

CBL0137 hydrochloride

Catalog No: tcsc0033711

Available Sizes

Size:	2mg
Size:	5mg
Size:	10mg
Size:	25mg
Size:	50mg
	Specifications

CAS No: 1197397-89-9

Formula:

 $\mathsf{C}_{21}\mathsf{H}_{25}\mathsf{CIN}_2\mathsf{O}_2$

Pathway: Apoptosis;NF-κB

Target: MDM-2/p53;NF-κB

Purity / Grade:

>98%

Solubility:

H2O : 15.2 mg/mL (40.76 mM; Need ultrasonic and warming); DMSO : 30 mg/mL (80.45 mM; Need ultrasonic and warming)

Alternative Names:

Curaxin-137 hydrochloride;CBL-C137 hydrochloride

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Observed Molecular Weight:

372.89

Product Description

CBL0137 hydrochloride is an inhibitor of the histone chaperone, **FACT**. CBL0137 hydrochloride can also activate **p53** and inhibits **NF-\kappaB** with **EC**₅₀s of 0.37 and 0.47 μ M, respectively.

IC50 & Target: FACT^[1]

EC50: 0.37 μM (p53), 0.47 μM (NF-Kb)^[2]

In Vitro: Treatment with CBL0137 hydrochloride leads to complete absence of living cells at concentrations above 2.5 μM. CBL0137 hydrochloride causes a greater reduction in the number of colonies formed of not only MiaPaCa-2 cells when combines with gemcitabine, but also gemcitabine-resistant PANC-1 cells. Treatment of human pancreatic cancer cells with CBL0137 hydrochloride results in a dose dependent reduction of protein and mRNA levels of RRM1 and RRM2^[1].

In Vivo: The CBL0137 hydrochloride monotherapy group and the CBL0137 hydrochloride-gemcitabine combination group samples show large necrotic fields, numerous apoptotic bodies and loss of tumor cells. Sub-optimal doses of 50 to 60 mg/kg CBL0137 hydrochloride causes similar enhancement of gemcitabine antitumor activity as that produced by the maximum tolerated dose (MTD) of 90 mg/kg as indicated by the lack of statistically significant differences among the combination groups. CBL0137 hydrochloride inhibits FACT function through depletion of the pool of active FACT involved in transcription elongation^[1]. CBL0137 hydrochloride, given by oral gavage at a nontoxic dose of 30 mg/kg per day on a 5 days on/2 days off schedule, suppresses tumor growth in xenografts of colon (DLD-1), renal cell carcinoma (Caki-1), and melanoma (Mel-7) tumor cell lines and transplanted surgical samples from patients with pancreatic ductal adenocarcinoma^[2].



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