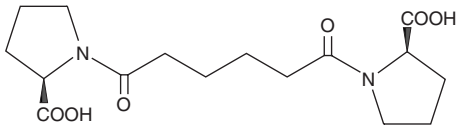
# PRODUCT INFORMATION

## CPHPC

*Item No. 75500*

**CAS Registry No.:** 224624-80-0
**Formal Name:** 1,1'-(1,6-dioxo-1,6-hexadiyl)*bis*-D-proline
**Synonym:** GSK2315698, Miridesap, Ro 63-8695
**MF:** $C_{16}H_{24}N_2O_6$
**FW:** 340.4
**Purity:** ≥95%
**Supplied as:** A crystalline solid
**Storage:** -20°C
**Stability:** ≥4 years




*Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis*

### Laboratory Procedures

CPHPC is supplied as a crystalline solid. A stock solution may be made by dissolving the CPHPC in the solvent of choice, which should be purged with an inert gas. CPHPC is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide. The solubility of CPHPC in these solvents is approximately 30, 20, and 25 mg/ml, respectively.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. Organic solvent-free aqueous solutions of CPHPC can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of CPHPC in PBS (pH 7.2) is approximately 10 mg/ml. We do not recommend storing the aqueous solution for more than one day.

### Description

CPHPC is an inhibitor of the protein-protein interaction between serum amyloid P (SAP) and amyloid fibrils ($IC_{50}$ = 0.9 µM).[1] It also crosslinks with pentameric SAP in the plasma to form a decameric complex, which is eliminated *via* hepatic clearance. As plasma and amyloid deposits of SAP exist in a dynamic equilibrium, CPHPC induces the dissociation of SAP from tissue amyloid deposits, leading to its redistribution into the plasma, where it is eliminated. DPHC (1 mg/ml in the drinking water) reduces serum human SAP levels in a transgenic mouse model of amyloidosis.

### Reference

1. Pepys, M.B., Herbert, J., Hutchinson, W.L., *et al*. Targeted pharmacological depletion of serum amyloid P component for treatment of human amyloidosis. *Nature* **417(6886)**, 254-259 (2002).



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