

Product Data Sheet

Product Name: NCP26 TFA

Cat. No.: GC81879

Chemical Properties

Cas. No.

Formula $C_{22}H_{24}F_3N_5O_4$

M.Wt 479.45

Solubility

Storage Store at 4°C, 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

NCP26 TFA is an ATP-competitive ProRS inhibitor (with a K_D of 271 nM in the absence of proline, and a K_D of 0.35 nM in the presence of 100 μM proline). NCP26 TFA activates AAR, induces G0/G1

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

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phase arrest, Apoptosis, Caspase cleavage, and PARP cleavage. NCP26 TFA downregulates MYC, TCF3, and CCND1. NCP26 TFA inhibits the growth of multiple myeloma cells. NCP26 TFA can be used for the research of multiple myeloma [1] [2].

In Vivo, NCP26 (2.5-10 mg/kg; i.p.; once daily; 21 days) TFA significantly inhibits multiple myeloma tumour growth ($P = 0.01$ and $P < 0.001$, respectively) and prolongs overall survival ($P = 0.01$ and $P < 0.001$, respectively) in CB17 SCID mice bearing AMO1 xenografts, without causing significant body weight loss^[1]. NCP26 (10 mg/kg; q.d. [daily]) TFA significantly inhibits multiple myeloma tumor growth ($P = 0.005$) and prolongs host survival ($P < 0.001$) in an AMO1 xenograft mouse model without inducing weight loss^[2].

In Vitro, NCP26 TFA is an ATP-competitive inhibitor of recombinant ProRS. Its binding affinity increases by more than 750-fold in the presence of 100 μM proline, with a K_D of 0.35 nM^[1]. NCP26 (0.5 μM ; 48 h) TFA maintains antiproliferative activity against AMO1 and RPMI 8226 multiple myeloma (MM) cells over a proline concentration range of 0 to 20 mM, with no reduction in its potency at high proline levels^[1]. NCP26 (0.01-10 μM ; 96 h) TFA is 10-fold more potent against AMO1 multiple myeloma (MM) cells than against peripheral blood mononuclear cells (PBMCs) from healthy donors, with an EC_{50} of 0.1 μM for the former and approximately 1 μM for the latter after 96 h of treatment^[1]. NCP26 TFA potently inhibits the growth of most multiple myeloma (MM) cell lines, with EC_{50} values ranging from 135 nM to 1.1 μM ^[1]. NCP26 (0.5 μM ; 6-48 h) TFA induces G0/G1 cell cycle arrest and caspase-dependent apoptosis in AMO1 and RPMI 8226 multiple myeloma (MM) cells^[1].

References:

- [1]. Kurata K, et al. Prolyl-tRNA synthetase as a novel therapeutic target in multiple myeloma. Blood Cancer J. 2023;13(1):12. Published 2023 Jan 12.
- [2]. Kurata K, et al. Discovery of Prolyl-tRNA Synthetase As a Novel Target in Multiple Myeloma[J]. Blood, 2021, 138: 890.

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