

Perifosine

Catalog No: tcsc0209



Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg

Size: 200mg

Size: 500mg

Size: 1g



Specifications

CAS No:

157716-52-4

Formula:

$C_{25}H_{52}NO_4P$

Pathway:

PI3K/Akt/mTOR;Autophagy

Target:

Akt;Autophagy

Purity / Grade:

>98%

Solubility:

H₂O : ≥ 153.33 mg/mL (332.13 mM); DMSO :

Alternative Names:

KRX-0401;NSC 639966;D21266

Observed Molecular Weight:

461.66

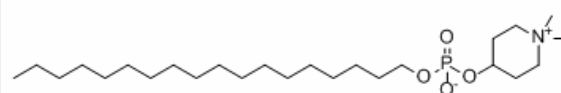
Product Description

Perifosine is an oral **Akt** inhibitor. All cells are sensitive to the antiproliferative properties of Perifosine with an IC_{50} of ~ 0.6 - $8.9 \mu M$.

IC_{50} & Target: Akt^[1]

In Vitro: The IC_{50} for growth of Ntv-a/LacZ cell lines is determined by MTT assay. When the cells are cultured for 48 hours in 10% FCS-supplemented media, the IC_{50} for cells with constitutively active PDGF, Ras, or Akt signaling is similar and found to be $\sim 45 \mu M$ ^[1]. Perifosine, a oral-bioavailable alkylphospholipid (ALK), on the cell cycle kinetics of immortalized keratinocytes (HaCaT) as well as head and neck squamous carcinoma cells. Proliferation is assessed by the incorporation of [³H]thymidine into cellular DNA. Exposure to Perifosine (0.1 - $30 \mu M$) for 24 h results in a dose-dependent inhibition of [³H]thymidine uptake in all cell lines tested. The IC_{50} s for growth are between 0.6 and $8.9 \mu M$, reaching IC_{80} s of $\sim 10 \mu M$. Perifosine blocks cell cycle progression of head and neck squamous carcinoma cells at G_1 -S and G_2 -M by inducing p21^{WAF1}, irrespective of p53 function, and may be exploited clinically because the majority of human malignancies harbor p53 mutations. Perifosine ($20 \mu M$) induces both G_1 -S and G_2 -M cell cycle arrest, together with p21^{WAF1} expression in both p53 wild-type and p53^{-/-} clones^[2].

In Vivo: Mice are identified with tumors by bioluminescence imaging and either treated them with 100 mg/kg Temozolomide, or 30 mg/kg Perifosine, or a combination with 100 mg/kg Temozolomide and 30 mg/kg Perifosine (Temozolomide+Perifosine) for 3 to 5 days. The mice are sacrificed and tumors analyzed histologically for cell proliferation by Ki-67 immunostaining. Ki-67 staining index is significantly reduced in mice treated with either Temozolomide (Ki-67 staining index= $5.5 \pm 1.2\%$, n=4, P=0.0019) or Perifosine (Ki-67 staining index= $3.2 \pm 1.1\%$, n=3, P=0.001) compared with Control, demonstrating the inhibitory effect on proliferation. Most importantly, the tumors treated with Temozolomide+Perifosine have the lowest Ki-67 staining index ($1.7 \pm 1.2\%$, n=3, P=0.0005). The additional treatment with Perifosine results in a significantly lower proliferation rate than Temozolomide alone (P=0.0087)^[1]. Perifosine markedly decreases p-Akt from 10 min to 24 hours and subsequently, moderately decreased p-S6 from 1h to 24 h after injection^[3].



All products are for RESEARCH USE ONLY. Not for diagnostic & therapeutic purposes!