BAY-1895344 hydrochloride

Cat. No.: HY-101566A
Molecular Formula: C₂₀H₂₂ClN₇O
Molecular Weight: 411.89
Target: ATM/ATR
Pathway: Cell Cycle/DNA Damage; PI3K/Akt/mTOR
Storage:
- Powder -20°C 3 years
  4°C 2 years
- In solvent -80°C 6 months
  -20°C 1 month
Solubility: DMSO: ≥ 100 mg/mL
* "<1 mg/mL" means slightly soluble or insoluble. "≥" means soluble, but saturation unknown.

**PREPARING STOCK SOLUTIONS**

<table>
<thead>
<tr>
<th>Volume Concentration</th>
<th>Mass</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM</td>
<td>2.4278 mL</td>
<td>12.1392 mL</td>
<td>24.2783 mL</td>
<td></td>
</tr>
<tr>
<td>5 mM</td>
<td>0.4856 mL</td>
<td>2.4278 mL</td>
<td>4.8557 mL</td>
<td></td>
</tr>
<tr>
<td>10 mM</td>
<td>0.2428 mL</td>
<td>1.2139 mL</td>
<td>2.4278 mL</td>
<td></td>
</tr>
</tbody>
</table>

Please refer to the solubility information to select the appropriate solvent.

**BIOLOGICAL ACTIVITY**

**Description**

BAY-1895344 hydrochloride is a potent, orally available and selective ATR inhibitor, with IC₅₀ of 7 nM.

**IC₅₀ & Target**

ATM, IC₅₀: 7 nM

**In Vitro**

BAY 1895344 is a selective low-nanomolar inhibitor of ATR kinase activity, potently inhibiting proliferation of a broad spectrum of human tumor cell lines (median IC₅₀ of 78 nM). In cellular mechanistic assays BAY 1895344 inhibits hydroxyurea-induced H2AX phosphorylation demonstrating the anticipated mode of action. In cellular mechanistic assays BAY 1895344 potently inhibits hydroxyurea-induced H2AX phosphorylation (IC₅₀=36 nM). Moreover, BAY 1895344 reveals significantly improved aqueous solubility, bioavailability across species and no activity in the hERG patch-clamp assay. BAY 1895344 also demonstrates very promising efficacy in monotherapy in DNA damage deficient tumor models as well as combination treatment with DNA damage inducing therapies.

**In Vivo**

BAY 1895344 exhibits strong in vivo anti-tumor efficacy in monotherapy in a variety of xenograft models of different indications that are characterized by DDR deficiencies, inducing stable disease in ovarian and colorectal cancer or even complete tumor remission in mantle cell lymphoma models.
REFERENCES
