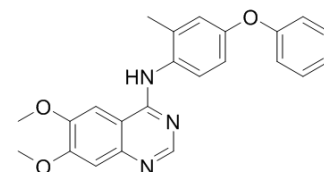


## APS-2-79

<b>Cat. No.:</b>	HY-100627		
<b>CAS No.:</b>	2002381-25-9		
<b>Molecular Formula:</b>	C <sub>23</sub> H <sub>21</sub> N <sub>3</sub> O <sub>3</sub>		
<b>Molecular Weight:</b>	387.43		
<b>Target:</b>	MEK		
<b>Pathway:</b>	MAPK/ERK Pathway		
<b>Storage:</b>	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



### SOLVENT & SOLUBILITY

<b>In Vitro</b>	DMSO : 20 mg/mL (51.62 mM; Need ultrasonic)			
		Solvent Concentration	Mass	
			1 mg	5 mg
			10 mg	
<b>Preparing Stock Solutions</b>	<b>1 mM</b>	2.5811 mL	12.9056 mL	25.8111 mL
	<b>5 mM</b>	0.5162 mL	2.5811 mL	5.1622 mL
	<b>10 mM</b>	0.2581 mL	1.2906 mL	2.5811 mL
Please refer to the solubility information to select the appropriate solvent.				
<b>In Vivo</b>	<ol style="list-style-type: none"> <li>Add each solvent one by one: 10% DMSO &gt;&gt; 40% PEG300 &gt;&gt; 5% Tween-80 &gt;&gt; 45% saline Solubility: ≥ 2 mg/mL (5.16 mM); Clear solution</li> <li>Add each solvent one by one: 10% DMSO &gt;&gt; 90% (20% SBE-β-CD in saline) Solubility: ≥ 2 mg/mL (5.16 mM); Clear solution</li> <li>Add each solvent one by one: 10% DMSO &gt;&gt; 90% corn oil Solubility: ≥ 2 mg/mL (5.16 mM); Clear solution</li> </ol>			

### BIOLOGICAL ACTIVITY

<b>Description</b>	APS-2-79 is a KSR-dependent MEK antagonist. APS-2-79 inhibits ATP <sup>biotin</sup> binding to KSR2 within the KSR2-MEK1 complex with an IC <sub>50</sub> of 120 nM. APS-2-79 makes the stabilization of the KSR inactive state antagonizes oncogenic Ras-MAPK signaling [1].	
<b>IC<sub>50</sub> &amp; Target</b>	KSR2 120 nM (IC <sub>50</sub> )	MEK1

## In Vitro

APS-2-79 (5  $\mu$ M) suppresses KSR-stimulated MEK and ERK phosphorylation in 293H cells<sup>[1]</sup>.

APS-2-79 (1  $\mu$ M) enhances the efficacy of the clinical MEK inhibitor trametinib within cancer cell lines containing K-Ras mutations<sup>[1]</sup>

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

## PROTOCOL

### Cell Assay <sup>[1]</sup>

Cell viability assays are performed in 96 well plates. Optimal cell densities for 96 well plate assays are determined to obtain linear growth over the time course of assays. A549, HCT-116, A375, SK-MEL-239, COLO-205, LOVO, SK-MEL-2, CALU-6, MEWO, SW620 and SW1417 cells are plated at 500 cells per well and treated with inhibitors (e.g., APS-2-79; 100-3,000 nM) for 72hrs before measuring viability. H2087 and HEPG2 cells are plated at 2000 cells per well, and treated with inhibitors (e.g., APS-2-79; 100-3,000 nM) for 72hrs. Cell viability is measured using Resazurin, and the percent cell viability is determined by normalizing inhibitor-treated samples to DMSO controls<sup>[1]</sup>.

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

## REFERENCES

[1]. Dhawan NS, et al. Small molecule stabilization of the KSR inactive state antagonizes oncogenic Ras signalling. Nature. 2016 Aug 24;537(7618):112-116.

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

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