



Pr **VYVGART**<sup>®</sup>  
efgartigimod alfa  
400 mg/20 mL

The **first and only** IgG Fc-antibody fragment for the treatment of gMG<sup>1</sup>

## **VYVGART**<sup>®</sup>: recharging the neuromuscular junction

**Explore how VYVGART<sup>®</sup> reduces pathogenic IgG levels  
in patients with gMG**

VYVGART<sup>®</sup> (efgartigimod alfa) is indicated for the treatment of adult patients with generalized Myasthenia Gravis (gMG) who are anti-acetylcholine receptor (AChR) antibody positive.

AChR=anti-acetylcholine receptor; Fc=crystallizable fragment; gMG=generalized Myasthenia Gravis; IgG=immunoglobulin G.

# VYVGART®: a unique mechanism of action

The **first and only** IgG Fc-antibody fragment for the treatment of adults with gMG who are anti-AChR antibody positive<sup>1</sup>

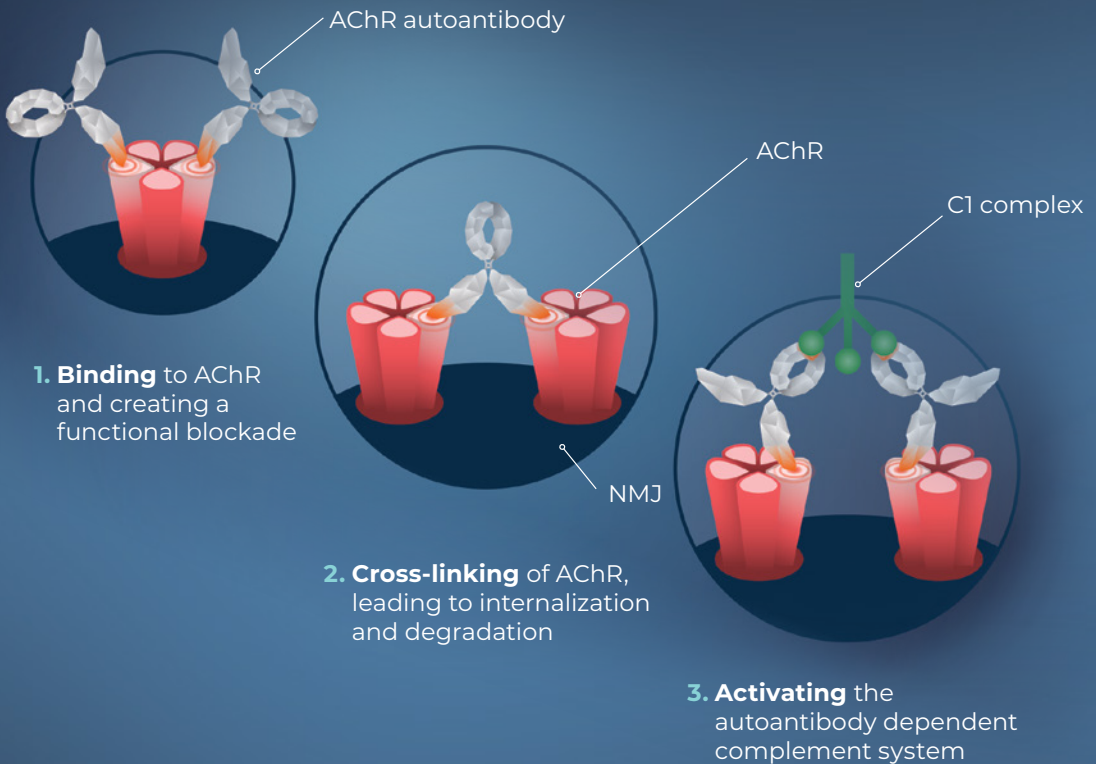


VYVGART® (efgartigimod alfa) is the Fc fragment of an IgG antibody<sup>†</sup> engineered for high affinity to FcRn.<sup>1,4</sup>

- By lowering AChR autoantibodies levels in the body, VYVGART® targets the key pathogenic driver in gMG.<sup>1,4,5</sup>

