ATAGENIX LABORATORIES

Catalog Number:ATEP00497HU

Recombinant Human C5 protein ,No Tag

Product Details

Summary

English name	Recombinant Human C5 protein ,No Tag
Purity	>90% as determined by SDS-PAGE
Endotoxin level	Please contact with the lab for this information.
Construction	A DNA sequence encoding the human C5(Leu 679-Arg 751) was fused without Tag
Accession #	P01031
Host	E.coli
Species	Homo sapiens (Human)
Predicted Molecular Mass	8.14kDa
Formulation	Supplied as solution form in PBS or lyophilized from PBS .
Shipping	In general, proteins are provided as lyophilized powder/frozen liquid. They are shipped out with dry ice/blue ice unless customers require otherwise.
Stability &Storage	Use a manual defrost freezer and avoid repeated freeze thaw cycles. Store at 2 to 8 °C for one week . Store at -20 to -80 °C for twelve months from the date of receipt.
Reconstitution	Reconstitute in sterile water for a stock solution.A copy of datasheet will be provided with the products, please refer to it for details.

Background

Background	Derived from proteolytic degradation of complement C5, C5 anaphylatoxin is a mediator of local inflammatory process. C5 precursor is first processed by the removal of 4 basic residues, forming two chains, beta and alpha, linked by a disulfide bond. C5 convertase activates C5 by cleaving the alpha chain, releasing C5a anaphylatoxin and generating C5b (beta chain + alpha' chain). Activation of C5 by a C5 convertase initiates the spontaneous assembly of the late complement components, C5-C9, into the membrane attack complex. C5b has a transient binding site for C6. The C5b-C6 complex is the foundation upon which the lytic complex is assembled. The C5a anaphylatoxin interacts with C5AR1 and tick complement inhibitor. C5a is also a potent chemokine which stimulates the

	locomotion of polymorphonuclear leukocytes and directs their migration toward sites of inflammation.
Alternative Names	Complement C5,C5,CPAMD4
References	Menon, Barnett, Bril (2020) Novel Treatments in Myasthenia Gravis Frontiers in neurology 11() 538

Frontier progress

Myasthenia gravis (MG) is the prototypical autoimmune disorder caused by specific autoantibodies at the neuromuscular junction. Broad-based immunotherapies, such as corticosteroids, azathioprine, mycophenolate, tacrolimus, and cyclosporine, have been effective in controlling symptoms of myasthenia. While being effective in a majority of MG patients many of these immunosuppressive agents are associated with long-term side effects, often intolerable for patients, and take several months to be effective. With advances in translational research and drug development capabilities, more directed therapeutic agents that can alter the future of MG treatment have been developed. This review focuses on the aberrant immunological processes in MG, the novel agents that target them along with the clinical evidence for efficacy and safety. These agents include terminal complement C5 inhibitors, Fc receptor inhibitors, B cell depleting agents (anti CD 19 and 20 and B cell activating factor [BAFF)] inhibitors), proteosome inhibitors, T cells and cytokine based therapies (chimeric antigen receptor T [CART-T] cell therapy), autologous stem cell transplantation, and subcutaneous immunoglobulin (SCIG). Most of these new agents have advantages over conventional immunosuppressive treatment (IST) for MG therapy in terms of faster onset of action, favourable side effect profile and the potential for a sustained and long-term remission.