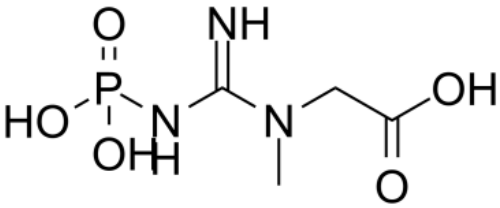
Product Data Sheet

Product Name: Phosphocreatine
Cat. No.: GC36898

Chemical Properties

Cas. No.	67-07-2		
SMILES	O=C(O)CN(C(NP(O)(O)=O)=N)C		
Formula	$C_4H_{10}N_3O_5P$	M.Wt	211.11
Solubility	Water : 100mg/mL	Storage	Store at -20°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



Product Data Sheet

Protocol

Cell experiment [1]:

Cell lines	Human umbilical vein endothelial cells (HUVECs)
Preparation Method	HUVECs were incubated in DMEM medium supplemented with 10% fetal calf serum (FCS) in a humidified atmosphere at 37°C with 5% CO_2. Cells were plated at a density of 2×10^4/well in a 96-well tissue culture plate for 24h and preincubated with various concentrations of Phosphocreatine (0, 10, 20, 30mM) for 2h, then cells were stimulated with 200μg/ml oxLDL for 24h in the presence of Phosphocreatine. The cell viability of HUVECs was determined.
Reaction Conditions	0, 10, 20, 30mM; preincubated for 2h
Applications	Phosphocreatine treatment significantly reduced oxLDL-induced death of HUVECs in a dose-dependent manner.

Animal experiment [2]:

Animal models	Sprague-Dawley rats
Preparation Method	Male Sprague-Dawley rats (180-200g) were housed in controlled conditions (22±2°C, 50-70% humidity, 12-hour light/dark cycle) with unlimited access to standard commercial feed and water. Rats were acclimatized for one week before the investigation. Diabetes was induced through a single intraperitoneal injection of Streptozotocin at a dose of 50mg/kg. Hyperglycemia was confirmed when fasting blood glucose exceeded 250mg/dl. Kidney fibrosis was induced through unilateral ureteral obstruction (UUO) performed under anesthesia. Diabetic fibrotic rats treated with Phosphocreatine (20mg/kg/day; intraperitoneally) for 8 weeks, starting one week after UUO. Collect the renal tissues of rats for analysis.

Caution: Product has not been fully validated for medical applications. For research use only.
Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA

Product Data Sheet

Dosage form	20mg/kg/day for 8 weeks; i.p.
Applications	Phosphocreatine treatment significantly improved the renal function of the diabetic-nephropathy fibrosis rat models.

References:
[1] Ahsan A, Han G, Pan J, et al. Phosphocreatine protects endothelial cells from oxidized low-density lipoprotein-induced apoptosis by modulating the PI3K/Akt/eNOS pathway[J]. Apoptosis, 2015, 20(12): 1563-1576.
[2] Wang F H, Alwesabi A K, Liu W, et al. Phosphocreatine Attenuates Diabetes-Exacerbated Kidney Fibrosis via TGF-β/Smad and PI3K/Akt Pathways in a Dual Rat Model[J]. Tissue and Cell, 2026: 103439.

Background

Phosphocreatine is a high-energy phosphate compound that is abundant in muscle and brain [1]. Under non-steady-state conditions, Phosphocreatine maintains the ATP homeostasis at specific high-energy turnover sites (such as muscle fibers), especially in rapidly contracting skeletal muscles, and prevents a significant increase in the intracellular free ADP concentration, thereby minimizing the loss of intracellular adenosine nucleotides [2]. Phosphocreatine has been widely used to alter lipid phase transitions, influencing the aggregation of membranes and protecting cell membranes from various damages[3].

In vitro, Phosphocreatine treatment (30mM) for 2 hours significantly inhibited the apoptosis of human umbilical vein endothelial cells (HUVECs) induced by oxidized low-density lipoprotein (oxLDL), and reduced the loss of mitochondrial membrane permeability [4]. Phosphocreatine (5mM) treatment for 4 hours significantly inhibited the increase in intracellular reactive oxygen species (ROS) in rat striatal slices induced by 6-hydroxydopamine, restored the level of tyrosine hydroxylase (TH), and reduced cell death[5]. 500μM Phosphocreatine pretreatment for 1 hour significantly inhibited doxorubicin (1μM; 24h)-induced necrotic cell death in H9c2 cells, accompanied by a decrease in the expression of RIP3 and CaMKII[6].

Product Data Sheet

In vivo, Phosphocreatine treatment via intraperitoneal injection at a dose of 20mg/kg/day for 8 weeks significantly improved the renal function of the diabetic-nephropathy fibrosis rat model, inhibited renal inflammation, and restored mitochondrial function[7]. Intraperitoneal injection of 40mg/kg/day dose of Phosphocreatine for 6 weeks significantly alleviated renal damage in the diabetic rat model and lowered blood glucose levels[8].

References:

[1] Chen L, Schär M, Chan K W Y, et al. In vivo imaging of phosphocreatine with artificial neural networks[J]. Nature communications, 2020, 11(1): 1072.
[2] Greenhaff P L. The creatine-phosphocreatine system: there's more than one song in its repertoire[J]. The Journal of physiology, 2001, 537(Pt 3): 657.
[3] Tokarska-Schlattner M, Epand R F, Meiler F, et al. Phosphocreatine interacts with phospholipids, affects membrane properties and exerts membrane-protective effects[J]. 2012.
[4] Ahsan A, Han G, Pan J, et al. Phosphocreatine protects endothelial cells from oxidized low-density lipoprotein-induced apoptosis by modulating the PI3K/Akt/eNOS pathway[J]. Apoptosis, 2015, 20(12): 1563-1576.
[5] Cunha M P, Martín-de-Saavedra M D, Romero A, et al. Both creatine and its product phosphocreatine reduce oxidative stress and afford neuroprotection in an in vitro Parkinson's model[J]. ASN neuro, 2014, 6(6): 1759091414554945.
[6] Wang C, Hu L, Guo S, et al. Phosphocreatine attenuates doxorubicin-induced cardiotoxicity by inhibiting oxidative stress and activating TAK1 to promote myocardial survival in vivo and in vitro[J]. Toxicology, 2021, 460: 152881.
[7] Wang F H, Alwesabi A K, Liu W, et al. Phosphocreatine Attenuates Diabetes-Exacerbated Kidney Fibrosis via TGF-β/Smad and PI3K/Akt Pathways in a Dual Rat Model[J]. Tissue and Cell, 2026: 103439.
[8] Shopit A, Niu M, Wang H, et al. Protection of diabetes-induced kidney injury by phosphocreatine via the regulation of ERK/Nrf2/HO-1 signaling pathway[J]. Life sciences, 2020, 242: 117248.

Caution: Product has not been fully validated for medical applications. For research use only.
Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA