General Information

Gene Name Synonym:
C5a; C5b; C5D; C5Da; Complement 5a; CPAMD4; ECLZB

Protein Construction:
A DNA sequence encoding the human C5 (NP_001726.2) (Gln19-Cys1676) was expressed with a polyhistidine tag at the C-terminus and a FLAG tag at the N-terminus.

Source: Human

Expression Host: HEK293 Cells

QC Testing

Purity: > 95 as determined by SDS-PAGE.

Endotoxin:
< 1.0 EU per μg protein as determined by the LAL method.

Stability:
Samples are stable for up to twelve months from date of receipt at -70 °C.

Predicted N terminal: Asp

Molecular Mass:
The recombinant human C5 consists of 1677 amino acids and predicts a molecular mass of 188.8 kDa.

Formulation:
Lyophilized from sterile PBS, pH 7.4.

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

Usage Guide

Storage:
Store it under sterile conditions at -20°C to -80°C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:
Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:

Protein Description

C5a is a protein fragment released from complement component C5. This 74 amino acid peptide in humans is generated by the cleavage of C5a convertase on the C5 α-chain during the classical, alternative, and lectin pathways of complement activation. The structure of C5a includes a core region consisting of four, anti-parallel alpha-helices held together by three disulfide linkages and a structured C-terminal tail, and C5a is rapidly metabolised by carboxypeptidase B to a 73 amino acid low activity form, C5a des-Arg. C5a is an extremely potent proinflammatory mediator, as well as a potent chemotactic factor for neutrophils and other leukocytes. It causes histamine release, increases in vascular permeability, induces several cytokines production from leukocytes, enhances neutrophil-endothelial cell adhesion, and augments the humoral and cell-mediated immune response. C5a is quickly metabolised by carboxypeptidases, forming the less potent C5adesArg. Acting via a classical G protein-coupled receptor, CD88, C5a and C5adesArg exert a number of effects essential to the innate immune response, while their actions at the more recently discovered non-G protein-coupled receptor, C5L2 (or GPR77), remain unclear. The widespread expression of C5a receptors throughout the body allows C5a to elicit a broad range of effects. Thus, C5a has been found to be a significant pathogenic driver in a number of immuno-inflammatory diseases, making C5a inhibition an attractive therapeutic strategy. C5a is a strong chemotactant and is involved in the recruitment of inflammatory cells such as neutrophils, eosinophils, monocytes, and T lymphocytes, in activation of phagocytic cells and release of granule-based enzymes and generation of oxidants, all of which may contribute to innate immune functions or tissue damage. Accordingly, the anaphylatoxin C5a is implicated in a variety of diseases such as rheumatoid arthritis, systemic lupus erythematosus, reperfusion injury, Alzheimer's disease, and sepsis.

References