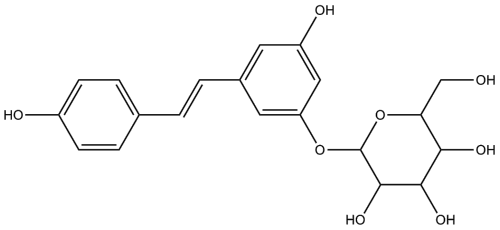
Product Data Sheet

Product Name: Polydatin
Cat. No.: GN10331

Chemical Properties

Cas. No.	27208-80-6		
Chemical Name	(2S,3R,4S,5S,6R)-2-[3-hydroxy-5-[(E)-2-(4-hydroxyphenyl)ethenyl]phenoxy]-6-(hydroxy methyl)oxane-3,4,5-triol		
SMILES	C1=CC(=CC=C1C=CC2=CC(=CC(=C2)OC3C(C(C(C(O3)CO)O)O)O)O)O		
Formula	$C_{20}H_{22}O_8$	M.Wt	390.38
Solubility	150mg/ml in DMSO	Storage	Store at 2-8°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.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure



Protocol

Cell experiment [1]:

Cell lines	Human lung epithelial cells (BEAS-2B) and human lung adenocarcinoma cells (A549)

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Preparation Method	BEAS-2B and A549 cells were cultured in DMEM medium with 10% fetal bovine serum and 1% penicillin/streptomycin in a 5% CO_2 incubator at 37°C. When the cell density reached 70–80%, the cells were treated with Polydatin at various concentrations (0, 20, 40, 60, 80, 100, and 120μM) for 24, 48, and 72 hours. For combined irradiation (IR) treatment, Polydatin was added 1 hour before 8Gy γ-ray irradiation, and cell proliferation was measured at 24, 48, and 72 hours post-IR.
Reaction Conditions	0 to 120μM; 24, 48, and 72 hours.
Applications	Polydatin significantly inhibited the proliferation of both BEAS-2B and A549 cells in a concentration- and time-dependent manner. Polydatin enhanced the radiosensitivity of A549 lung cancer cells.

Animal experiment [2]:

Animal models	Athymic BALB/c female nude mice
Preparation Method	H460 non-small cell lung cancer cells (2.5×10^6 cells per 100μl PBS/Matrigel mixture, 1:1) were subcutaneously injected into the flanks of nude mice. When tumor volumes reached ~100mm³, mice were randomly divided into five groups (n = 5 per group): control (vehicle only), cisplatin (2mg/kg, i.p., every 3 days), Polydatin (PD, 50mg/kg, i.p., every 2 days), cisplatin+PD (same doses and schedules as single treatments), and cisplatin+PD+NAC (N-acetyl-L-cysteine, 0.5g/l in drinking water). Treatments lasted for 2 weeks.
Dosage form	50mg/kg; i.p.

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Applications	Polydatin significantly reduced tumor volume and weight, enhanced the antitumor efficacy of cisplatin, and increased intratumoral ROS levels. These effects were associated with upregulation of NOX5, activation of ER stress (p-eIF2α, ATF4), and phosphorylation of JNK and p38 MAPK. Additionally, Polydatin decreased Ki-67-positive proliferating cells and showed no significant toxicity in major organs (heart, liver, kidney, lung). Co-administration of the ROS scavenger NAC reversed the antitumor effects, confirming the ROS-dependent mechanism.

References:
[1] Guo J, Ding W, Cai S, et al. Polydatin radiosensitizes lung cancer while preventing radiation injuries by modulating tumor-infiltrating B cells. J Cancer Res Clin Oncol. 2023 Sep;149(12):9529-9542.
[2] Wu S, Zhao Q, Liu S, Vanbever R, et al. Polydatin, a potential NOX5 agonist, synergistically enhances antitumor activity of cisplatin by stimulating oxidative stress in non?small cell lung cancer. Int J Oncol. 2024 Aug;65(2):77.

Background

Polydatin is a natural stilbene glucoside extracted from the traditional Chinese medicine *Polygonum cuspidatum*, and Polydatin is the glycoside form of resveratrol [1]. Polydatin exhibits a wide range of biological activities and is widely used in research on metabolic, cardiovascular, and neurodegenerative diseases[2]. Polydatin mechanisms of action involve multiple signaling pathways, including SIRT1, NF-κB, MAPK, and Nrf2/HO-1[3]. Studies have shown that Polydatin can alleviate ischemia-reperfusion injury, inhibit oxidative stress, and reduce inflammatory responses[4].

