Tasisulam sodium

Cat. No.: HY-14804A

CAS No.: 519055-63-1

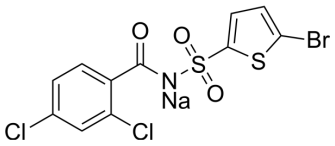
Molecular Formula: $C_{11}H_5BrCl_2NNaO_3S_2$

Molecular Weight: 437.09

Target: Apoptosis

Pathway: Apoptosis

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Tasisulam is a anticancer agent and induces apoptosis via the intrinsic pathway, resulting in cytochrome c release and caspase-dependent cell death. Tasisulam inhibits mitotic progression and induces vascular normalization[1].

In Vitro

Tasisulam sodium (200 nM-200 μM; 48 hours) induces an antiproliferative response across a wide range of tumor histologies with EC_{50}s of 10 μM and 25 μM for Calu-6 and A-375 cell lines, respectively[1].
Tasisulam sodium (25, 50 μM; 72 hours) induces a concentration-dependent increase in 4N DNA and G2-M accumulation[1].
Tasisulam sodium (200 nM-200 μM; 48 hours) induces apoptosis in a broad range of in vitro cancer cell models[1].
Tasisulam sodium also blocks VEGF, epidermal growth factor, and fibroblast growth factor-induced endothelial cell cord formation[1].

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

Cell Proliferation Assay[1]

Cell Line:	Calu-6 non-small cell lung carcinoma and A-375 melanoma models
Concentration:	200 nM-200 μM
Incubation Time:	48 hours
Result:	Induced an antiproliferative response across a wide range of tumor histologies with EC_{50}s is 10 μM and 25 μM, respectively.

Cell Cycle Analysis[1]

Cell Line:	Calu-6 and A-375 cell lines
Concentration:	25, 50 μM
Incubation Time:	72 hours
Result:	Induced a concentration-dependent increase in 4N DNA and G2-M accumulation.

Apoptosis Analysis[1]

Cell Line:	Calu-6 non-small cell lung carcinoma and A-375 melanoma models

Concentration:	200 nM-200 μM
Incubation Time:	48 hours
Result:	Induced apoptosis in a broad range of in vitro cancer cell models.

REFERENCES

[1]. Meier T, et al. Tasisulam sodium, an antitumor agent that inhibits mitotic progression and induces vascular normalization. Mol Cancer Ther. 2011 Nov;10(11):2168-78.

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