Evolution of gMG Therapy:
From Broader Immunosuppression to Precision Therapy

argenx

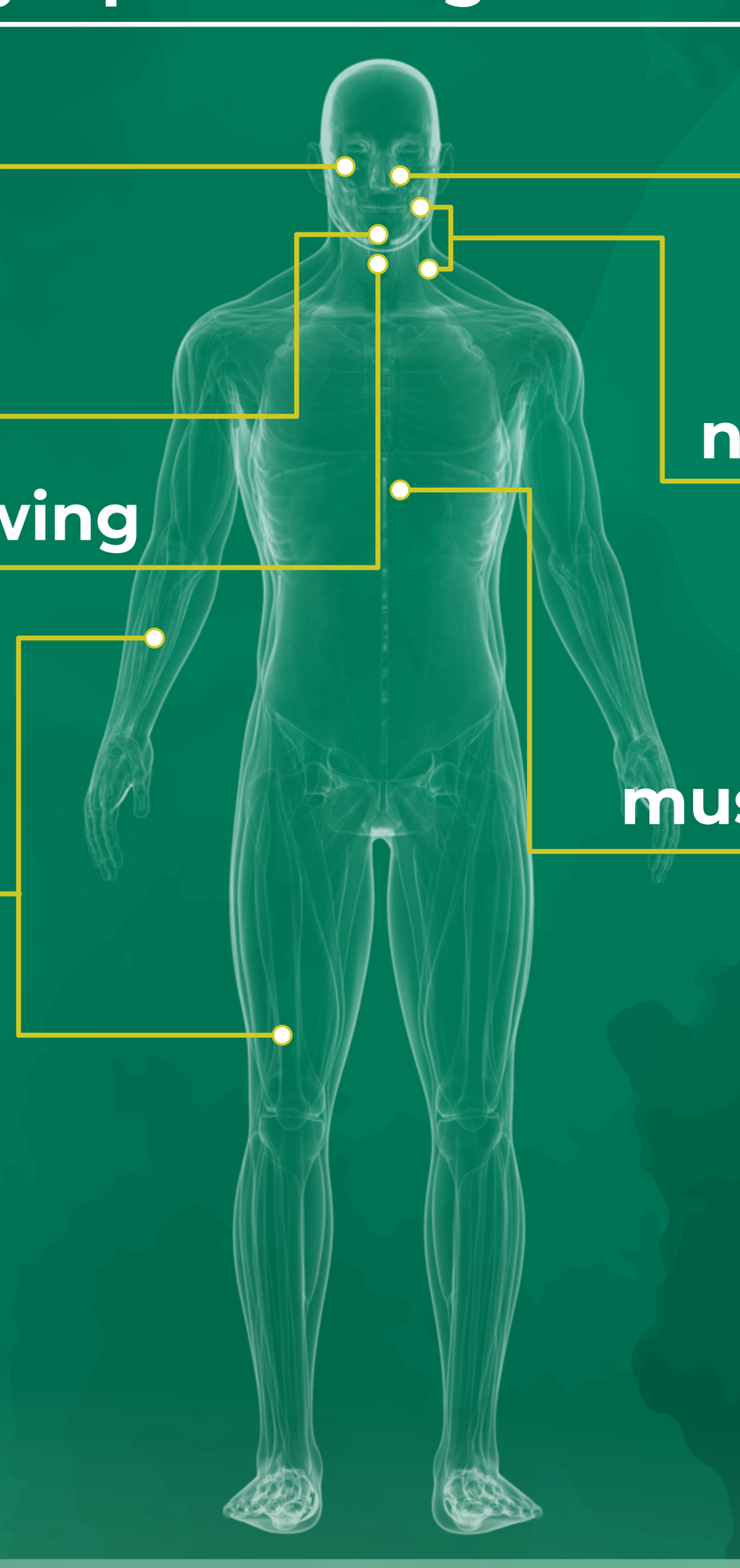
Efgartigimod▼ is indicated in the EU as an add-on to standard therapy for adult patients with gMG who are AChR antibody-positive. Prescribing information can be found here. Adverse event reporting details can be found here. The publication of this promotional infographic was supported and funded by argenx. This content is intended for healthcare professionals outside the US only.

