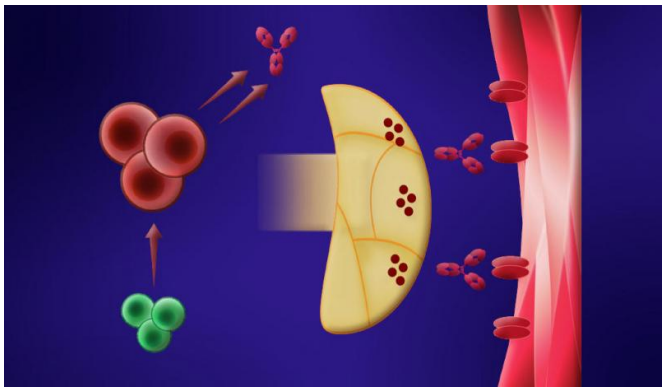
Study explains why myasthenia patients relapse after treatment

8 September 2017



Credit: Yale University

Autoantibody-producing plasmablasts after B cell depletion identified in muscle-specific kinase myasthenia gravis, *JCI Insight* (2017). DOI: 10.1172/jci.insight.94263

Provided by Yale University

A new Yale-led study helps explain why some myasthenia gravis (MG) patients relapse after initially responding to a drug called rituximab, commonly used to treat the incurable autoimmune disease marked by muscle weakness and fatigue.

In patients with MG, B cells—a subset of white cells that produce antibodies—are abnormal and attack the neuromuscular junction in muscle tissue creating weakness and fatigue.

"While therapy with rituximab eliminates B cells, they remain abnormal after regenerating and contribute to relapse," said Dr. Kevin C. O'Connor, associate professor of neurology and co-senior author of the report.

"Disease relapse following successful rituximab treatment could be predicted, allowing physicians to tailor therapy on an individual basis," said Dr. Richard Nowak, the co-senior author of the report and director of the Yale Myasthenia Gravis Clinic. The findings are reported Sept. 7 in the *Journal of Clinical Investigation-Insight*.

More information: Panos Stathopoulos et al.

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