**Product Name**: AMG-510  
**Catalog Number**: T8684  
**CAS Number**: 2296729-00-3  
**Molecular Formula**: C30H30F2N6O3  
**Molecular Weight**: 560.59  
**Appearance**:  
**Melting Point**:  

**Description**: AMG-510 is a selective and orally bioavailable KRAS G12C covalent inhibitor.

**Storage**: 2 years -80°C in solvent; 3 years -20°C powder;

<table>
<thead>
<tr>
<th>Solubility</th>
<th>H2O</th>
<th>33.33 mg/mL (59.46 mM), ultrasonic and adjust pH to 11 with NaOH</th>
</tr>
</thead>
<tbody>
<tr>
<td>DMSO</td>
<td>50 mg/mL (89.19 mM), Need ultrasonic</td>
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( < 1 mg/ml refers to the product slightly soluble or insoluble )

**Receptor (IC50)**: KRAS G12C

**In vitro Activity**
In cellular assays, AMG 510 covalently modified KRASG12C and inhibited KRASG12C signaling as measured by phosphorylation of ERK1/2 (p-ERK) in all KRAS p.G12C-mutant cell lines tested but did not inhibit p-ERK in cell lines with various other KRAS mutations. AMG 510 also selectively impaired viability of KRAS p.G12C mutant cell lines but did not affect cell lines with other KRAS mutations[1].

**In vivo Activity**
In vivo pharmacodynamic assays demonstrated dose- and time-dependent inhibition of KRASG12C signaling in human pancreatic and NSCLC tumor xenografts. Covalent modification of KRASG12C by AMG 510 was measured by mass spectrometry and correlated with p-ERK inhibition in tumors. AMG 510 significantly inhibited the growth of KRAS p.G12C xenografts and resulted in tumor regression. Combination treatment of AMG 510 with standard-of-care and targeted agents demonstrated enhanced tumor growth inhibition compared to either single agent. In a syngeneic model of KRAS p.G12C mutant cancer, AMG 510 treatment significantly inhibited tumor growth and caused regression[1].

**Reference**

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