

Product Data Sheet

Product Name: BCS12
Cat. No.: GC81595

Chemical Properties

Cas. No. 329719-71-3

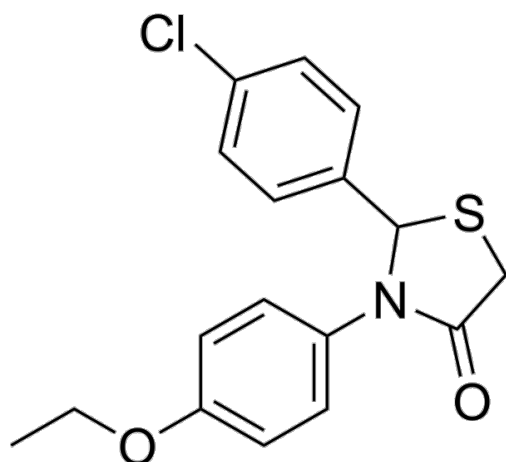
Formula $C_{17}H_{16}ClNO_2S$ M.Wt 333.83

Solubility DMSO: 100 mg/mL (299.55 mM; Need ultrasonic) Storage Store at 4°C, stored under nitrogen

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.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request.

Structure



Background

BCS12 is a CAIX inhibitor. BCS12 inhibits the viability of cancer cells under hypoxic conditions. BCS12 elevates ROS levels and suppresses the expression of HIF-1 α and CA9. BCS12

Caution: Product has not been fully validated for medical applications. For research use only.

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downregulates Bcl-2, activates caspase-9, caspase-3, caspase-7 and Bax, and induces PARP cleavage and apoptosis. BCS12 reduces tumor burden, increases the number of apoptotic nuclei and restores tissue structure in rats bearing breast tumors. BCS12 can be used in research related to triple-negative breast cancer [1].

In Vivo, BCS12 (20-40 mg/kg; i.p.; once daily; for 28 consecutive days) exerts dose-dependent anti-breast cancer activity in DMBA-induced rat breast tumor models, reduces tumor burden, inhibits body weight loss, and induces apoptosis[1].

In Vitro, BCS12 (96 h) selectively inhibits the viability of triple-negative breast cancer cells MDA-MB-231 and MDA-MB-468 under hypoxic conditions (IC_{50} = 15.49 μ M and 18.81 μ M), while showing extremely low toxicity toward non-tumorigenic MCF-10A cells (IC_{50} = 121.6 μ M)[1]. BCS12 (5-10 μ M; 14 days) dose-dependently inhibits the clonogenic survival capacity of MDA-MB-231 and MDA-MB-468 cells[1]. BCS12 (20 μ M) inhibits hypoxia-induced expression of HIF-1 α and CAIX proteins in MDA-MB-231 and MDA-MB-468 cells[1]. BCS12 (2-20 μ M; 24 h) dose-dependently increases the ROS level in MDA-MB-231 cells, with the strongest effect observed at 20 μ M[1]. BCS12 (5-20 μ M; 24 h) dose-dependently regulates the expression of apoptotic proteins in MDA-MB-231 cells, downregulates the anti-apoptotic protein Bcl-2, activates the pro-apoptotic caspase-9 and caspase-3, and induces PARP cleavage. It also induces apoptosis in MDA-MB-231 cells under hypoxic conditions, upregulates the expression of pro-apoptotic genes Bax, caspase-9, caspase-3 and caspase-7, and downregulates the anti-apoptotic gene Bcl-2 as well as hypoxia-related CA9 and HIF-1 α [1].

References:

[1]. Ranjan G, et al. CAIX-HIF-1 α Inhibition by 2-(4-Chlorophenyl)-3-(4-Ethoxyphenyl) Thiazolidin-4-one (BCS12) Activates Caspase Cascade and PARP Cleavage, Driving Mitochondrial Apoptosis in Hypoxic Breast Cancer. Recent Pat Anticancer Drug Discov. 2026 May 14.

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