

D8-MMAE

Catalog No: tcsc3368



Available Sizes

Size: 1mg

Size: 5mg

Size: 10mg



Specifications

CAS No:

2070009-72-0

Formula:

$C_{39}H_{59}D_8N_5O_7$

Pathway:

Cell Cycle/DNA Damage;Cytoskeleton;Antibody-drug Conjugate/ADC Related

Target:

Microtubule/Tubulin;Microtubule/Tubulin;ADC Cytotoxin

Purity / Grade:

>98%

Solubility:

DMSO : \geq 40 mg/mL (55.09 mM)

Alternative Names:

D8-Monomethyl auristatin E;D8-Vedotin

Observed Molecular Weight:

726.03

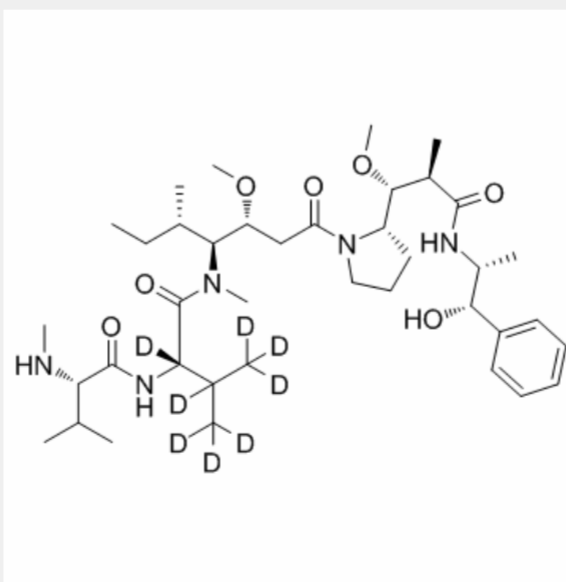
Product Description

D8-MMAE is a deuterated form of MMAE, which is a **microtubule**-disrupting agent.

IC50 & Target: Microtubule^[1]

In Vitro: Antibody-drug conjugates (ADC) comprise targeting antibodies armed with potent small-molecule payloads. ADCs are generated to target different receptors on the anaplastic large cell lymphoma line L-82, but delivered the same cytotoxic payload (monomethyl auristatin E, MMAE), and the intracellular concentration of released MMAE correlated with in vitro ADC-mediated cytotoxicity independent of target expression or drug:antibody ratios. LC-MS is used to measure the concentration of MMAE in a parallel cohort of L-82 tumors with an identical treatment regimen. Although tumor volume is not different among treatment groups 3 days after dose, the intratumoral MMAE measurement reveals two patterns. First, intratumoral MMAE concentration increases proportionally to the ADC dose, which corresponds to stronger antitumor activity. Second, the intratumoral MMAE concentration obtained from treatment with both cOKT9-vcMMAE and cAC10-vcMMAE is similar at each dose, consistent with the observation that tumor responded similarly to these two ADCs^[1].

In Vivo: Intratumoral MMAE concentrations consistently correlates with the extent of tumor growth inhibition in tumor xenograft models. IHC analysis reveals that nonbinding control-treated tumors consist of both CD30⁺ and CD30⁻ cells, presumably because they do not kill either CD30⁺ or CD30⁻ Karpas 299 cells. Only CD30⁻ cells are found in cAC10-vcMMAF-treated tumors, illustrating that cAC10-vcMMAF eliminates most CD30⁺ cells. Interestingly, the two tumors that relapses from cAC10-vcMMAE treatment are also found to be CD30⁻ by the end of study, indicating a small fraction of CD30⁻ cells might have escaped from bystander killing in these two remaining tumors^[1].



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