

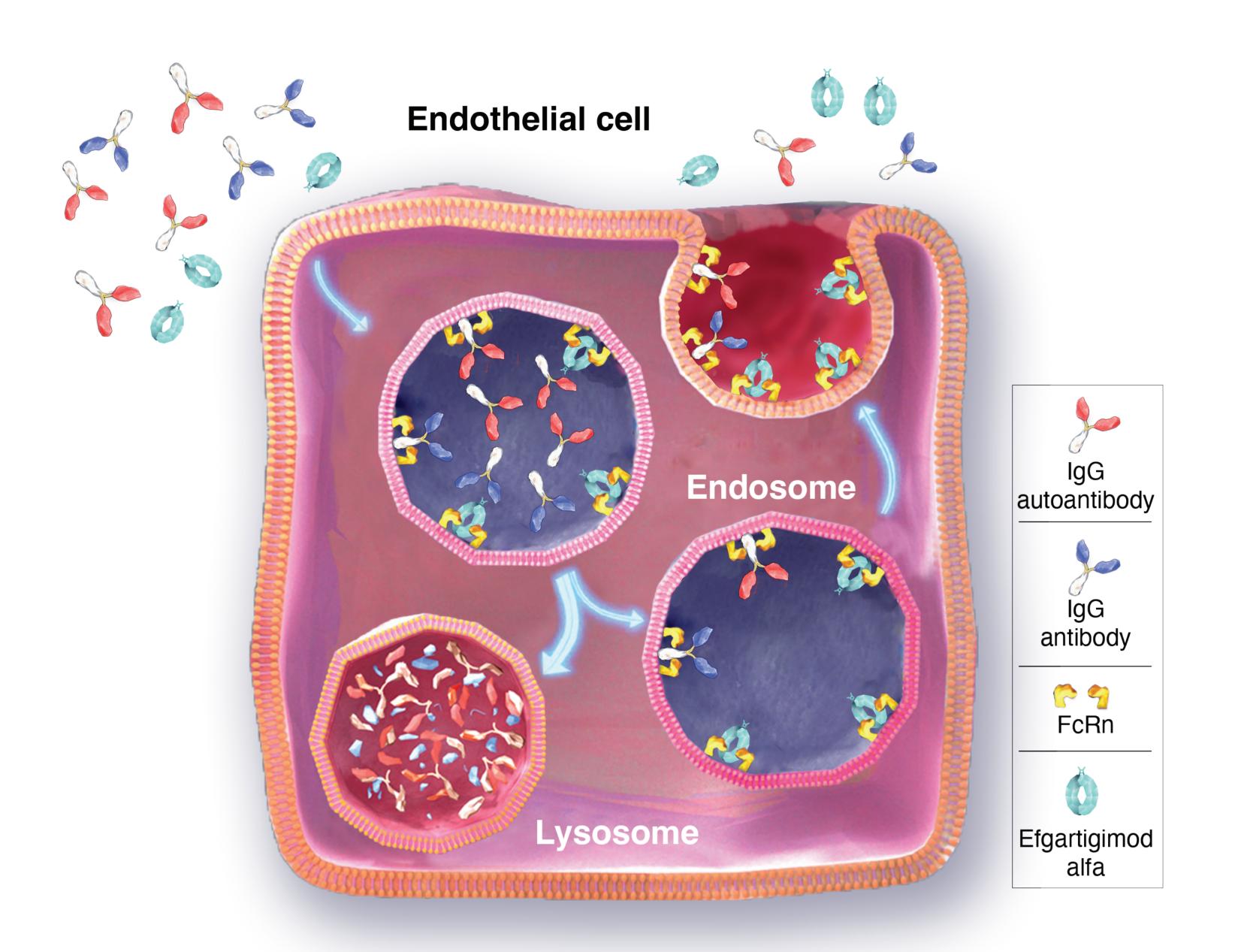
# Efgartigimod Demonstrates Consistent Improvements in Generalized Myasthenia Gravis Across Patient Subgroups, Including Early in Diagnosis Vera Bril,<sup>1,2</sup> Tuan Vu,<sup>3</sup> Edward Brauer,<sup>4</sup> René Kerstens,<sup>4</sup> James F. Howard, Jr,<sup>5</sup> in collaboration with the ADAPT Investigator Study Group