gMG is a rare, chronic, IgG-mediated autoimmune neuromuscular disease[1–7]

IgG autoantibodies cause unpredictable and fluctuating muscle weakness, which profoundly impacts patients' lives, even on standard therapies[1–10,*]

- **Fatigue and inability to participate in hobbies or social activities[6]**
- **Depression and anxiety[3,7]**
- **Risk of severe side effects of long-term broad immunosuppressive therapy[8]**

Most common symptoms of gMG[2,5,6]



IgG autoantibodies drive pathogenesis in AChR antibody–positive gMG through three mechanisms[5,11]

~85% of patients with gMG have AChR antibody–positive disease[5,12]

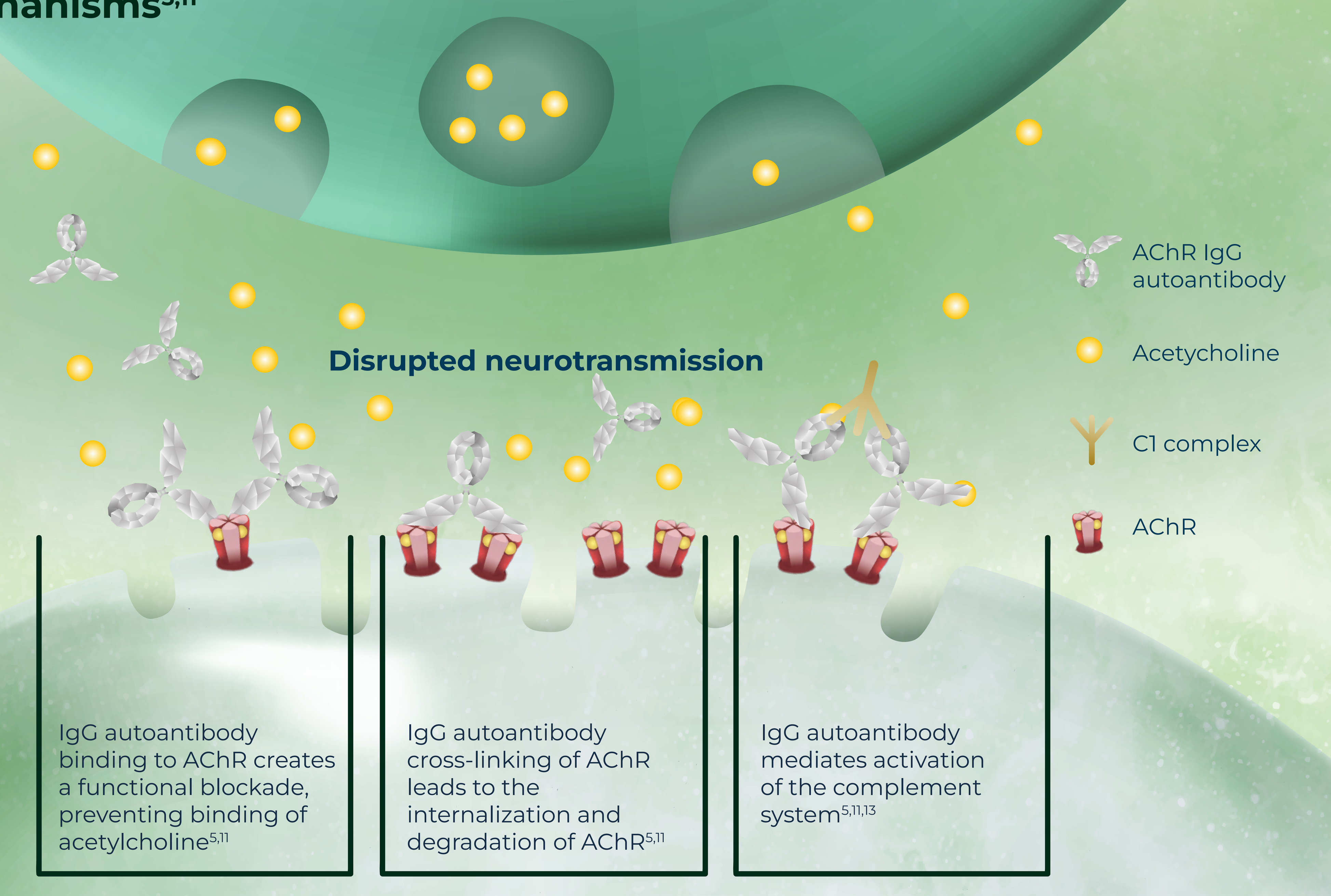


Figure adapted from Wolfe GI, et al. J Neurol Sci. 2021;430:118074 (CC BY 4.0).