In vitro, treatment with Polydatin (10–40μM) in BRL-3A and HepG2 cells significantly inhibited fructose-induced oxidative stress, inflammation, and lipid accumulation. Polydatin upregulated miR-200a expression, which targeted and inhibited Keap1, thereby activating the Nrf2 antioxidant pathway. This led to a reduction in reactive oxygen species (ROS) levels and decreased expression of thioredoxin-interacting protein (TXNIP) and NLRP3 inflammasome-related proteins (NLRP3, ASC, Caspase-1, IL-1β). Additionally, Polydatin restored the expression of lipid metabolism-related proteins (PPAR-α, CPT-1) and inhibited the expression of SREBP-1 and SCD-1, thereby reducing the

accumulation of triglycerides (TG) and total cholesterol (TC)[5]. Polydatin (0–120μM) treatment in human lung epithelial cell line BEAS-2B and lung cancer cell line A549 significantly inhibited cell proliferation in a concentration- and time-dependent manner. Pretreatment with Polydatin also reduced the survival rate of A549 cells exposed to radiation (8Gy) [6].

In vivo, Polydatin (50mg/kg) was administered via intraperitoneal injection to BALB/c nude mice bearing H460 non-small cell lung cancer (NSCLC) xenografts. The treatment was given every two days for two weeks. Polydatin alone significantly inhibited tumor growth, and when combined with cisplatin (2mg/kg; i.p.; every 3 days), Polydatin synergistically enhanced the antitumor effect, markedly reducing tumor volume and weight. Furthermore, Polydatin promoted the expression of NADPH oxidase 5 (NOX5), increased ROS production, and activated endoplasmic reticulum (ER) stress and the c-Jun N-terminal kinase (JNK) and p38 mitogen-activated protein kinase (MAPK) signaling pathways[7]. Polydatin (5mg/kg), administered via intraperitoneal injection, was used to treat male C57BL/6 mice with nonalcoholic steatohepatitis (NASH) and liver fibrosis induced by a methionine-choline-deficient (MCD) diet. The treatment was given throughout the 4-week dietary induction period. At this dose, Polydatin significantly alleviated hepatic steatosis, hepatocyte injury, and inflammation, reduced serum ALT and AST levels, decreased hepatocyte apoptosis and oxidative stress, and inhibited the progression of liver fibrosis. Moreover, Polydatin suppressed the TLR4/NF-κB inflammatory signaling pathway and reduced the expression of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6[8].

References:
[1] Karami A, Fakhri S, Kooshki L, et al. Therapeutic Targets, Biological Activities, and Health Benefits. Molecules. 2022 Oct 1;27(19):6474.
[2] Luo J, Chen S, Wang L, et al. Pharmacological effects of polydatin in the treatment of metabolic diseases: A review. Phytomedicine. 2022 Jul 20;102:154161.
[3] Tang D, Zhang Q, Duan H, et al. Polydatin: A Critical Promising Natural Agent for Liver Protection via Antioxidative Stress. Oxid Med Cell Longev. 2022 Feb 10;2022:9218738.
[4] Tang KS. Protective Effects of Polydatin Against Dementia-Related Disorders. Curr Neuropharmacol. 2021;19(2):127-135.
[5] Zhao XJ, Yu HW, Yang YZ, et al. Polydatin prevents fructose-induced liver inflammation and lipid deposition through increasing miR-200a to regulate Keap1/Nrf2 pathway. Redox Biol. 2018 Sep;18:124-137.
[6] Guo J, Ding W, Cai S, et al. Polydatin radiosensitizes lung cancer while preventing radiation

injuries by modulating tumor-infiltrating B cells. J Cancer Res Clin Oncol. 2023 Sep;149(12):9529-9542.
[7] Wu S, Zhao Q, Liu S, Vanbever R, et al. Polydatin, a potential NOX5 agonist, synergistically enhances antitumor activity of cisplatin by stimulating oxidative stress in non-small cell lung cancer. Int J Oncol. 2024 Aug;65(2):77.
[8] Li R, Li J, Huang Y, et al. Polydatin attenuates diet-induced nonalcoholic steatohepatitis and fibrosis in mice. Int J Biol Sci. 2018 Jul 30;14(11):1411-1425.