产品名称：ALPHA-[2-[4-(3,4-二氯苯基)-2-噻唑基]亚肼基]-2-硝基苯丙酸
产品别名：4EGI-1

生物活性：

Description	4EGI-1 is an inhibitor of eIF4E/eIF4G interaction, with a K_d of 25 μM against eIF4E binding.
IC_{50} & Target	Kd: 25 μM (eIF4E/eIF4G)[1]
In Vitro	4EGI-1 is an inhibitor of eIF4E/eIF4G interaction, with a Kd of 25 μM against eIF4E binding. 4EGI-1 disrupts the eIF4F complex and inhibits expression of oncogenic proteins in mammalian cells. 4EGI-1 (0-40 μM) also exhibits proapoptotic activity and inhibits the growth of multiple cancer cell lines[1]. 4EGI-1 is cytotoxic to breast cancer cells, such as SKBR-3, MCF-7 and MDA-MB-231 cells, with the IC50 of appr 30 μM, and to the non-CSCs (Cancer stem cells), the IC50 is about 22 μM. 4EGI-1 enhances breast CSC differentiation (40 μM), and suppresses breast CSC induced HUVEC tube-like structure formation (8 μM). Moreover, 4EGI-1 selectively inhibits translation that persists in CSC maintenance and dissemination[2]. 4EGI-1 (50 μM) impairs the formation of eIF4F complex in U87 cells. 4EGI-1 (10, 50 and 100 μM) inhibits cell proliferation via inducing apoptosis in U87 cells, and the apoptosis is via Bax activation. 4EGI-1 causes mitochondrial dysfunction, and induces ER stress via GRP-78 activation, in U87 cells[3].
In Vivo	4EGI-1 (75 mg/kg, i.p.) inhibits breast cancer stem cells (CSC) tumor growth and tumorangiogenesis in vivo[2]. 4EGI-1 (75 mg/kg, i.p.) shows inhibitory effect on the tumor volume and weight in mice bearing U87 cells[3].
Solvent&Solubility	***In Vitro:*** **DMSO : ≥ 35 mg/mL (77.56 mM)** * "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.2159 mL	11.0796 mL	22.1592 mL
	5 mM	0.4432 mL	2.2159 mL	4.4318 mL
	10 mM	0.2216 mL	1.1080 mL	2.2159 mL

*请根据产品在不同溶剂中的溶解度选择合适的溶剂配制储备液；一旦配成溶液，请分装保存，避免反复冻融造成的产品失效。

储备液的保存方式和期限：-80°C, 6 months; -20°C, 1 month。-80°C 储存时，请在 6 个月内使用，-20°C 储存时，请在 1 个月内使用。

References	[1]. Moerke NJ, et al. Small-molecule inhibition of the interaction between the translation initiation factors eIF4E and eIF4G. Cell. 2007 Jan 26;128(2):257-67. [2]. Yi T, et al. 4EGI-1 targets breast cancer stem cells by selective inhibition of translation that persists in CSC maintenance, proliferation and metastasis. Oncotarget. 2014 Aug 15;5(15):6028-37. [3]. Wu M, et al. Anti-Cancer Effect of Cap-Translation Inhibitor 4EGI-1 in Human Glioma U87 Cells: Involvement of Mitochondrial Dysfunction and ER Stress. Cell Physiol Biochem. 2016;40(5):1013-1028. Epub 2016 Dec 12.

实验参考：

Cell Assay	1×10^4 breast CSCs HMLER (CD44high/CD24low)FA cells and other indicated breast cancer cells are treated with DMSO, or [E]-4EGI-1 or [Z]-4EGI-1 at series of concentrations for 24 hours. The

	cells are performed cell viability assays with cell viability assay kit. Three independent experiments are performed. Average IC50 results are shown (mean ± SD, t-test, two-tailed)[2].
Animal Administration	Mice[2] In the tumor xenografted assay, 1×10^5 breast cancer stem cells (CSCs) are mixed with 100 μL Matrigel/DMEM mixture (Matrigel: DMEM = 1:2). Breast CSCs/Matrigel/DMEM mixtures are injected into NOD/SCID female mice mammary glands by subcutaneous injection. After the tumor formation (about 75 mm^3 in volume, 5 mice/group), DMSO, or 75 mg/kg [E]-4EGI-1, or 75 mg/kg [Z]-4EGI-1 is injected into the mice by intraperitoneal injection daily for 30 days. Tumor volumes are measured every three days. At the 30th day, mice are sacrificed and tumors are excised. Tumors weights are measured. Tumor tissue samples are used for immunohistostaining, Western blot and immunoprecipitation analyses[2].
References	[1]. Moerke NJ, et al. Small-molecule inhibition of the interaction between the translation initiation factors eIF4E and eIF4G. Cell. 2007 Jan 26;128(2):257-67. [2]. Yi T, et al. 4EGI-1 targets breast cancer stem cells by selective inhibition of translation that persists in CSC maintenance, proliferation and metastasis. Oncotarget. 2014 Aug 15;5(15):6028-37. [3]. Wu M, et al. Anti-Cancer Effect of Cap-Translation Inhibitor 4EGI-1 in Human Glioma U87 Cells: Involvement of Mitochondrial Dysfunction and ER Stress. Cell Physiol Biochem. 2016;40(5):1013-1028. Epub 2016 Dec 12.