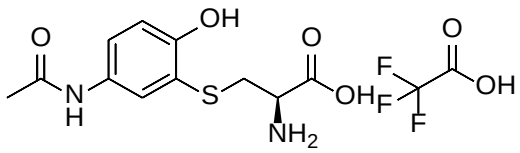
Product data sheet

MedKoo Cat#: 574298 Name: PQN91930 CAS: 1331891-93-0 Chemical Formula: $C_{13}H_{15}F_3N_2O_6S$ Exact Mass: 384.0603 Molecular Weight: 384.3262	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:
PQN91930, also known as 3-Cysteinylacetaminophen TFA salt and APAP-Cys, is an acetaminophen-protein adduct formed during the metabolism of acetaminophen. In mice, 3-cysteinylacetaminophen decreases renal glutathione (GSH) levels --- an effect that can be blocked by the γ-glutamyl inhibitor acivicin. This product has no formal name at the moment. For the convenience of communication, a temporary code name was therefore proposed according to MedKoo Chemical Nomenclature (see web page: https://www.medkoo.com/page/naming).

2. CoA, QC data, SDS, and handling instruction
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	0.17	0.44

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.60 mL	13.01 mL	26.02 mL
5 mM	0.52 mL	2.60 mL	5.20 mL
10 mM	0.26 mL	1.30 mL	2.60 mL
50 mM	0.05 mL	0.26 mL	0.52 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator
Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study
In vitro study

1. Koenderink JB, van den Heuvel JJMW, Bilos A, Vredenburg G, Vermeulen NPE, Russel FGM. Human multidrug resistance protein 4 (MRP4) is a cellular efflux transporter for paracetamol glutathione and cysteine conjugates. Arch Toxicol. 2020 Sep;94(9):3027-3032. doi: 10.1007/s00204-020-02793-4. Epub 2020 May 29. PMID: 32472168; PMCID: PMC7415487.

In vivo study

1. Wei M, Gu X, Li H, Zheng Z, Qiu Z, Sheng Y, Lu B, Wang Z, Ji L. EGR1 is crucial for the chlorogenic acid-provided promotion on liver regeneration and repair after APAP-induced liver injury. Cell Biol Toxicol. 2023 Feb 21. doi: 10.1007/s10565-023-09795-9. Epub ahead of print. PMID: 36809385.
2. Stern ST, Bruno MK, Horton RA, Hill DW, Roberts JC, Cohen SD. Contribution of acetaminophen-cysteine to acetaminophen nephrotoxicity II. Possible involvement of the gamma-glutamyl cycle. Toxicol Appl Pharmacol. 2005 Jan 15;202(2):160-71. doi: 10.1016/j.taap.2004.06.029. PMID: 15629191.

7. Bioactivity
Biological target:

Product data sheet

PQN91930, also known as 3-Cysteinylacetaminophen TFA salt is an acetaminophen-protein adduct formed during the metabolism of acetaminophen.

In vitro activity

This study examined whether ATP-binding cassette (ABC) transporters play a role in the cellular efflux of APAP, APAP-GSH, and APAP-CYS. The ABC transport proteins P-gp/ABCB1, BSEP/ABCB11, BCRP/ABCG2, and MRP/ABCC1-5 were overexpressed in HEK293 cells and membrane vesicles were produced. Whereas P-gp, BSEP, MRP3, MRP5, and BCRP did not transport any of the compounds, uptake of APAP-GSH was found for MRP1, MRP2 and MRP4. APAP-CYS appeared to be a substrate of MRP4 and none of the ABC proteins transported APAP. The results suggest that the NAPQI metabolite APAP-CYS can be excreted into plasma by MRP4, where it could be a useful biomarker for APAP exposure and toxicity. Characterization of the cellular efflux of APAP-CYS is important for its development as a biomarker, because plasma concentrations might be influenced by drug-transporter interactions and upregulation of MRP4.

Reference: Arch Toxicol. 2020 Sep;94(9):3027-3032. https://pubmed.ncbi.nlm.nih.gov/32472168/

In vivo activity

Acetaminophen (APAP) nephrotoxicity has been observed both in humans and research animals. Recent investigations have focused on the possible involvement of glutathione-derived APAP metabolites in APAP nephrotoxicity and have demonstrated that administration of acetaminophen-cysteine (APAP-CYS) potentiated APAP-induced renal injury with no effects on APAP-induced liver injury. Additionally, APAP-CYS treatment alone resulted in a dose-responsive renal GSH depletion. This APAP-CYS-induced renal GSH depletion could interfere with intrarenal detoxification of APAP or its toxic metabolite N-acetyl-p-benzoquinoneimine (NAPQI) and may be the mechanism responsible for the potentiation of APAP nephrotoxicity.

Reference: Toxicol Appl Pharmacol. 2005 Jan 15;202(2):160-71. https://pubmed.ncbi.nlm.nih.gov/15629191/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.