

# 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:**

H<sub>2</sub>O : ≥ 153.33 mg/mL (332.13 mM); DMSO :

**Alternative Names:**

KRX-0401;NSC 639966;D21266

**Observed Molecular Weight:**

461.66

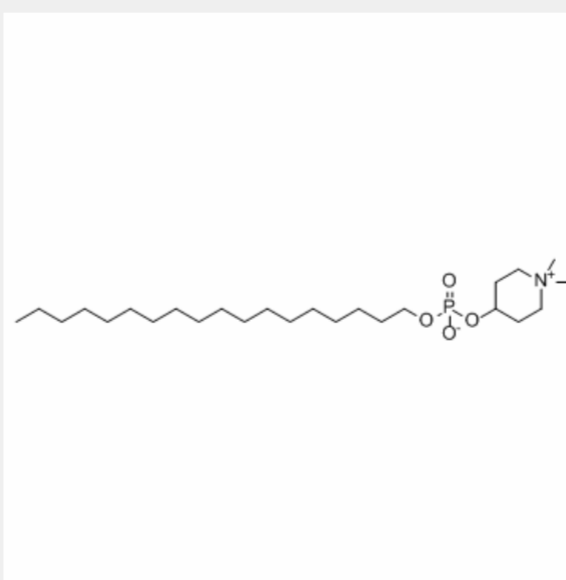
**Product Description**

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

IC50 & Target: Akt<sup>[1]</sup>

**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  $\sim 45 \mu M$ <sup>[1]</sup>. 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 [<sup>3</sup>H]thymidine into cellular DNA. Exposure to Perifosine (0.1-30  $\mu M$ ) for 24 h results in a dose-dependent inhibition of [<sup>3</sup>H]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<sub>1</sub>-S and G<sub>2</sub>-M by inducing p21<sup>WAF1</sup>, 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<sub>1</sub>-S and G<sub>2</sub>-M cell cycle arrest, together with p21<sup>WAF1</sup> expression in both p53 wild-type and p53<sup>-/-</sup> clones<sup>[2]</sup>.

**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)<sup>[1]</sup>. Perifosine markedly decreases p-Akt from 10 min to 24 hours and subsequently, moderately decreased p-S6 from 1h to 24 h after injection<sup>[3]</sup>.



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