Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

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 :

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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 μM.

IC50 & 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 ~45 μM[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 [^{3}H]thymidine into cellular DNA. Exposure to Perifosine (0.1-30 μM) for 24 h results in a dose-dependent inhibition of [^{3}H]thymidine uptake in all cell lines tested. The IC_{50}s for growth are between 0.6 and 8.9 μM, reaching IC_{80}s of ~10 μM. 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 μM) 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±1.2%, n=4, P=0.0019) or Perifosine (Ki-67 staining index=3.2±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±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].