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## Catalog Number: CM05962

产品信息

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CAS号: 1383716-40-2

分子式: C<sub>17</sub>H<sub>17</sub>N<sub>7</sub>

主要靶点: Autophagy|PI3K

主要通路: PI3K/Akt/mTOR信号通路|自噬

分子量: 319.36 溶解度:

H2O:<1 mg/mL,DMSO:59 mg/mL (184.7 mM),Ethanol:59 mg/mL (184.7 mM)

Vps34:0.018  $\mu$  M|Pl3K  $\delta$  :1.2  $\mu$  M

体外活性

VPS34 enzymatic function is essential for LC3 lipidation in mammalian cells and PIK-III is a robust inhibitor of autophagy and LC3 lipidation in mammalian cells. In H4 cells, PIK-III inhibits the formation of autolysosomes and increases the cytosolic signal of LC3 under basal conditions and when autophagy is induced with the mTOR inhibitor AZD8055. In a CCCP-induced mitophagy model, PIK-III inhibits the clearance of mitochondria.PIK-III treatment leads to an increase in the levels of LC3-I in H4 and PSN1 cells. In Panc10.05 cells, PIK-III increases the levels of LC3-I in parallel with LC3-I suggesting a cell type-specific response[1].

体内活性

The DFX-induced NCOA4-dependent turnover of FTH1 and FTL is blocked with PIK-III which suggests an autophagy-dependent

细胞实验

To determine whether inhibition of VPS34 function impacts autophagy,LC3 and known autophagy substrates such as damaged mitochondria or the autophagy cargo receptor p62 are monitored. H4 cells expressing mCherry–GFP–LC3 are treated overnight with the indicated compounds, fixed, stained with Hoechst 33342 and imaged by automated acquisition. HeLa cells expressing GFP–Parkin are treated with PIK-III for 12 h followed by the addition of CCCP for 12 h, fixed, stained for endogenous Tom20 and imaged (Only for Reference) imaged. (Only for Reference)

Vps34-PIK-III (VPS34-IN2), a selective inhibitor of VPS34 enzymatic activity, inhibits autophagy and results in the stabilization of autophagy substrates.

储存

Powder: -20°C for 3 years | In solvent: -80°C for 1 year