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

Ailanthone

Catalog No: tcsc0018254

Available Sizes

Size: 5mg

Size: 10mg

Specifications

CAS No:
981-15-7

Formula:
$C_{20}H_{24}O_7$

Pathway:
Others

Target:
Androgen Receptor

Purity / Grade:
>98%

Solubility:
DMSO : 83.3 mg/mL (221.31 mM; Need warming)

Alternative Names:
Δ13-Dehydrochaparrinone

Observed Molecular Weight:
376.4

Product Description

Ailanthone (Δ13-Dehydrochaparrinone) is a potent inhibitor of both full-length **androgen receptor (AR)** (**IC_{50}**=69 nM) and constitutively active truncated AR splice variants ($AR_{1\text{-}651}$**IC_{50}**=309 nM).

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IC50 & Target: IC50: 69 nM (Full-length androgen receptor), 309 nM (AR_{1-651})[1]

In Vitro: Ailanthone is a potent inhibitor of both full-length AR (AR-FL) and constitutively active truncated AR splice variants (AR-Vs). Ailanthone binds to the co-chaperone protein p23 and prevents AR\'s interaction with HSP90, thus resulting in the disruption of the AR-chaperone complex followed by ubiquitin/proteasome-mediated degradation of AR as well as other p23 clients including AKT and Cdk4, and downregulates AR and its target genes in PCa cell lines and orthotopic animal tumours. In addition, Ailanthone blocks tumour growth and metastasis of CRPC[1]. Ailanthone has been shown to possess an growth-inhibitory effect against several cancer cell lines including HepG2, Hep3B, R-HepG2, Jurkat, HeLa, MCF-7, MDA-MB-231 and A549 cells. Ailanthone inhibits Huh7 cell growth through the induction of mitochondrion-mediated cell apoptosis and G0/G1 cell cycle arrest. Ailanthone-induced apoptosis is mitochondrion-mediated and involved the PI3K/AKT signaling pathway in Huh7 cells[2].

In Vivo: Not only i.p. administration but also p.o. administration of Ailanthone has excellent efficiency for blocking the growth of CRPC xenografts. In pharmacokinetic studies, Ailanthone exhibits good solubility in water and good bioavailability (>20%). In addition, Ailanthone effectively suppresses CRPC tumour growth, despite not reaching a steady state of plasma drug concentration during the course of treatment[1].