

Small Molecules

NU7026

NHEJ pathway inhibitor; Inhibits DNA-dependent protein kinase (DNA-PK)

Catalog # 74172
74174

5 mg
25 mg



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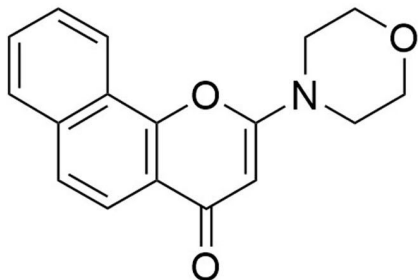
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Product Description

NU7026 is an inhibitor of DNA-dependent protein kinase (DNA-PK), an enzyme involved in the non-homologous end joining (NHEJ) DNA repair pathway. NU7026 sensitizes cells to radiation and has potential for anticancer therapies (Veuger et al.). NU7026 is also reported to increase the efficiency of homology-directed repair (HDR) in CRISPR-Cas9 genome editing (Riesenberg & Maricic; Zhang et al.).

Molecular Name:	NU7026
Alternative Names:	LY293646; DNA-PK Inhibitor II
CAS Number:	154447-35-5
Chemical Formula:	C ₁₇ H ₁₅ NO ₃
Molecular Weight:	281.3 g/mol
Purity:	≥ 95%
Chemical Name:	2-(4-morpholinyl)-4H-naphtho[1,2-b]pyran-4-one
Structure:	



Properties

Physical Appearance:	A crystalline solid
Storage:	Product stable at -20°C as supplied. Protect product from prolonged exposure to light. For long-term storage, store with a desiccant. Stable as supplied for 12 months from date of receipt.
Solubility:	· DMF ≤ 530 μM For example, to prepare a 300 μM stock solution in DMF, resuspend 1 mg in 12 mL of DMF.

Prepare stock solution fresh before use. Information regarding stability of small molecules in solution has rarely been reported, however, as a general guide we recommend storage in DMF at -20°C. Aliquot into working volumes to avoid repeated freeze-thaw cycles. The effect of storage of stock solution on compound performance should be tested for each application.

Compound has low solubility in aqueous media. For use as a cell culture supplement, stock solution should be diluted into culture medium immediately before use. Avoid final DMF concentration above 0.1% due to potential cell toxicity.

Published Applications

GENOME EDITING

- Increases precise genome editing by promoting HDR at the expense of NHEJ in human pluripotent stem cells (Riesenberg & Maricic; Zhang et al.).

CANCER RESEARCH

- Sensitizes human cancer cell lines to DNA double-strand break-inducing therapy (chemo- or radio-therapy) by inhibiting DNA-PK activity and inducing cell cycle arrest at G2/M phase (Albarakati et al.; Ma et al.; Niazi et al.; Willmore et al.; Yang et al.).

References

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- Riesenberg S & Maricic T (2018). Targeting repair pathways with small molecules increases precise genome editing in pluripotent stem cells. *Nat Comm* 9(1): 2164.
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- Zhang J-P et al. (2017) Efficient precise knockin with a double cut HDR donor after CRISPR/Cas9-mediated double-stranded DNA cleavage. *Genome Biol* 18(1): 35.

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