Targeted therapies for gMG may reduce the need for broad and unselective immunosuppression and improve patients' ability to perform daily activities[14–18]

Evolution of treatment options for gMG

Traditional treatment for gMG includes AChEIs and the off-label use of broad-acting immunosuppressants, which can be associated with potential serious adverse events[9,19,20]

- **Thymectomy[19,21]**
- **AChEIs[19,21]** – Reduce the breakdown of ACh in the NMJ
- **Steroids[19,21,22]** – Broad immune suppression
- **IVIg or PLEX[19,21,23]**
- **NSISTs[19,21,22]** – Suppress B and/or T cell proliferation and activity
- **B cell depletion[19,21]** – Prevent B cell activation and proliferation
- **C5i[19,21]** – Complement 5 inhibition

FcRn blockers

- **Full-length FcRn monoclonal antibodies[19,21]** – Block IgG binding to FcRn, reducing circulating IgG
- **Efgartigimod** – First and only Fc-fragment approved for treatment of gMG[24–26]

Precision

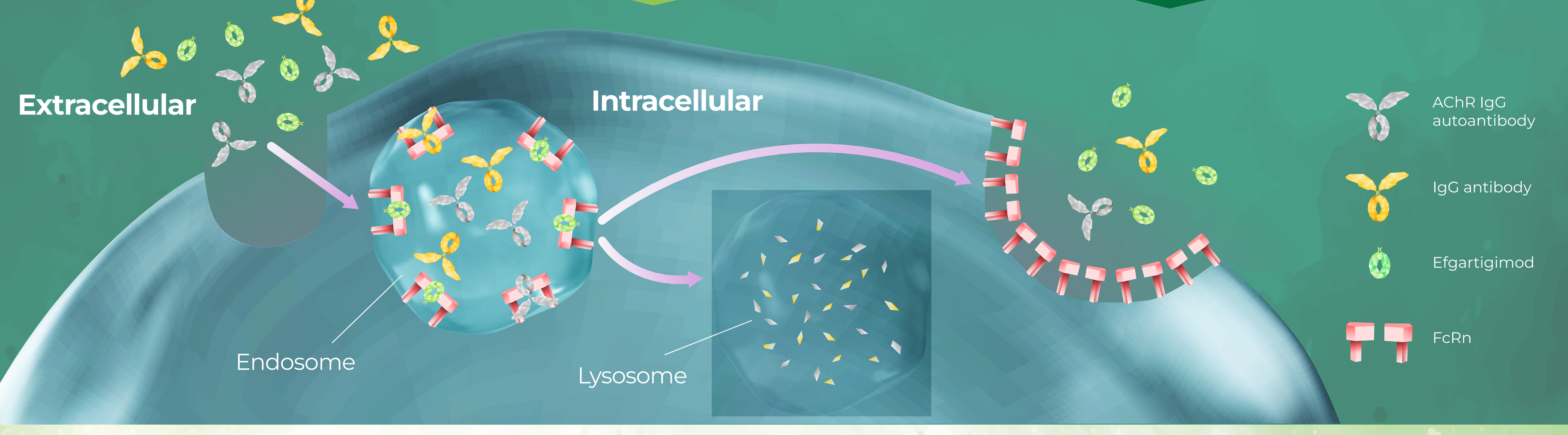
Efgartigimod is the first and only precision Fc-fragment therapy approved to treat adults with AChR antibody-positive gMG.[24–27] Efgartigimod helps to restore neurotransimission by blocking FcRn-mediated IgG recycling[14,17,24–28]

Efgartigimod targets FcRn, resulting in the reduction of circulating IgG antibodies, including AChR autoantibodies[14,17,24–28]

Blocks FcRn
Efgartigimod outcompetes IgG antibodies, including AChR IgG autoantibodies, to bind to FcRn, thus blocking FcRn[27]

