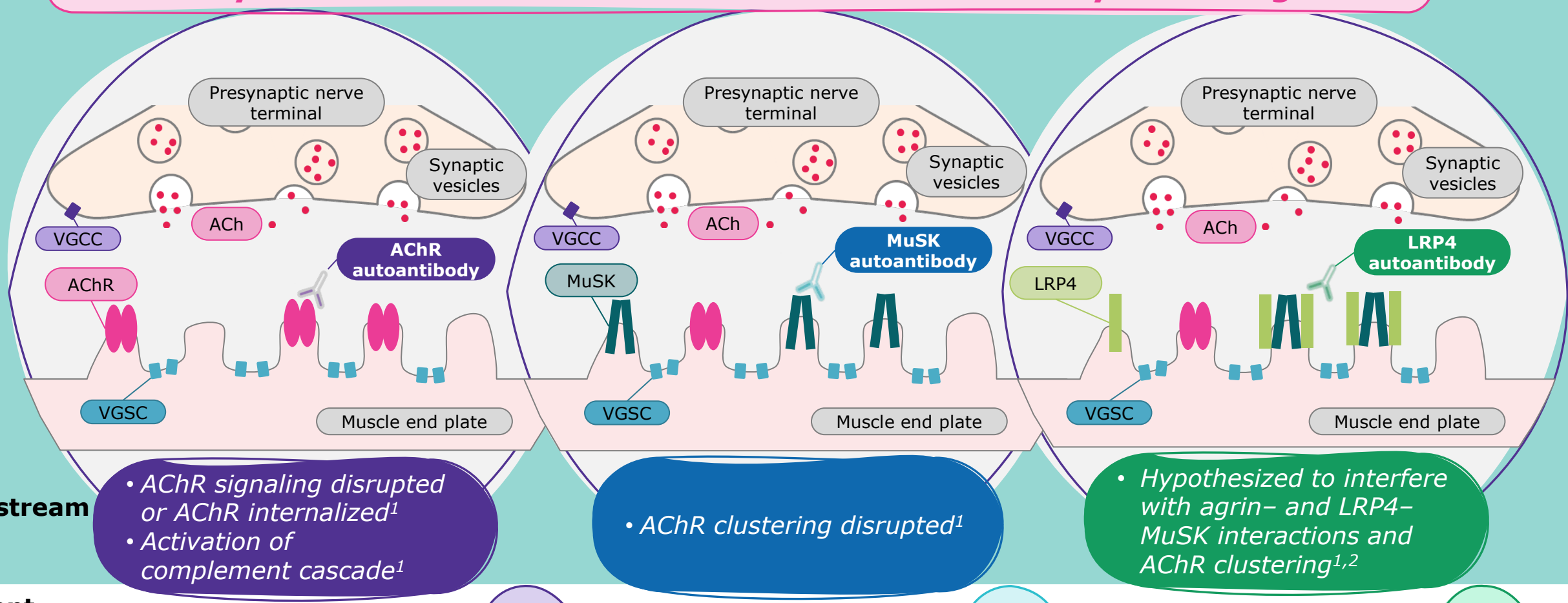
A variety of autoantibodies are known to contribute to myasthenia gravis*



	Anti-AChR (~85%)	Anti-MuSK (~6%)	Anti-LRP4 (~2%)
Downstream effect	• *AChR signaling disrupted or AChR internalized*[1] • *Activation of complement cascade*[1]	• *AChR clustering disrupted*[1]	• *Hypothesized to interfere with agrin– and LRP4–MuSK interactions and AChR clustering*[1,2]
Percent prevalence in pwMG[1]			
Autoantibody IgG subclass[1,3,4]	• IgG1 and IgG3	• IgG4	• IgG1
Cell type producing auto-antibodies[3,4]	• Short-lived plasmablasts and long-lived plasma cells	• Short-lived plasmablasts	• Further research is needed to determine the cell types that secrete LRP4 autoantibodies
Common symptoms[1,3-6]	• **Ocular** and **bulbar** • **Muscle** and **limb** weakness • **Thymic hyperplasia** or **atrophy**	• Acute onset mainly affecting the **facial-bulbar** muscles • Frequent early **respiratory crises**	• **Ocular** and **bulbar** • **Muscle** and **limb** weakness

~10% of pwMG are **seronegative** and **lack detectable autoantibodies**[3]

*Other rare autoantibody types have been identified (e.g., anti-agrin) and are being studied.[3,6]

ACh, acetylcholine; AChR, acetylcholine receptor; Ig, immunoglobulin; LRP4, low-density lipoprotein receptor-related protein 4; MuSK, muscle-specific kinase; pwMG, people with myasthenia gravis; VGCC, voltage-gated calcium channel; VGSC, voltage-gated sodium channel.

1. Lazaridis K, Tzartos SJ. Front Neurol. 2020;11:596981. **2.** Yu Z, et al. Neurology. 2021;97(10):e975–e987. **3.** Iorio R. Nat Rev Neurol. 2024;20(2):84–98. **4.** Fichtner ML, et al. Front Immunol. 2020;11:776. **5.** Rodolico C, et al. Front Neurol. 2020;11:660. **6.** Rivner MH, et al. Muscle Nerve. 2020;62(3):333–343.

US-NONNI-02461 | GL-NONNI-02599 | June 2026
This material is funded and organized by Merck KGaA, Darmstadt, Germany. EMD Serono is the healthcare business of Merck KGaA, Darmstadt, Germany, in the US and Canada. This material is provided for educational and supplemental purposes only and is not intended to replace clinical judgment. Assessment, monitoring, and diagnosis should be based on a comprehensive evaluation by the treating physician.