# AChR autoantibodies exert a direct pathogenic effect in gMG<sup>2</sup>

## AChR autoantibodies disrupt neurotransmission in three ways:<sup>3</sup>

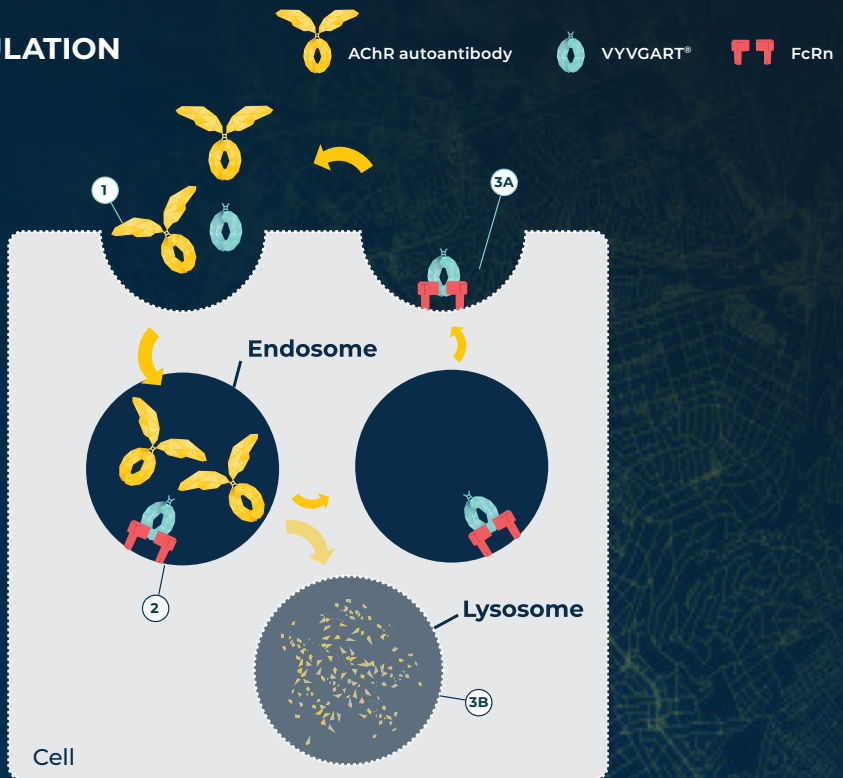


- Together, these three mechanisms reduce the function of AChR, decrease the total number of AChRs, and damage the structural integrity of the NMJ.<sup>2,3</sup>

# VYVGART® targets the underlying disease biology of gMG<sup>4,5</sup>

## VYVGART® targets FcRn to reduce IgG and AChR autoantibodies

### BLOOD CIRCULATION



- 1 IgG and VYVGART® are taken into the cell
- 2 VYVGART® binds to FcRn, competing with IgG
- 3A FcRn-bound VYVGART® is rescued from cellular degradation pathways
- 3B Non-FcRn bound IgG (auto)antibodies are degraded in the lysosome

### VYVGART® works upstream to effectively target the underlying gMG disease biology.

#### VYVGART® BLOCKS FcRn:

By blocking FcRn, VYVGART® reduces levels of circulating IgG antibodies, including AChR autoantibodies.

