

Product Data Sheet

Product Name: MRC37
Cat. No.: GC79630

Chemical Properties

Cas. No.

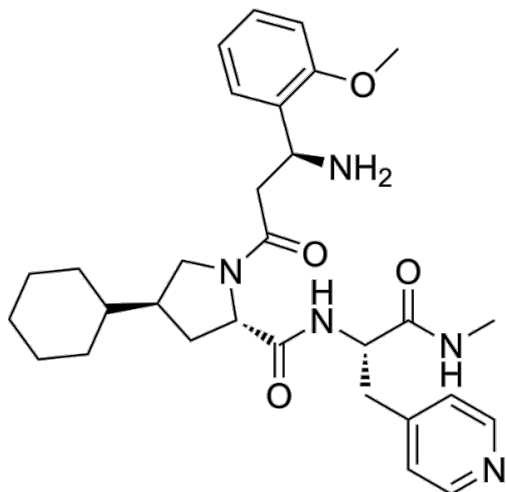
Formula $C_{30}H_{41}N_5O_4$ M.Wt 535.68

Solubility DMSO: ≥ 250 mg/mL (466.70 mM) 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

MRC37 is a high-affinity TRIM21 ligand with a K_d of 0.4 μ M. MRC37 targets the IgG Fc-binding pocket in the PRYSPRY domain of TRIM21, mimics the "HHNY" epitope, and inhibits substrate-induced aggregation and activation of TRIM21 via competitive blockade of antibody binding. MRC37 effectively blocks TRIM21-mediated antiviral immunity (such as adenovirus neutralization) and Trim-Away targeted protein degradation without cytotoxicity. MRC37 antagonizes TRIMTAC activity, and its multiple modification sites do not affect target binding, making it suitable as a degrader warhead for research on viral immunity and neurodegenerative diseases such as tauopathies [1].

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

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In Vitro, MRC37 (0.4 μ M; 48 h) binds to purified TRIM21 PRYSPRY domain with high affinity (0.4 μ M) and exhibits no TRIM21-dependent cytotoxicity in wild-type RPE-1 cells at concentrations up to 20 μ M for 48 h^[1]. MRC37 (1 μ M) monovalent ligand does not drive ternary complex formation between TRIM21 and H2B-mEGFP-Halo in RPE-1 cells, nor does it affect TRIM21 PRYSPRY binding when modified to generate TRIMTAC derivatives^[1]. MRC37 (0.01-100 μ M; 48 h) does not induce degradation of monomeric mEGFP-Halo, oligomeric CAV1-mEGFP-Halo, or oligomeric Cavin1-mEGFP-Halo in RPE-1 cells at concentrations up to 100 μ M for 48 h^[1]. MRC37 (50-100 μ M; 15 h) inhibits TRIM21-mediated antiviral activity in HEK293T cells and TRIM21-dependent protein degradation in RPE-1 cells in a dose-dependent manner^[1]. MRC37 (5 μ M) does not degrade oligomeric Myd88 Myddosomes in stimulated cells, but can compete with TRIMTAC MRC414 to partially reverse inhibition of seeded tau aggregation in HEK293 reporter cells^[1].

References:

[1]. Luptak J, et al. State-selective small molecule degraders that preferentially remove aggregates and oligomers. Nature communications. 2025 Nov 25;16(1):10486.

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