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Anti-human LT alpha (LT-α) Monoclonal Antibody

Catalog No.: YR0370

Basic Information

Molecular Weight
150kDa

Endotoxin
<1EU/mg (<0.001EU/μg)Determined by LAL gel clotting assay

Sterility
0.2 μm filtration

Aggregation
<5% Determined by SECP

Purity
>95% Determined by SDS-PAGE

Reported Applications

ELISA,neutralization,functional assays such as bioanalytical P K and ADA assays,and those assays for studying biological pathways

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Background

Pateclizumab Biosimilar uses the same protein sequences as the therapeutic antibody pateclizumab. Pateclizumab is an immunoglobulin G1 kappa (IgG1κ) monoclonal antibody targeting lymphotoxin alpha (LT-α) for the treatment of rheumatoid arthritis. A phase I study has assessed the safety, pharmacokinetics, and biologic activity of pateclizumab, and found that pateclizumab was generally well-tolerated in RA patients. Pateclizumab also has been investigated in clinical trial to study its efficacy and safety in combination with a disease-modifying anti-rheumatic drug (DMARD) compared with adalimumab in combination with a DMARD in patients with active rheumatoid arthritis. LT-α is a member of tumor necrosis factor superfamily family (TNFSF) and products by predominately by activated cells of the innate and adaptive immune response. Lymphotoxin α formerly named tumor necrosis factor-beta (TNF-β) as it is a homologous protein to TNFα. When LTβ is discovered, TNF-β was renamed LT-α. LT-α plays different roles in immune regulation as its different secreted forms. LTα binds to TNF receptor 1 (TNFR1) and TNFR2 to promote inflammation as a form of soluble homotrimeric molecule (LTα3); whereas cell-bound LTα1β2 (LT-α complex with LTβ as LTα1β2 heterotrimers on the surface of activated B, Th1 and Th17 cells) bind LTβ receptors (LTβR) to mediate signaling pathway. Rheumatoid arthritis (RA) is an autoimmune disorder associated with progressive joint damage, pain, fatigue, and disability. TNF α is reported to be the main factor promoting the development of RA, so targeting TNFα is regarded as the routine method of RA treatment. However, a large number of RA patients did not respond to TNFα therapy, which prompted us to seek new treatments. In addition to TNFα, other cytokines have also been reported to be involved in the pathogenesis of RA, and LT- α is one of them. It was found that two forms of LTα homotrimer (LTα3 and LTα1β2) increased in synovial fluid of RA patients, while the LTα, LTβ and LTβR transcripts increased in synovium respectively. Study has demonstrated that the depletion of CD4 T helper (Th) subsets Th1 and Th17 (with high levels of surface LTα1β2) by mouse LTα specific monoclonal antibody showed therapeutic efficacy in the preclinical mouse model of RA, which suggests the treatment possibility targeting LTα. Thus, humanized pateclizumab was designed to target LTα, binding to both the soluble LTα3 homotrimeric form and the surface-expressed LTα1β2 heterotrimer, for the treatement of RA. By blocking the binding of LTα3 and LTα1β2 to its cognate receptors LTβR and TNFR, pateclizumab specificly deplete of activated cells and inhibit the immune cell trafficking and/or recruitment to inflammatory sites. Depletion is limited to cells that express LTα1β2 on the surface, which improves the targeting of therapy.

Immunogen Information

Clone
Pateclizumab Biosimilar

Isotype
Human IgG1 kappa

Immunogen
Human LT alpha (LT-α)

RecommendedIsotype Control(s)
In Vivo Grade Recombinant Human IgG1 Kappa Isotype Control Antibody

Recommended Dilution Buffer
1×PBS pH 7.4

Product Information

Production
Purified from cell culture supernatant in an animal-free facility

Purification
Protein A/G

Storage
Store at -20℃