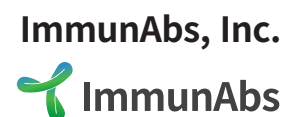


# IM-101: anti-C5 monoclonal antibody for myasthenia gravis



| NEUROLOGY                | Phase 1  |
|--------------------------|--|
| Product Type             | Antibody   |
| Indication               | Myasthenia Gravis  |
| Target                   | Human Complement 5(C5)   |
| MoA(Mechanism of Action) | IM-101 binds to complement C5, a gate keeper protein of the complement system, to prevent its proteolytic breakdown into C5a and C5b. C5a then becomes anaphylatoxin that induces inflammation, while C5b mediates the formation of membrane attack complexes (MACs) that cause cell death and tissue damage.  |
| Competitiveness          | <p>Current treatments such as FcRn inhibitors or C5 inhibitors achieve complete remission in only 20–30% of myasthenia gravis patients, primarily due to incomplete suppression of the complement system, particularly activation via the alternative pathway. Their efficacy also tends to diminish or fluctuate over time. IM-101, a 3rd generation complement inhibitor, delivers exceptional efficacy by simultaneously blocking both the classical and alternative pathways.</p> <p>FcRn inhibitor: Efgartigimod, Rozanolixizumab, Nipocalimab<br/>           C5 inhibitor: Eculizumab, Ravulizumab, Zilucoplan</p> |
| Development Stage        | Phase 1  |
| Route of Administration  | Intravenous  |

*Any unauthorized distribution or reproduction of this material is strictly prohibited.*