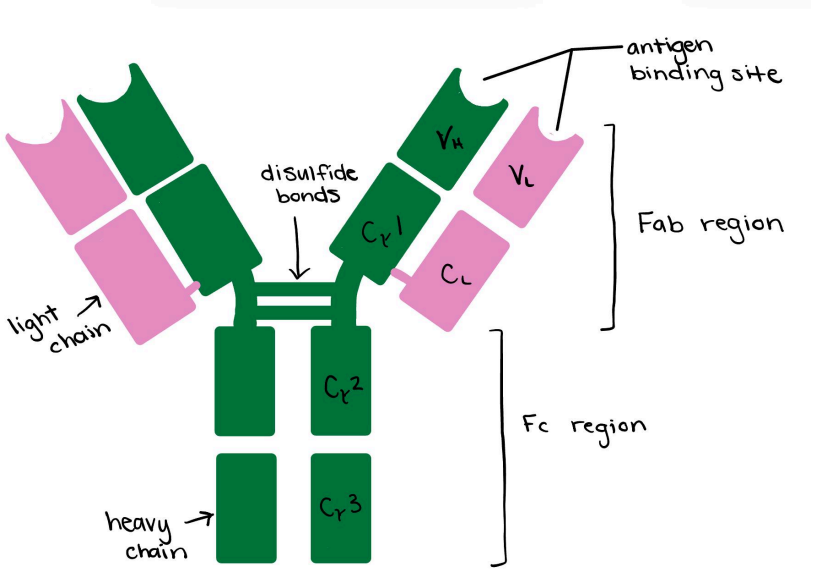
Monoclonal Antibody: Rozanolixizumab

Rozanolixizumab is a monoclonal antibody used to treat the autoimmune disorder myasthenia gravis. This disease causes muscular weakness as a result of autoantibodies produced that bind to acetylcholine receptors (Matic & Bril, 2025). In a regular muscle contraction, starting at the neuromuscular junction, an action potential is generated by acetylcholine which allows calcium to be released by the sarcoplasmic reticulum. Calcium can then bind to troponin on actin, exposing an active site for myosin to bind, initiating the "powerstroke" responsible for a muscle contraction. In the presence of the autoantibodies that lead to the autoimmune disorder myasthenia gravis, acetylcholine receptors are blocked, thus limiting the action potential necessary to release calcium to allow for regular muscle contraction. The reason for the presence of these autoantibodies is directly related to the thymus. It has been noted in a recent study that individuals with myasthenia gravis still have their thymus present in adulthood. The thymus usually atrophies after puberty and is replaced with fat. It is believed that the presence of this primary immune organ is producing faulty T-cells that cause the immune system to attack itself (National Institute of Health, 2025). As a result, individuals who suffer from this autoimmune disease experience severe muscle weakness (Matic & Bril, 2025). Some symptoms of this autoimmune disorder include drooping of one or both eyelids, and difficulty controlling the muscles of the face and throat. Difficulty swallowing, chewing, controlling facial expressions, and speaking are all hallmark signs of myasthenia gravis (Mayo Clinic, 2025).

Rozanolixizumab is an IgG antibody and is illustrated as such:



Rozanolixizumab functions by interacting with the harmful self antibodies in the neuromuscular junction to inhibit the degradation of acetylcholine receptors. Rozanolixizumab binds to the FcRn proteins in the neuromuscular junction, the place where nerves and muscles interact with one another, to prevent the harmful antibodies produced by individuals with myesthenia gravis from binding instead (Matic & Bril, 2025). Rozanolixizumab has a higher binding affinity with FcRn than the harmful antibodies that it is designed to eliminate, therefore these harmful antibodies are eliminated from the cell as a result of having no particular receptor to bind to. This will allow for the binding of acetylcholine to its receptors in the neuromuscular junction, allowing for the generation of action potential to release calcium ions from the sarcoplasmic reticulum, to complete a muscle contraction. A higher affinity of muscle contraction would decrease the muscle weakness experienced by individuals with myasthenia gravis. The entire mechanism by which Rozanolixizumab functions in the neuromuscular junction is not 100% understood, but it is currently more effective and safer than other treatments on the market such as the use of corticosteroids. As mentioned earlier, one of the possible causes of myasthenia gravis is the production of the harmful autoantibodies that are produced specifically in the thymus. Corticosteroids function to suppress T cells, however the chronic use of steroids can lead to a number of severe side effects including hyperglycemia, gastrointestinal complications, musculoskeletal issues, and ophthalmic complications (Matic & Bril, 2025). As of right now, the immune suppressive route appears to be the safest in treating myasthenia gravis.

## References

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