AP39
Item No. 17100

CAS Registry No.: 1429173-57-8
Formal Name: [10-oxo-10-[4-(3-thioxo-3H-1,2-dithiol-5-yl)phenoxyl]decyl]triphenylphosphonium
MF: C37H38O2PS3
FW: 641.9
Purity: ≥95%
Stability: ≥1 year at -20°C
Supplied as: A solution in dichloromethane
UV/Vis: λmax: 226, 267, 319, 430 nm

Laboratory Procedures

For long term storage, we suggest that AP39 be stored as supplied at -20°C. It should be stable for at least one year. AP39 is supplied as a solution in dichloromethane. To change the solvent, simply evaporate the dichloromethane under a gentle stream of nitrogen and immediately add the solvent of choice. Solvents such as ethanol, DMSO, and dimethyl formamide purged with an inert gas can be used. The solubility of AP39 in these solvents is approximately 16, 20, and 5 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. If an organic solvent-free solution of AP39 is needed, it can be prepared by evaporating the dichloromethane and directly dissolving the neat oil in aqueous buffers. The solubility of AP39 in PBS, pH 7.2, is approximately 1 mg/ml. We do not recommend storing the aqueous solution for more than one day.

AP39 is a compound used to increase the levels of hydrogen sulfide (H2S) within mitochondria. It consists of a mitochondria-targeting motif (triphenylphosphonium) coupled to an H2S-donating moiety (dithiolethione) by an aliphatic linker. AP39 (30-300 nM) dose-dependently increases H2S levels in endothelial cells, predominantly in mitochondrial regions. It stimulates mitochondrial electron transport and improves cellular bioenergetic function at lower concentrations (30-100 nM), while having an inhibitory effect at 300 nM. Under oxidative stress conditions induced by glucose oxidase, AP39 has antioxidant and cytoprotective effects.

AP39 is effective in vivo, inhibiting voltage-dependent T-type calcium channels and improving hemodynamic parameters in rats.

References

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