Data Sheet

# Pifusertib

Product Code: F151605

CAS No.: 1402602-94-1

Mol. Weight: C26H24N4O2

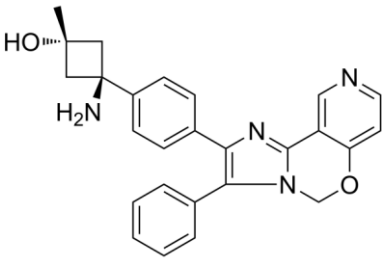
Mol. Formula: 424.49

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

Synonyms: TAS-117

Target: Akt; Apoptosis; Autophagy

Pathway: PI3K/Akt/mTOR; Apoptosis; Autophagy




## Biological Activity

**制备储备液:**

| 质量 / 溶剂体积 / 浓度 | 1 mg | 5 mg | 10 mg |
|---|---|---|---|
| 1 mM | 2.3558 mL | 11.7788 mL | 23.5577 mL |
| 5 mM | 0.4712 mL | 2.3558 mL | 4.7115 mL |
| 10 mM | 0.2356 mL | 1.1779 mL | 2.3558 mL |

Bioactive: Pifusertib (TAS-117) is a potent, selective, orally active allosteric Akt inhibitor (with IC50s of 4.8, 1.6, and 44 nM for Akt1, 2, and 3, respectively). Pifusertib triggers anti-myeloma activities and enhances fatal endoplasmic reticulum (ER) stress induced by proteasome inhibition. Pifusertib induces apoptosis and autophagy.

In Vivo: Pifusertib (12-16 mg/kg; p.o.; daily for 5 days a week, 21 days) inhibits tumor growth in murine xenograft models of human MM. Pifusertib enhances bortezomib-induced MM cytotoxicity in vivo. Animal Model: SCID mice (xenograft models bearing MM.1S cells) Dosage: 12, 16 mg/kg

Administration:P.o.; daily for 5 days a week, 21 days
Result:Significantly reduced MM.1S tumor growth versus vehicle control.

In Vitro: Pifusertib (1 μM; 6 hours) blocks basal phosphorylation of Akt and downstream p-FKHR/FKHRL1 in MM cells with high baseline p-Akt.Pifusertib (0-10 μM; 72 hours) selectively inhibits Akt and induces cytotoxicity in MM cells with high baseline phosphorylation of Akt.Pifusertib abrogates the cytoprotective effect of the bone marrow microenvironment associated with Akt inhibition in both MM cells and BMSCs. Pifusertib enhances Carfilzomib-induced cytotoxicity and fatal ER stress in MM cells. Pifusertib (0.5, 1 μM) triggers G0/G1 arrest followed by apoptosis, associated with induction of autophagy and endoplasmic reticulum stress response.Pifusertib enhances bortezomib-induced cytotoxicity, associated with increased CHOP (a fatal ER-stress marker) and PARP cleavage and blockade of bortezomib-induced p-Akt, suggesting that Pifusertib augments Bortezomib-induced ER stress and apoptotic signaling.