Web: www.taiclone.com
Tel: +886-2-2735-9682
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Phenformin (hydrochloride)

Catalog No: tcsc1850

Available Sizes

Size: 1g

Size: 5g

Size: 10g

Size: 50g

Specifications

CAS No:
834-28-6

Formula:
$C_{10}H_{16}ClN_5$

Pathway:
Epigenetics;PI3K/Akt/mTOR

Target:
AMPK;AMPK

Purity / Grade:
>98%

Solubility:
DMSO :

Alternative Names:
Phenethylbiguanide hydrochloride

Observed Molecular Weight:
241.72

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Product Description

Phenformin (hydrochloride) is a hydrochloride salt of phenformin that is an anti-diabetic drug from the biguanide class, can activate **AMPK** activity.

IC50 & Target: AMPK[2]

In Vitro: Phenformin stimulates the phosphorylation and activation of AMPKalpha1 and AMPKalpha2 without altering LKB1 activity[1]. Phenformin increases AMPK activity and phosphorylation in the isolated heart, the increase in AMPK activity is always preceded by and correlated with increased cytosolic [AMP][2]. Phenformin is a 50-fold more potent inhibitor of mitochondrial complex I than metformin. Phenformin robustly induces apoptosis in LKB1 deficient NSCLC cell lines. Phenformin at 2 mM similarly induces AMPK signaling as shown by increased P-AMPK and P-Raptor levels. Phenformin induces higher levels of cellular stress, triggering induction of P-Ser51 eIF2α and its downstream target CHOP, and markers of apoptosis at later times. Phenformin induces a significant increase in survival and therapeutic response in KLluc mice following long-term treatment[3]. Phenformin and AICAR increases AMPK activity in H441 cells in a dose-dependent fashion, stimulating the kinase maximally at 5-10 mm and 2 mm, respectively. Phenformin significantly decreases basal ion transport (measured as short circuit current) across H441 monolayers by approximately 50% compared with that of controls. Phenformin and AICAR significantly reduce amiloride-sensitive transepithelial Na+ transport compared with controls. Phenformin and AICAR suppress amiloride-sensitive Na^+ transport across H441 cells via a pathway that includes activation of AMPK and inhibition of both apical Na+ entry through ENaC and basolateral Na^+ extrusion via the Na^+,K^+-ATPase[4]. Phenformin-treated rats reveals a tendency towards a decrease in blood insulin level (radioimmunoassay)[5].

In Vivo: Phenformin increases levels of P-eIF2α and its target BiP/Grp78 in normal lung as well as in lung tumors of mice[3].