mL (385.97 mM; Need ultrasonic) Storage Store at 4°C, away from moisture and light

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Background

BP79 is a potent TSLP receptor inhibitor. BP79 disrupts TSLP-mediated ternary complex formation, blocks TSLPR - IL7R α co-localization, binds and stabilizes TSLPR, and inhibits phosphorylated STAT3/6. BP79 suppresses immune cell infiltration, secretion of IL-13, IL-4. BP79 can be used for the research of inflammation diseases, such as atopic dermatitis, allergic asthma, and allergic rhinitis.

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

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[1] [2] [3].

In Vitro, BP79 (5-20 μ M; 36 h) inhibits TSLP-induced IL-13 (80% inhibition at 20 μ M) and IL-4 (60% inhibition at 20 μ M) secretion in HuT78 cells in a concentration-dependent manner^[1]. BP79 (0.15-20 μ M; 36 h) potently inhibits TSLP-induced IL-4 (IC_{50} = 2.88 μ M) and IL-13 (IC_{50} = 0.97 μ M) secretion in primary human CD4⁺ T cells, with $\geq$ 80% inhibition at 10 μ M and 20 μ M^[1]. BP79 (0.15-20 μ M; 24 h) shows no significant cytotoxicity at concentrations up to 20 μ M in primary human CD4⁺ T cells, keratinocytes, and fibroblasts, with viability remaining $\geq$ 80%^[1]. BP79 (20 μ M; 1-5 days) abrogates TSLP-mediated hyperproliferation of primary human CD4⁺ T cells^[1]. BP79 (20 μ M) inhibits TSLP-induced STAT6 phosphorylation in primary human CD4⁺ T cells and STAT3 phosphorylation in primary human keratinocytes^[1]. BP79 (20 μ M; 24 h) inhibits TSLP-induced OX-40L expression and CCL17 secretion in primary human myeloid dendritic cells, and reduces dendritic cell-mediated IL-13 secretion from naive CD4⁺ T cells^[1]. BP79 (20 μ M; 10 min) directly interacts with recombinant TSLPR, thermally stabilizing the protein up to 45 °C^[1]. BP79 (20 μ M; topical; once daily for 4 days) suppresses pro-inflammatory cytokine secretion, restores skin barrier protein expression, inhibits TSLP production, and blocks CD4⁺ T-cell infiltration in a human atopic diseases multi-organ chip model, with beneficial effects on both skin and lung tissues^[1]. BP79 (10 μ M) exhibits moderate binding to some kinases, but does not inhibit JAK2/JAK3/TYK2 kinases in the TSLP signaling pathway^[2]. BP79 prevents the co-localization of TSLPR and IL-7R α in primary keratinocytes following TSLP treatment, supporting its targeting of the TSLP signaling pathway^[2].

References:

- [1]. Adhikary PP, et al. Disrupting TSLP-TSLP receptor interactions via putative small molecule inhibitors yields a novel and efficient treatment option for atopic diseases. EMBO Mol Med. 2024;16(7):1630-1656.
- [2]. Christopher Hoang. The development of small molecule inhibitors of thymic stromal lymphopoietin signaling for the treatment of atopic dermatitis. \t University of British Columbia. December 2023.
- [3]. Sarah Hedtrich, et al. N1-(3,4-dichlorophenyl)maleamide derivatives and the use thereof in the treatment of inflammatory disorders. WO2025213277A1. 2025-10-16.

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