Chemical Properties

- **Product Name:** Splitomicin
- **Cas No.:** 5690-03-9
- **M.Wt:** 198.22
- **Formula:** C13H10O2
- **Chemical Name:** 1,2-dihydrobenzo[f]chromen-3-one
- **Canonical SMILES:** C1CC(=O)OC2=C1C3=CC=CC=C3C=C2
- **Solubility:** >9.05mg/mL in DMSO
- **Storage:** Store at -20°C
- **General tips:** For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.
- **Shopping Condition:** Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request

Biological Activity

- **Targets:** Chromatin/Epigenetics
- **Pathways:** Sirtuin
- **Description:** Splitomicin is a selective inhibitor of Sir2p with IC50 value of 60 μM and also a inhibitor of fMLP-induced free radicals with IC50 value of 40.79 ± 9.85 μM [1, 3]. Inhibition of the HDA of Sir2p was the most likely mechanism by which splitomicin caused its phenotypic changes. The direct target of splitomicin is Sir2p deacetylase activity. In addition, splitomicin specific inhibited fMLP-induced superoxide anion release. In vitro, splitomicin inhibited NAD+-dependent histone deacetylase activity of the Sir2 protein with an IC50 value of 60μM. By using a [3H]-acetylated histone H4 peptide and measuring the NAD+-dependent release of free [3H]acetate in the presence of whole yeast cell extract from an hst2
strain overexpressing yeast SIR2, a cell extract was obtained from a SIR2-overexpressing hst2 strain. The result established Sir2p deacetylase activity as a direct target of splitomicin. In addition, neutrophils induced by either fMLP (1 μM) or PMA (100 nM) were observed using a flow cytometer and the intracellular production of superoxide anions was investigated at different splitomicin concentrations. Splitomicin inhibited fMLP-induced Mac-1 expression and increase cAMP levels in human neutrophils [1, 3].

Splitomicin’s naphthoic moiety might be responsible for its inhibitory effects on platelets. By using washed human platelets, the inhibitory effects of splitomicin on platelet aggregation were studied and platelet aggregation and ATP release induced by thrombin (0.1 U/ml), collagen (2 μg/ml), arachidonic acid (0.5 mM), U46619 (2 μM) or ADP (10 μM) was monitored. Splitomicin inhibited platelet aggregation in a concentration dependent manner. Splitomicin increased cAMP and this effect was enhanced when splitomicin (150 μM) was combined with PGE1 (0.5 μM). The inhibitory mechanism of splitomicin on platelet aggregation may increase cyclic AMP levels via inhibition of cyclic AMP phosphodiesterase activity and subsequent inhibition of intracellular Ca ion mobilization, TXB2 formation and ATP release [2].

Reference:

Caution

FOR RESEARCH PURPOSES ONLY.

NOT FOR HUMAN, VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

Specific storage and handling information for each product is indicated on the product datasheet. Most ApexBio products are stable under the recommended conditions. Products are sometimes shipped at a temperature that differs from the recommended storage temperature. Short-term storage of many products are stable in the short-term at temperatures that differ from that required for long-term storage. We ensure that the product is shipped under conditions that will maintain the quality of the reagents. Upon receipt of the product, follow the storage recommendations on the product data sheet.