Reduces IgG
Unbound IgG antibodies, including AChR IgG autoantibodies, are then degraded in the lysosome[27]

Restores function
By binding to and blocking FcRn, efgartigimod helps clear IgG autoantibodies that disrupt neurotransmission[27,28]

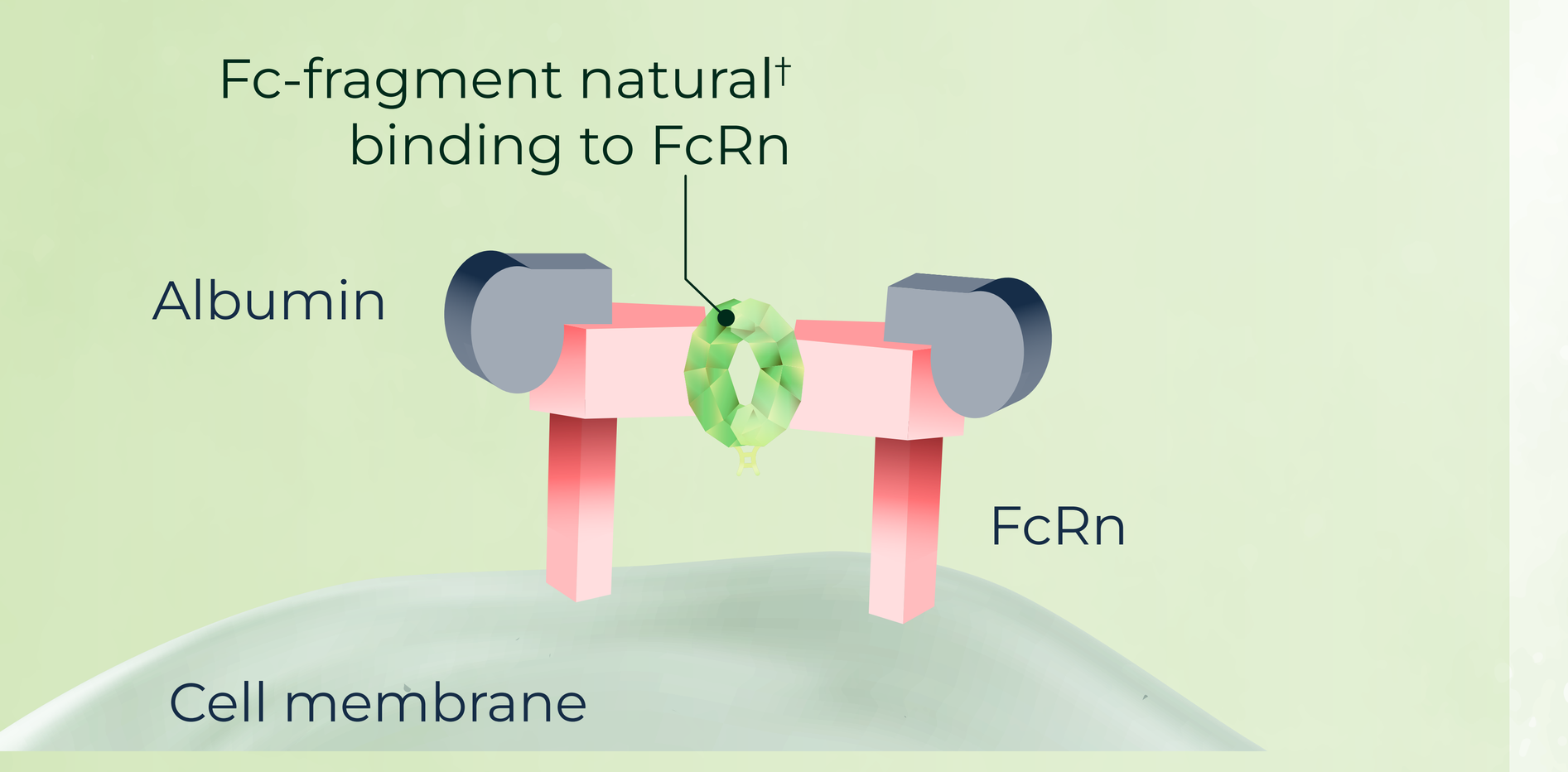


Efgartigimod has been engineered for natural†, high-affinity binding to FcRn[27–29]

