

Product Data Sheet

Product Name: Phosphoenolpyruvic acid

Cat. No.: GB60458

Chemical Properties

Cas. No. 138-08-9

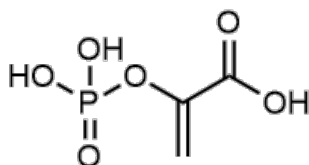
Formula $C_3H_5O_6P$ M.Wt 168.04

Solubility PBS (pH 7.2): 10 mg/ml Storage $-20^{\circ}C$

General tips For obtaining a higher solubility, please warm the tube at $37^{\circ}C$ and shake it in the ultrasonic bath for a while. Stock solution can be stored below $-20^{\circ}C$ 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 HeLa cells (human epithelial immortal cell line)

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Preparation Method HeLa cells were maintained in Minimum Essential Medium (MEM) supplemented with 10% fetal bovine serum (FBS), 100IU/mL penicillin, and 100µg/mL streptomycin at 37°C, 5% CO₂. Confluent cultures were washed with phosphate-buffered saline, detached with EDTA and trypsin, centrifuged, and sub-cultured in 96-well plates or 60mm dishes. Cells were seeded and grown on culture dishes for 24h before treatment. HeLa cells were then treated with Phosphoenolpyruvic acid at 0.1–10mM either alone or co-administered with hydrogen peroxide (H₂O₂; 0.1mM) for 24 hours.

Reaction Conditions 0.1–10mM; 24h

Applications Phosphoenolpyruvic acid significantly reduced the H₂O₂-induced drop in cell viability in a dose-dependent manner, prevented the decrease in Calcein-AM positive live cells and the increase in Propidium Iodide positive dead cells caused by H₂O₂, and significantly lowered the H₂O₂-stimulated rise in intracellular reactive oxygen species.

Animal experiment [2]:

Animal models Male Wistar rats

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After fasting overnight with free access to water, rats were anesthetized by light ether inhalation; a catheter was placed into the ileocecal vein and positioned just distal to the portal vein for post-ischemia infusion. Whole-liver warm ischemia was induced by occluding the portal vein, hepatic artery, and bile duct Preparation Method with a microvascular clamp: two 15-min ischemia periods separated by a 2-min reperfusion interval. Immediately after completing the second ischemic period, rats received a 5-min intra-portal-vein infusion of test solution (infusion started at reperfusion): either Phosphoenolpyruvic acid (400mM) or a glucose solution (400mM) as control, delivered at 2.5mL/300g over 5min.

Dosage form 400mM; 2.5mL/300g; portal-vein infusion; infused over 5min

Applications Phosphoenolpyruvic acid significantly increased ATP levels in liver tissue, significantly decreased AMP levels, and improved liver energy charge.

References:

- [1] Saiki S, Yamaguchi K, Chijiwa K, et al. Phosphoenolpyruvate prevents the decline in hepatic ATP and energy charge after ischemia and reperfusion injury in rats. J Surg Res. 1997 Nov;73(1):59-65.
- [2] Shi C, Feng S, Liu T, et al. Diabetes Mellitus Facilitates Gallstone Formation Through CXCR2-NETs-Mediated Liver-Bile Barrier Damage. Adv Sci (Weinh). 2026 Apr;13(24):e19500.

Background

Phosphoenolpyruvic acid is a high-energy phosphorylated α -keto acid intermediate of the glycolytic pathway, participates in glycolysis and gluconeogenesis, and serves as an energy source for ATP generation under energy-limited conditions. Phosphoenolpyruvic acid can donate its very high-transfer-potential phosphoryl group to ADP to produce ATP and pyruvate, a reaction catalyzed by pyruvate kinase (PK). Phosphoenolpyruvic acid can be used in studies related to glycolysis/gluconeogenesis regulation, cellular metabolic reprogramming, and tumor energy metabolism^[1-4].