#### REDUCING IgG:

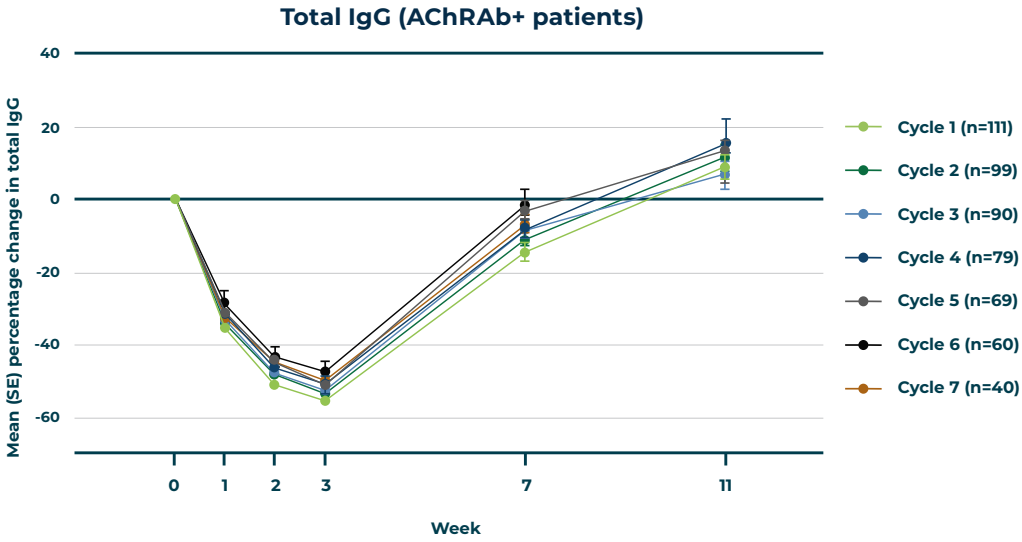
By reducing IgG antibodies, including AChR autoantibodies, VYVGART® targets the key pathogenic driver of gMG.

#### RESTORING FUNCTION:

Lower levels of AChR autoantibodies result in less damage and dysfunction at the NMJ.



**In ADAPT+, mean total IgG levels decreased with maximum reductions observed at Week 3 and reached baseline between Weeks 7-11<sup>†</sup>**



IgG monitoring is not required before or during VYVGART® treatment.

AChR=acetylcholine receptor; IgG=immunoglobulin G; SE=standard error.  
<sup>†</sup>ADAPT+ was an open-label, single-arm, multicenter, up to 3-year extension of the pivotal phase 3 ADAPT study. Efgartigimod was administered in treatment cycles of 4 intravenous infusions (one 10 mg/kg infusion per week). Initiation of subsequent treatment cycles was individualized based on clinical evaluation.



# MyPATH

VYVGART® Patient Support Program

Supporting you and your patients  
with the VYVGART® treatment journey



**Enrol your patients in the MyPATH  
Patient Support Program today!**

☎ 1-877-697-2840

🏠 1-877-697-2844

✉ info@mypathpsp.ca

Monday to Friday, 8 a.m. – 8 p.m. EST  
Bilingual services are available.

Please consult the VYVGART® Product Monograph at [https://www.argenx.com/product/vyvgart\\_product\\_monograph.pdf](https://www.argenx.com/product/vyvgart_product_monograph.pdf) for important information relating to:

- Contraindications in patients with hypersensitivity to VYVGART® or any ingredient in the formulation or component of the container.
- Other relevant warnings and precautions regarding infections including upper respiratory tract infections and urinary tract infections, vaccination, fertility, hypersensitivity and infusion related reactions, pregnancy, and breast-feeding.
- Conditions of clinical use, adverse reactions, drug interactions, and dosing information.

The Product Monograph is also available by calling argenx Medical Information at 1-800-731-8917.

**References:** **1.** VYVGART® Product Monograph. argenx. September 19<sup>th</sup>, 2023. **2.** Gilhus NE, et al. Myasthenia gravis - autoantibody characteristics and their implications for therapy. *Nat Rev Neurol*. 2016 May;12(5):259-68. **3.** Huijbers MC, et al. Pathogenic immune mechanisms at the neuromuscular synapse: the role of specific antibody-binding epitopes in myasthenia gravis. *J Intern Med* 2014; 275: 12–26. **4.** Howard JF, et al. Safety, efficacy, and tolerability of efgartigimod in patients with generalised myasthenia gravis (ADAPT): a multicentre, randomised, placebo-controlled, phase 3 trial. *Lancet Neurol*. 2021 Jul;20(7):526-536. **5.** Roopenian DC & Akilesh S. FcRn: the neonatal Fc receptor comes of age. *Nat Rev Immunol*. 2007 Sep;7(9):715-25. **6.** Howard JF, et al. Long-term safety, tolerability, and efficacy of efgartigimod (ADAPT+): interim results from a phase 3 open-label extension study in participants with generalized myasthenia gravis. *Front Neuro*. 2024.

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