

# Eculizumab Biosimilar, Human C5 Monoclonal Antibody

Catalog No.: YR0002

## Basic Information

### Molecular Weight

150 kDa

### Endotoxin

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

### Sterility

0.2 μm filtration

### Aggregation

<5% Determined by SECP

### Purity

89.8% Determined by SDS-PAGE

## Reported Applications

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

## Contact

☎ | 400-999-6126

✉ | [cn.market@abclonal.com.cn](mailto:cn.market@abclonal.com.cn)

🌐 | [www.abclonal.com.cn](http://www.abclonal.com.cn)

## Background

Eculizumab, a recombinant humanized anti-C5 (the terminal Complement component 5) monoclonal antibody, selectively targets and inhibits the terminal portion of the complement cascade. Eculizumab is a first-in-class terminal complement inhibitor to treat paroxysmal nocturnal hemoglobinuria (PNH) with excessive destruction of red blood cells (hemolysis). Eculizumab is also the first agent to treat atypical hemolytic uremic syndrome (aHUS) with abnormal blood clots to form in small blood vessels throughout the body, leading to kidney failure, damage to other vital organs and premature death. The complement immune system destroys and removes foreign particles by the complement cascade triggered by foreign particles. The complement proteins activated in order create holes or pores in the invading organisms, leading to their destruction. The complement immune system in patients can also destroy healthy cells and tissue, resulting in excessive destruction of red blood cells (hemolysis) or abnormal blood clots to form in small blood vessels throughout the body. When activated, C5 at a late stage in the complement cascade is involved in activating host cells, thereby attracting pro-inflammatory immune cells, while also destroying cells by triggering pore formation. Eculizumab specifically binds to C5 and inhibits the cleavage of C5 to C5a (a potent anaphylatoxin with prothrombotic and proinflammatory properties) and C5b by the C5 convertase, preventing the generation of the terminal complement complex C5b-9 (which also has prothrombotic and proinflammatory effects). Both C5a and C5b-9 cause the terminal complement-mediated events that are characteristic of PNH and aHUS. By doing so, the normal, disease-preventing functions of proximal complement system are largely preserved, while the properties of C5 that promote inflammation and cell destruction are impeded.

## Immunogen Information

### Clone

Eculizumab

### Isotype

Human IgG2/4 kappa

### Immunogen

Human C5

### Recommended Isotype Control(s)

In Vivo Grade Recombinant Human IgG4-S228P Kappa Isotype Control Antibody

### Recommended Dilution Buffer

1×PBS pH 7.0

## Product Information

### Production

Purified from cell culture supernatant in an animal-free facility

### Purification

Protein A or G purification

### Storage

2 - 8°C for up to 4 weeks and -80°C for long term storage (Avoid repeated freezing and thawing)