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In vitro, LLC-PK₁ cells (porcine proximal tubular epithelial cell line) were treated with Phosphoenolpyruvic acid (0.5–10mM) in the presence of hydrogen peroxide (H₂O₂; 0.1mM) for 24h. Phosphoenolpyruvic acid dose-dependently and significantly attenuated H₂O₂-induced cellular injury and the decrease in cell viability, and significantly enhanced cellular decomposition/clearance of H₂O₂[5]. HeLa cells were treated with Phosphoenolpyruvic acid (0.1–10mM) in the presence of H₂O₂ (0.1mM) for 24h. Phosphoenolpyruvic acid dose-dependently and significantly attenuated the H₂O₂-induced loss of cell viability, inhibited the H₂O₂-mediated reduction of Calcein-AM-positive viable cells and the increase in propidium iodide-positive non-viable cells, and significantly suppressed the H₂O₂-stimulated rise in intracellular reactive oxygen species[6].

In vivo, after completion of two episodes of 15-min whole-liver warm ischemia (occlusion of the portal vein, hepatic artery, and bile duct; separated by a 2-min reperfusion interval), Phosphoenolpyruvic acid (400mM; 2.5mL/300g; infused over 5min) was administered via an ileocecal/portal venous catheter immediately upon reperfusion in Wistar rats. Phosphoenolpyruvic acid significantly increased hepatic ATP concentration, significantly lowered hepatic AMP concentration, and significantly improved the hepatic energy charge[7]. In a myocardial infarction-related arrhythmia model, immediately before left anterior descending coronary artery (LAD) occlusion, Phosphoenolpyruvic acid (0.10mg/kg or 0.50mg/kg; single bolus) was administered intravenously in Wistar rats. Phosphoenolpyruvic acid (0.50mg/kg) significantly prolonged ventricular arrhythmia latency, shortened ventricular tachycardia duration, and reduced the number of premature ventricular beats[8].

References:

- [1] Forest C, Tordjman J, Glorian M, et al. Fatty acid recycling in adipocytes: a role for glyceroneogenesis and phosphoenolpyruvate carboxykinase. *Biochem Soc Trans.* 2003 Dec;31(Pt 6):1125-9.
- [2] Hanson RW, Reshef L. Glyceroneogenesis revisited. *Biochimie.* 2003 Dec;85(12):1199-205.
- [3] Tannen RL. Ammonia metabolism. *Am J Physiol.* 1978 Oct;235(4):F265-77.
- [4] Nakamura MT, Yudell BE, Loor JJ. Regulation of energy metabolism by long-chain fatty acids. *Prog Lipid Res.* 2014 Jan;53:124-44.
- [5] Yuki Kondo, Yoichi Ishitsuka, Daisuke Kadowaki, et al. Phosphoenolpyruvic Acid, an Intermediate of Glycolysis, Attenuates Cellular Injury Induced by Hydrogen Peroxide and 2-Deoxy-D-glucose in the Porcine Proximal Kidney Tubular Cell Line, LLC-PK1. *Journal of Health Science.* 2010 Dec 56(6):727-732.

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[6] Kondo Y, Ishitsuka Y, Kadowaki D, et al. Phosphoenolpyruvic acid, an intermediary metabolite of glycolysis, as a potential cytoprotectant and anti-oxidant in HeLa cells. Biol Pharm Bull. 2012;35(4):606-11.

[7] Saiki S, Yamaguchi K, Chijiwa K, et al. Phosphoenolpyruvate prevents the decline in hepatic ATP and energy charge after ischemia and reperfusion injury in rats. J Surg Res. 1997 Nov;73(1):59-65.

[8] Balashov VP, Kostin IaV, Sedova DG, et al. The effect of fructose-1,6-diphosphate and phosphoenolpyruvate on the course of early occlusion and reperfusion arrhythmias in rats. Biull Eksp Biol Med. 1993 Jun;115(6):629-30.

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