

Product Data Sheet

Product Name: PL120131 acetate

Cat. No.: GC79338

Chemical Properties

Cas. No.

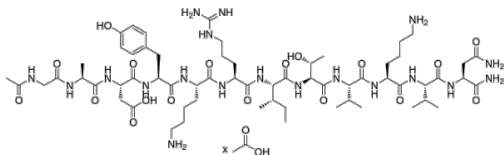
Formula $C_{62}H_{105}N_{19}O_{18} \cdot xC_2H_4O_2$ M.Wt 1404.61

Solubility H_2O : 5 mg/mL (Need ultrasonic) Storage Sealed storage, away from moisture

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

PL120131 acetate is a PD-1 antagonist, can specifically blocking the interaction between PD-1 and

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

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PD-L1, thereby effectively inhibiting the PD-1-mediated apoptosis signaling pathway. PL120131 acetate rescues lymphocytes from apoptosis, maintains the survival and activity of T cells, and induces cytotoxic T lymphocytes to exert killing effects and recognize macrophages and dendritic cells. PL120131 acetate can be used in research related to breast cancer and various malignant tumors [1] [2] [3] [4].

In Vitro, PL120131 acetate (32.0-33.4 μ M) rescues Jurkat cells from PD-L1-induced apoptosis, reducing apoptotic signals by 25% with an IC_{50} of 32.0 μ M (Annexin V) and 33.4 μ M (Caspase 3/7)^[2]. PL120131 acetate (11.6 μ M; 24 h) rescues primary mouse T-cells from apoptosis in 4T1 co-cultures, increases 4T1 cell death, and induces a significant, dose-dependent increase in Granzyme B production in CD3+/CD8+ T-cells^[2]. PL120131 acetate (0.2-12.5 μ g/mL; 24 h) induces dose-dependent increases in IFN γ and dose-dependent decreases in IL-12p70 and IL-10 in co-cultures of primary mouse lymphocytes and 4T1 cells, indicating activated T-cell function^[2]. PL120131 acetate (30 μ M; 30 min (Jurkat pre-treatment); 24 h (co-culture in microfluidics chip)) enables Jurkat cells to infiltrate a 3D MDA-MB-231 tumor environment, induce greater tumor cell death, and maintain higher Jurkat cell viability than anti-PD-1 antibody treatment^[2].

References:

- [1]. Tseng T S, et al. Structure-guided discovery of PD-1/PD-L1 interaction inhibitors: peptide design, screening, and optimization via computation-aided phage display engineering[J]. Journal of Chemical Information and Modeling, 2024, 64(5): 1615-1627.
- [2]. Boohaker R J, et al. Rational design and development of a peptide inhibitor for the PD-1/PD-L1 interaction[J]. Cancer letters, 2018, 434: 11-21.
- [3]. Gu W, et al. Undo the brake of tumour immune tolerance with antibodies, peptide mimetics and small molecule compounds targeting PD-1/PD-L1 checkpoint at different locations for acceleration of cytotoxic immunity to cancer cells[J]. Clinical and Experimental Pharmacology and Physiology, 2019, 46(2): 105-115.
- [4]. Sun M, et al. Fibroblast Activation Protein- α Responsive Peptide Assembling Prodrug Nanoparticles for Remodeling the Immunosuppressive Microenvironment and Boosting Cancer Immunotherapy. Small. 2022;18(9):e2106296.

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