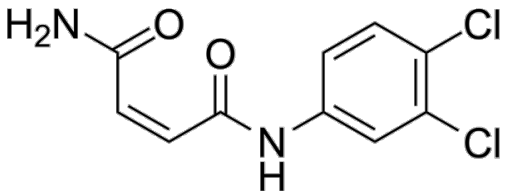
Product Data Sheet

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 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ 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 - IL7Rα 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 μM; 36 h) inhibits TSLP-induced IL-13 (80% inhibition at 20 μM) and IL-4 (60% inhibition at 20 μM) secretion in HuT78 cells in a concentration-dependent manner [1]. BP79 (0.15-20 μM; 36 h) potently inhibits TSLP-induced IL-4 (IC_{50} = 2.88 μM) and IL-13 (IC_{50} = 0.97 μM) secretion in primary human CD4+ T cells, with ≥80% inhibition at 10 μM and 20 μM[1]. BP79 (0.15-20 μM; 24 h) shows no significant cytotoxicity at concentrations up to 20 μM in primary human CD4+ T cells, keratinocytes, and fibroblasts, with viability remaining ≥80%[1]. BP79 (20 μM; 1-5 days) abrogates TSLP-mediated hyperproliferation of primary human CD4+ T cells[1]. BP79 (20 μM) inhibits TSLP-induced STAT6 phosphorylation in primary human CD4+ T cells and STAT3 phosphorylation in primary human keratinocytes[1]. BP79 (20 μM; 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 μM; 10 min) directly interacts with recombinant TSLPR, thermally stabilizing the protein up to 45 °C[1]. BP79 (20 μM; 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 μM) 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-7Rα 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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