

Product Data Sheet

Product Name: Pyrogallol

Cat. No.: GC32391

Chemical Properties

Cas. No. 87-66-1

SMILES OC1=CC=CC(O)=C1O

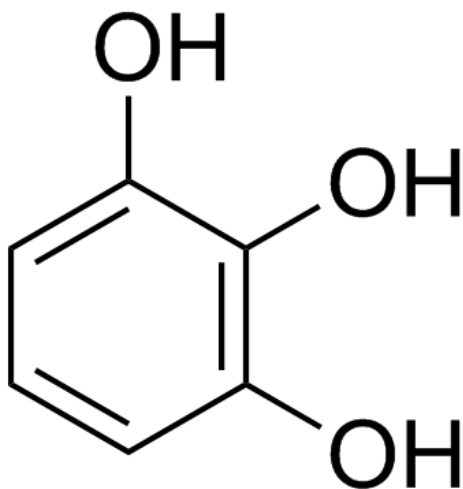
Formula $C_6H_6O_3$ M.Wt 126.11

Solubility DMSO : ≥ 100 mg/mL (792.96 mM); Water : 50 mg/mL (396.48 mM) Storage Store at RT

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: Briefly, 5×10^3 HPF cells per well in 96-well microtiter plates are exposed to 0, 50 or 100 μ M Pyrogallol with or without each MAPK inhibitor at 37°C for 24 h. Changes in cell viability induced by Pyrogallol and/or a given MAPK inhibitor are determined by measuring the MTT; dye absorbance[1].

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

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References:

[1]. Han BR, et al. MAPK inhibitors enhance cell death in pyrogallol-treated human pulmonary fibroblast cells via increasing O₂^{•-} levels. *Oncol Lett.* 2017 Jul;14(1):1179-1185.

Background

Pyrogallol is a polyphenol compound, which has anti-fungal and anti-psoriatic properties. Pyrogallol is a reductant that is able to generate free radicals, in particular superoxide anions.

Pyrogallol (PG) is a reductant that is able to generate free radicals, in particular superoxide anions (O₂^{•-}), so has frequently been used as a photographic developing agent and in the hair dying industry. Pyrogallol inhibits Calu-6 and A549 lung cancer cell growth via apoptosis and depletion of glutathione (GSH). Pyrogallol (PG) induces apoptosis in lung cancer cells via the overproduction of O₂^{•-} and affects mitogen activated protein kinases (MAPKs) in these cells[1]. The effect of Pyrogallol on human pulmonary fibroblast (HPF) cell viability and necrotic cell death is examined. For these experiments, 0, 50 or 100 µM Pyrogallol is used to differentiate the levels of cell viability inhibition or death with or without a given MAPK inhibitor. Treatment with 50 and 100 µM Pyrogallol decreases HPF viability by ~40 and 65% at 24 h, respectively. Treatment with an MEK inhibitor slightly enhances the inhibition of cell viability in 50 µM Pyrogallol-treated HPF cells, whereas treatment with a p38 inhibitor mildly attenuates the inhibition of viability. In 100 µM Pyrogallol-treated HPF cells, all the MAPK inhibitors increase the inhibition of viability to a certain extent, with treatment with the p38 inhibitor alone augmenting HPF control cell viability. Necrotic cell death is determined by measuring lactate dehydrogenase (LDH) release from cells. While treatment with 50 µM Pyrogallol does not affect LDH release from HPF cells, 100 µM Pyrogallol significantly increases LDH release[1].

[1]. Han BR, et al. MAPK inhibitors enhance cell death in pyrogallol-treated human pulmonary fibroblast cells via increasing O₂^{•-} levels. *Oncol Lett.* 2017 Jul;14(1):1179-1185.

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