Benefits of precision

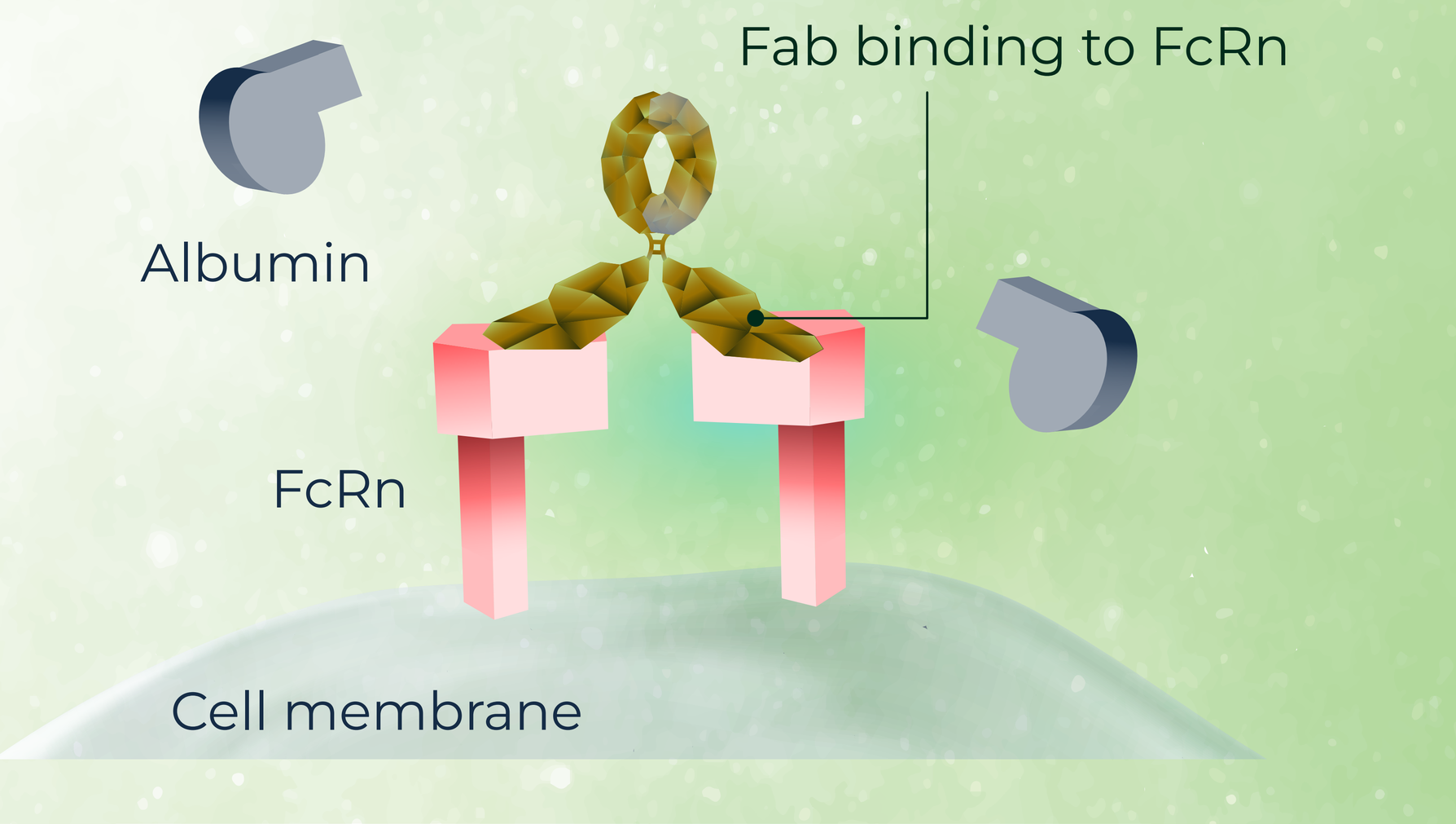
- **No impact** on IgM, IgA, IgD, IgE levels[27]
- **No impact** on IgG production or cellular immunity[30]
- **No decrease** in FcRn levels[31]
- **No vaccination** or pre-medication required prior to treatment with efgartigimod[24]

Efgartigimod (Fc-fragment)



No reduction in albumin or increase in cholesterol levels[24,27,28,31]

Full-length IgG FcRn blockers



May impact albumin and cholesterol levels[32]

Data (references 15 and 16) will be made available by argenx upon request. The illustrations depict cellular processes for visualization purposes and do not fully represent the complete biological situation.

