www.neobiolab.com
info@neobiolab.com
888.754.5670, +1 617.500.7103 United States
0800.088.5164, +44 020.8123.1558 United Kingdom

Cyclosporin A

Catalog #:PRPS-415

For research use only.

Description: Cyclosporin is a cyclic polypeptide immunosuppressant agent consisting of 11 amino acids and having a molecular weight of 1202.64. It is produced as a metabolite by the fungus species Beauveria nlyea. Chemically, cyclosporin is designated as [R-[R*,R*-(E)]]-cyclic(L-alanyl-D-alanyl-N-methyl-L-leucyl-N-methyl-L-leucyl-N-methyl-L-valyl-3-hydroxy-N, 4-dimethyl-L-2-amino-6-octenoyl-L--amino-butyryl- N-methylglycyl-N-methyl-L-leucyl-L-valyl-N-methyl-L-leucyl). Molecular Formula: C62H111N11O12.

Source: Beauveria Nlyea.

Physical Appearance: Sterile Filtered White lyophilized (freeze-dried) powder.

Purity: Greater than 98.0% as determined by:(a) Analysis by RP-HPLC. (b) Mass Spectral Anaylsis {MALDI-TOF exhibits correct Mw}.

Formulation:

The Cyclosporin-A was lyophilized from a concentrated (1mg/ml) solution with no additives.

Stability:

Lyophilized Cyclosporin A althoμgh stable at room temperature for 3 weeks, should be stored desiccated below -18°C. Upon reconstitution Cyclosporin A should be stored at 4°C between 2-7 days and for future use below -18°C.Please prevent freeze-thaw cycles.

Usage:

NeoBiolab's products are furnished for LABORATORY RESEARCH USE ONLY. The product may not be used as drμgs, agricultural or pesticidal products, food additives or household chemicals.

Solubility:

It is recommended to reconstitute the lyophilized Cyclosporin-A in Ethanol.

Introduction:

Cyclosporin A is a noncytotoxic, natural, 11 amino acid cyclic peptide used clinically as an immunosuppressant for the treatment of autoimmune and inflammatory disorders and to prevent organ rejection after transplantation. Cyclosporin acts chiefly by inhibiting T lymphocyte function, which is vital for the propagation of inflammation. Cyclosporin A does not suppress the activity of other hematopoietic cells, does not cause bone marrow suppression and has a rapid onset of action as opposed to other immunosuppressive agents. Nevertheless, Cyclosporin A -induced nephrotoxicity remains an important clinical problem, and oxidative stress has been implicated as a potential responsible mechanism.

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