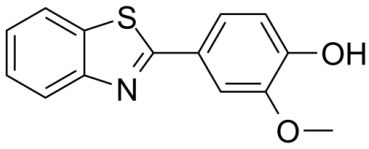
Data Sheet

Product Name:	YL-109
Cat. No.:	CS-3503
CAS No.:	36341-25-0
Molecular Formula:	C14H11NO2S
Molecular Weight:	257.31
Target:	Others
Pathway:	Others
Solubility:	DMSO : 100 mg/mL (388.64 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

YL-109 is a novel anticancer agent which has ability to inhibit breast cancer cell growth and invasiveness in vitro and in vivo. IC50 value: 85.7 nM(MCF-7 cells proliferation) [1] Target: AhR signaling activcator in vitro: YL-109 strongly inhibited cell proliferation of MCF-7 cells in a dose-dependent manner (IC50= 85.8 nM). Surprisingly, YL-109 had an anti-proliferative effect in a dose-dependent manner (IC50 = 4.02 μM) on MDA-MB-231 cells. YL-109 repressed the sphere-forming ability and the expression of stem cell markers in MDA-MB-231 mammosphere cultures. YL-109 increased the expression of carboxyl terminus of Hsp70-interacting protein (CHIP), which suppresses tumorigenic and metastatic potential of breast cancer cells by inhibiting the oncogenic pathway. YL-109 induced CHIP transcription because of the recruitment of the aryl hydrocarbon receptor (AhR) to upstream of CHIP gene in MDA-MB-231 cells. Consistently, the antitumor effects of YL-109 were depressed by CHIP or AhRknockdown in MDA-MB-231 cells [1]. in vivo: Mice treated with vehicle showed significantly enlarged tumors, whereas mice treated with YL-109 showed attenuated tumor growth using MCF-7 cells. Interestingly, YL-109 also suppressed tumor growth in mice injected with MDA-MB-231 cells. Compared with the vehicle control, YL-109 significantly reduced lung metastasis [1].

PROTOCOL (Extracted from published papers and Only for reference)

Cell assay(Invasion and migration assay) [1]: The invasive potentials of MDA-MB-231 and BT-20 cells were tested with Matrigel invasion chambers (24-well format, 8 μm pore size; BD Biosciences). After incubation in DMEM containing 1% charcoal-stripped FBS with DMSO or YL-109 (1 μM) for 48 h, suspensions (0.5 mL) containing 1 × 10 5 cells (MDA-MB-231) or 0.5 × 10 5 cells (BT-20) were added with vehicle alone (DMSO) or YL-109 (1 μM), and transferred into insert chambers. These cells were then incubated for 24 h at 37°C with 0.75 mL of DMEM containing 4% charcoal-stripped FBS and each ligand in the bottom chambers. After incubation, the cells on the upper surface of the filter were removed, and invading cells were fixed in methanol. Fixed cells were stained with crystal violet and counted under a microscope. Migration assays were performed using the same procedure, except that the insert chambers were not coated with Matrigel and cells in chamber were incubated for 12 h. Animal adminstration(Tumor xenograft models) [1]: BALB/cAjcl-nu/nu female mice at 4-5 weeks of age were purchased from CLEA Japan. The mice were kept in a pathogen-free environment under controlled conditions of light and humidity. MCF-7 or MDA-MB-231 cells were cultured as monolayers, trypsinized and resuspend in Matrigel (BD Biosciences) at each 1 × 10 8 or 1 × 10 7cells/ml. Each mouse was injected subcutaneously with 100μL of cell suspension (1 × 107 or 1 × 106 cells) in both flanks. YL-109 was subcutaneously injected in the scruff of the neck (15 mg/kg) for every 2 days. Tumor growth was monitored twice each week by measuring the tumor size using calipers; tumor volume was determined using the formula V = 1/2 × larger diameter × (smaller diameter)2. All animal experiments were performed in accordance with institutional guidelines.

References:

[1]. Hiyoshi H, et al. 2-(4-Hydroxy-3-methoxyphenyl)-benzothiazole suppresses tumor progression and metastatic potential of breast cancer cells by inducing ubiquitin ligase CHIP. Sci Rep. 2014 Nov 18;4:7095.

CAIndexNames:

Phenol, 4-(2-benzothiazolyl)-2-methoxy-

SMILES:

OC1=CC=C(C2=NC3=CC=CC=C3S2)C=C1OC

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