▼This medicinal product is subject to additional monitoring. This will allow quick identification of new safety information.[24] Healthcare professionals are asked to report any suspected adverse reactions.

*Standard therapies include: AChEI, steroids, NSISTs, or combined therapy.[5,6]
†Efgartigimod mimics the binding of the Fc regions of endogenous IgG antibodies to FcRn, retains natural pH-dependent binding, and does not impact FcRn levels.[14,28,31]

ACh=acetylcholine; AChEI=acetylcholinesterase inhibitor; AChR=acetylcholine receptor; C5i=C5 inhibitor; FcRn=neonatal Fc receptor; gMG=generalized myasthenia gravis; Ig=immunoglobulin; IVIg=intravenous immunoglobulin; NMJ=neuromuscular junction; NSIST=non-steroidal immunosuppressive treatment; PLEX=plasma exchange.

1. Grob D, et al. *Muscle Nerve.* 2008;37(2):141–149; 2. Twork S, et al. *Health Qual Life Outcomes*. 2010;8:129; 3. Berrih-Aknin S, et al. *BMJ Open.* 2023;13(5):e068104; 4. Carr AS, et al. *BMC Neurol*. 2010;10:46; 5. Gilhus NE, et al. *Nat Rev Neurol.* 2016;12(5):259-268; 6. Jackson K, et al. *Neurol Ther.* 2023;12(1):107-128; 7. Law N, et al. *Neurol Ther.* 2021;10(2):1103-1125; 8. Johnson S, et al. *Med Sci Monit.* 2021;27:e933296-1–e933296-9; 9. DeHart-McCoyle M, et al. *BMJ Med.* 2023;2(1):e000241; 10. Wiendl H, et al. *Ther Adv Neurol Disord*. 2023;16:17562864231213240; 11. Obaid AH, et al. *Neurol Neuroimmunol Neuroinflamm.* 2022;9(4):e1169; 12. Behin A, Le Panse R. *J Neuromuscul Dis.* 2018;5(3):265-277; 13. Daha NA, et al. *Mol Immunol.* 2011;48(14):1656-1665; 14. Howard Jr JF, et al. *Lancet Neurol.* 2021;20(7):526-536; 15. Hoffmann S, et al. Oral presentation at EAN; 1–4 July 2023; Budapest, Hungary; 16. Ruck T, et al. Poster presented at EAN; 29 June–2 July 2024; Helsinki, Finland; 17. Saccà F, et al. *Eur J Neurol*. 2023;31(6):e16180; 18. Wolfe GI, et al. *eNeurologicalSci.* 2024;37:100541; 19. Alhaidar MK, et al. *J Clin Med.* 2022;11(6):1597; 20. Sanders DB, et al. *Neurology.* 2016;87(4):419-425; 21. Nguyen-Cao TM, et al. *J Neurol Sci.* 2019;406:116428; 22. Lascano AM, Lalive PH. *Autoimmun Rev.* 2021;20(1):102712; 23. Pavlekovics M, et al. *Biomedicines.* 2023;11(12):3180; 24. argenx BV. Vyvgart. Summary of Product Characteristics (SmPC). March 2025; 25. Zhu LN, et al. *Neural Regen Res.* 2023;18(8):1637-1644; 26. Cavalcante P, et al. *Front Immunol*. 2024;15:1404191; 27. Wolfe GI, et al. *J Neurol Sci.* 2021;430:118074; 28. Ulrichts P, et al. *J Clin Invest.* 2018;128(10):4372-4386; 29. Brinkhaus M, et al. *Nat Commun.* 2022;13(1):6073; 30. Binazon O, et al. *J Immunotoxicol.* 2025;22(1):2459934; 31. Ma G, et al. *JCI Insight.* 2024;9(10):e176166; 32. Ward ES, et al. *Front Immunol.* 2022;13:892534.

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