CGK733

Cat. No.: HY-15520
CAS No.: 905973-89-9
Molecular Formula: C₂₃H₁₈Cl₃FN₄O₃S
Molecular Weight: 555.84
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

Solvent & Solubility

<table>
<thead>
<tr>
<th>In Vitro</th>
<th>10 mM in DMSO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preparing Stock Solutions</td>
<td></td>
</tr>
<tr>
<td>Solvent Concentration</td>
<td>Mass 1 mg</td>
</tr>
<tr>
<td>1 mM</td>
<td>1.7991 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>0.3598 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td>0.1799 mL</td>
</tr>
</tbody>
</table>

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

BIOLOGICAL ACTIVITY

Description
CGK733 is a potent ATM/ATR inhibitor, used for the research of cancer.

IC₅₀ & Target
ATM; ATR

In Vitro
CGK733 (4.2 ng/μL-12.5 ng/μL) enhances taxol-induced cytotoxicity in HBV-positive HCC cells. CGK733 (4.2 ng/μL) accelerates the formation of multinucleated cells and promotes the exit of mitosis in taxol-treated HBV-positive HCC cells[1]. CGK733 (10 μM) causes the loss of cyclin D1 through the ubiquitin-dependent proteasomal degradation pathway in MCF-7 and T47D breast cancer cell lines. CGK733 (0.6-40 μM) shows inhibitory activities against proliferation of LnCap prostate cancer cells, HCT116 colon cancer cells, MCF-7 and T47D estrogen receptor positive breast cancer cells, and MDA-MB436 ER negative breast cancer cells. Moreover, CGK733 inhibits proliferation of non-transformed mouse BALB/c 3T3 embryonic fibroblast cells. In addition, CGK733 (10 μM) inhibits MCF-7 proliferation, and the effect can not be suppressed by pan-caspase inhibition[2]. CGK733 (10 μM) results in 1.6-fold increase in ATM reporter activity in HEK-293 cells[3].
In Vivo

CGK733 (25 mg/kg, i.p.) increases the ATM reporter activity (reports inactivation of ATM kinase activity) compared to control mice, with 2.4-fold, 3.1-fold, and 1.3-fold changes at 1, 4, and 8 hours, respectively[^3].

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**PROTOCOL**

**Cell Assay[^2]**

Cells are seeded in 96-well plates at a predetermined optimal cell density to ensure exponential growth for duration of the assay. After a 24 h preincubation, growth medium is replaced with experimental medium containing the appropriate drug concentrations or 0.1% (v/v) vehicle control. After a 48 h incubation, cell proliferation is estimated using the sulforhodamine B colorimetric assay and expressed as the mean ± SE for six replicates as a percentage of vehicle control (taken as 100%). Experiments are performed independently at least three times. Statistical analyses are performed using a two-tailed Student's t test. P < 0.05 is considered to be statistically significant[^2].

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

**Animal Administration[^3]**

Four to six weeks old athymic CD-1 female mice are acclimatized for at least one week before use. The mice are injected sub-cutaneously with 2×10^6 D54-ATMR cells in each flank. Tumors are allowed to grow to the size of 100-150 mm^3. Mice are injected intraperitoneally with vehicle control (DMSO), CGK-733, KU-55933 (25 mg/kg) or irradiated with 5 Gy to each flank. Bioluminescence is acquired on Xenogen IVIS Spectrum system after injecting 400 μg/100 μL of D-luciferin at baseline (-3h) as well as 1, 4, and 8 hours after drug administration[^3].

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**REFERENCES**


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

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