Small Molecules

Hydroxychloroquine

Inhibits autophagy and heme

polymerase

Catalog # 74212 74214

50 mg 100 mg



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Product Description

Hydroxychloroquine is a synthetic derivative of quinolyl. It increases the pH within intracellular vacuoles and alters processes such as protein degradation by acidic hydrolases, assembly of macromolecules, and post-translation modification of proteins (Fox). By doing this, it prevents the stimulation of CD4+ cells, thus down-regulating the immune response (Fox). Hydroxychloroquine is also a potent autophagy inhibitor (van Loosdregt et al.) and heme polymerase inhibitor (Chou & Fitch). This product is supplied as the sulfate salt of the molecule.

Molecular Name: Hydroxychloroquine (Sulfate)

Alternative Names: HCQ; NSC 4375

CAS Number: 747-36-4

Chemical Formula: $C_{18}H_{26}CIN_3O \bullet H_2SO_4$

Molecular Weight: 434.0 g/mol Purity: \geq 95%

Chemical Name: 2-({4-[(7-chloroquinolin-4-yl)amino]pentyl}(ethyl)amino)ethan-1-ol;sulfuric acid

Structure:

$$H$$
 N
 OH
 H_2SO_4

Properties

Physical Appearance: A crystalline solid

Storage: Product stable at -20°C as supplied. Protect product from prolonged exposure to light. For long-term storage,

store with a desiccant.

Stable as supplied for 12 months from date of receipt.

Solubility: $\cdot PBS (pH 7.2) \le 10 \text{ mM}$

For example, to prepare a 5 mM stock solution in PBS, resuspend 10 mg in 4.61 mL of PBS (pH 7.2).

Prepare stock solution fresh before use. Information regarding stability of small molecules in solution has rarely been reported, however, as a general guide we recommend storage in PBS at -20°C. Aliquot into working volumes to avoid repeated freeze-thaw cycles. The effect of storage of stock solution on compound performance should be tested for each application.

For use as a cell culture supplement, stock solution should be diluted into culture medium immediately before

use.

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Published Applications

IMMUNOLOGY

- · Inhibits the survival pathway of autophagy by preventing the degradation of autophagosomes, resulting in increased apoptosis of human CD4+CD45RO+ memory and effector T cells (van Loosdregt et al.).

 CANCER RESEARCH
- · In combination with various other treatments, increases the overall response rate, progression-free survival, and 1-year overall survival rate of cancer therapies (for glioblastoma, brain metastases due to non-small-cell lung cancer and breast cancer, pancreatic ductal adenocarcinoma [PDAC], non-Hodgkin lymphoma, metastatic PDAC, and pancreatic cancer), when compared to cancer therapies that do not utilize autophagy inhibition (Xu et al.).

References

Chou AC & Fitch CD. (1992) Heme polymerase: modulation by chloroquine treatment of a rodent malaria. Life Sci 51(26): 2073–8. Fox RI. (1993) Mechanism of action of hydroxychloroquine as an antirheumatic drug. Semin Arthritis Rheum 23(2 Suppl 1): 82–91. van Loosdregt J et al. (2013) Hydroxychloroquine preferentially induces apoptosis of CD45RO+ effector T cells by inhibiting autophagy: A possible mechanism for therapeutic modulation of T cells. J Allergy Clin Immunol 131(5): 1443–6.e1.

Xu R et al. (2018) The clinical value of using chloroquine or hydroxychloroquine as autophagy inhibitors in the treatment of cancers. Medicine (Baltimore) 97(46): e12912.

Related Small Molecules

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