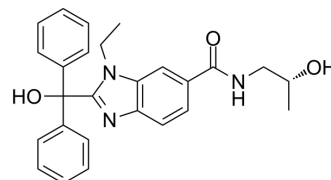


## VY-3-135

Cat. No.:	HY-145953
CAS No.:	1824637-41-3
Molecular Formula:	C <sub>26</sub> H <sub>27</sub> N <sub>3</sub> O <sub>3</sub>
Molecular Weight:	429.51
Target:	Others
Pathway:	Others
Storage:	4°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



### SOLVENT & SOLUBILITY

In Vitro	DMSO : 50 mg/mL (116.41 mM; Need ultrasonic)						
	Preparing Stock Solutions	Solvent Concentration	Mass	1 mg	5 mg	10 mg	
				1 mM	2.3282 mL	11.6412 mL	23.2823 mL
				5 mM	0.4656 mL	2.3282 mL	4.6565 mL
				10 mM	0.2328 mL	1.1641 mL	2.3282 mL
Please refer to the solubility information to select the appropriate solvent.							
In Vivo	1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 2.5 mg/mL (5.82 mM); Clear solution						
	2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.5 mg/mL (5.82 mM); Clear solution						
	3. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (5.82 mM); Clear solution						

### BIOLOGICAL ACTIVITY

Description	VY-3-135 is a potent, orally active, and stable ACSS2 inhibitor with an IC <sub>50</sub> value of 44 nM. VY-3-135 is specific to ACSS2 among the AcCoA synthetase family of enzymes. VY-3-135 does not inhibit ACSS1 or ACSS3 enzymatic activity. VY-3-135 can be used for the research of breast cancer <sup>[1]</sup> .
In Vitro	VY-3-135 (0.1, 1 μM; for 24 hours) blocks acetate dependent labeling of palmitate by 13C2-acetate in ACSS2 <sup>low</sup> A7C11 and ACSS2 <sup>high</sup> Brpkp110 cells <sup>[1]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	VY-3-135 (100 mg/kg/day; PO; 30 days) represses MDA-MB-468 (ACSS2 <sup>high</sup> ) tumor growth but is mostly ineffective at blocking

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WHIM12 (ACSS2<sup>low</sup>) growth<sup>[1]</sup>.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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## REFERENCES

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[1]. Katelyn D Miller, et al. Targeting ACSS2 with a Transition-State Mimetic Inhibits Triple-Negative Breast Cancer Growth. Cancer Res. 2021 Mar 1;81(5):1252-1264.

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

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