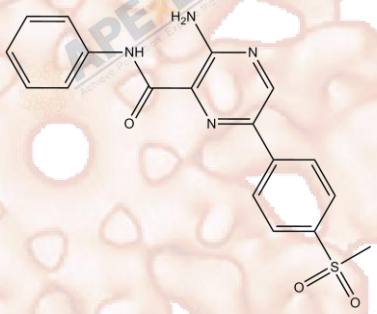
# Product Data Sheet

# VE-821



| | |
|---|---|
| **Cat. No.:** | A2521 |
| **CAS No.:** | 1232410-49-9 |
| **Formula:** | C18H16N4O3S |
| **M.Wt:** | 368.41 |
| **Synonyms:** | |
| **Target:** | Cell Cycle/Checkpoint |
| **Pathway:** | ATM/ATR |
| **Storage:** | Store at -20°C |

## Solvent & Solubility

In Vitro

≥62.5 mg/mL in DMSO; insoluble in EtOH; insoluble in H2O

Preparing Stock Solutions

| Mass / Solvent / Concentration | 1mg | 5mg | 10mg |
|---|---|---|---|
| **1 mM** | 2.7144 mL | 13.5718 mL | 27.1437 mL |
| **5 mM** | 0.5429 mL | 2.7144 mL | 5.4287 mL |
| **10 mM** | 0.2714 mL | 1.3572 mL | 2.7144 mL |

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

## Biological Activity

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|---|---|
| Shortsummary | ATR kinase inhibitor |
| $IC_{50}$ & Target | 13 nM/26 nM (Ki/IC50) (ATR) |

In Vitro

**Cell Viability Assay**

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|---|---|
| Cell Line: | HFL1 cells; HCT116 cancer cells; H23 cancer cell line. |
| Preparation method： | The solubility of this compound in DMSO is >10 mM. General tips for obtaining a higher concentration: Please warm the tube at 37°C for 10 minutes and/or shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months. |
| Reacting conditions: | 10 μM; 24, 48 or 96 h. |

| | | |
|---|---|---|
| | Applications: | HFL1 cells were pretreated with 10 μM VE-821 or DMSO before addition of 200 μM cisplatin (Cis), 1 μM gemcitabine (Gem), 100 μM etoposide (Etop) or 5 Gy ionizing radiation (IR), VE-821 blocks Chk1 Ser345 phosphorylation under all conditions and inhibits H2AX phosphorylation in treatment with cisplatin and gemcitabine. In the H23 cancer cell line, VE-821 shows marked synergy with cisplatin in growth arrest. |
| In Vivo | **Animal experiment** | |
| | Applications: | |

## Product Citations

1. Li Z, Liu B, et al."hDNA2 nuclease/helicase promotes centromeric DNA replication and genome stability." EMBO J. 2018 May 17. pii: e96729.PMID:29773570

2. Nanda Kumar Sasi, Flavie Coquel, et al. "DDK has a primary role in processing stalled replication forks to initiate downstream checkpoint signaling." bioRxiv. 2017.October 21.

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## References

[1]. Reaper PM1, Griffiths MR, Long JM, Charrier JD, Maccormick S, Charlton PA, Golec JM, Pollard JR. Selective killing of ATM- or p53-deficient cancer cells through inhibition of ATR. Nat Chem Biol, 2011, 7(7): 428-430.

## Caution

**FOR RESEARCH PURPOSES ONLY.**

**NOT FOR HUMAN, VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.**

*Specific storage and handling information for each product is indicated on the product datasheet. Most APExBIO products are stable under the recommended conditions. Products are sometimes shipped at a temperature that differs from the recommended storage temperature. Shortterm storage of many products are stable in the short-term at temperatures that differ from that required for long-term storage. We ensure that the product is shipped under conditions that will maintain the quality of the reagents. Upon receipt of the product, follow the storage recommendations on the product data sheet.*