

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

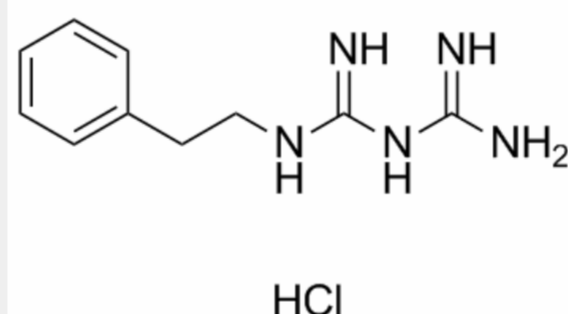
Product Description

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].



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