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### INTRODUCTION

### **Efgartigimod Mechanism of Action: Blocking FcRn**

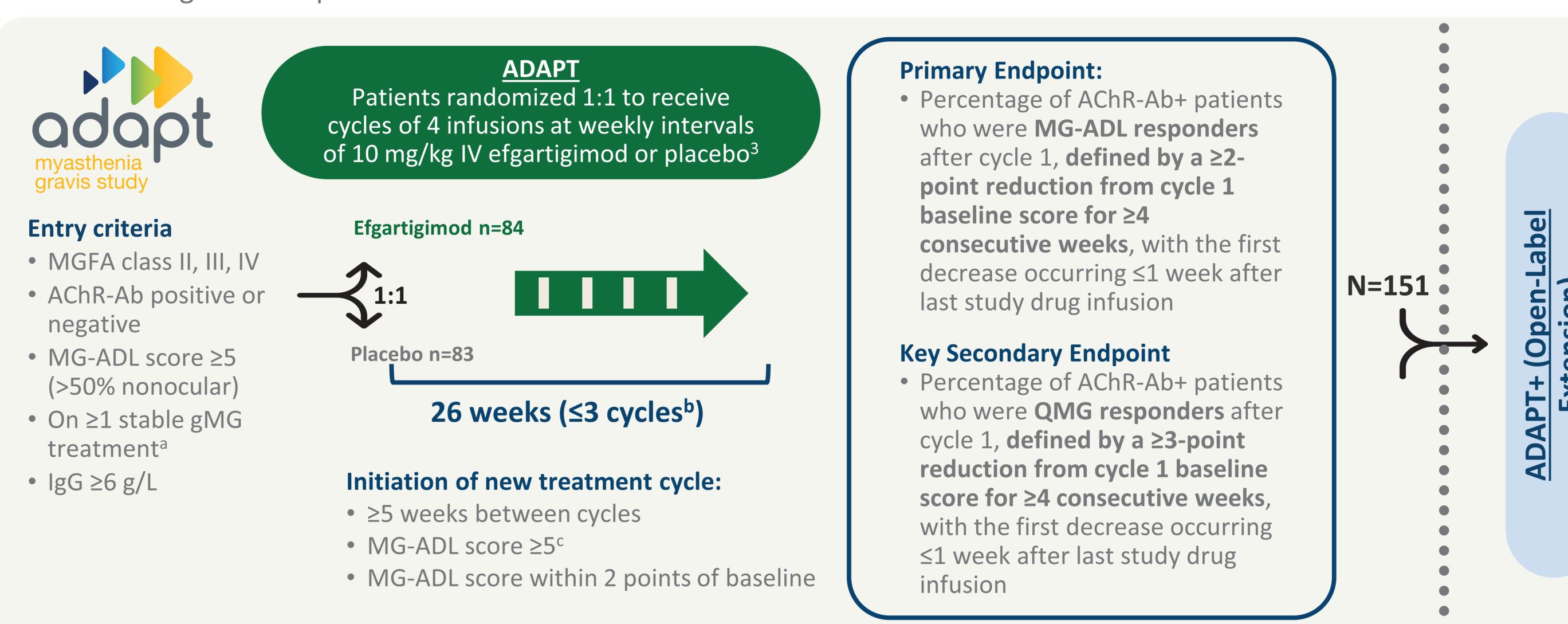


- FcRn recycles IgG, extending its half-life and maintaining its serum concentration<sup>1</sup>
- Efgartigimod is a human IgG1 Fc fragment, a natural ligand of FcRn, engineered for increased affinity to FcRn<sup>2,3</sup>
- Efgartigimod was designed to outcompete endogenous IgG, preventing recycling and promoting lysosomal degradation of IgG<sup>2-5</sup>
- Targeted reduction of all IgG subclasses No impact on IgM or IgA
- No reduction in albumin levels
- No increase in cholesterol
- No impact on IgG production

### **METHODS**

ADAPT was a 26-week, global, multicenter, randomized, double-blind, placebo-controlled, phase 3 trial evaluating the safety and efficacy of efgartigimod in patients with gMG<sup>3</sup>

• Data collected from AChR-Ab+ patients in ADAPT were analyzed based on subgroups by baseline disease factors and concomitant gMG therapies



immunosuppressive therapy. Patients could not change concomitant therapies in ADAPT. b≤3 cycles dosed at ≥8 weeks after initial cycle. cWith >50% from nonocular items.

### **SUMMARY**



Efgartigimod demonstrates consistent improvements compared to placebo, regardless of disease duration, concomitant therapy, prior therapies (including thymectomy), or prior treatment failures



Efgartigimod was well tolerated, with most adverse events being mild or moderate in severity



These data suggest that efgartigimod is an effective treatment in a broad patient population, including early in disease, and early in the treatment journey of patients with gMG



The ADAPT+ study is currently ongoing

## **RESULTS**

#### Table 1. Baseline Characteristics AChR-Ab+ Patients Efgartigimod (n=65) Age, mean, y (SD Female, n (%) 49.2 (15.5) 40 (62.5) Time since diagnosis, mean, y (SD) 8.93 (8.2) MG-ADL score, mean (SD) 8.6 (2.1) QMG score, mean (SD) 15.2 (4.4) MGFA class at screening, n (%) 28 (43.1) 25 (39.1) 36 (56.3) Class IV 3 (4.7) **Prior treatment with NSIST**, n (%) 43 (67.2) MG therapies at baseline, n (%) 40 (61.5) 37 (57.8) 51 (79.7)

ischemia, atrial fibrillation, spinal ligament ossification.

### **Table 2.** Safety Data, Overall Population

	Efgartigimod (n=84)	Placebo (n=83)
AEs, <sup>a</sup> n (%)	65 (77.4)	70 (84.3)
SAEs, n (%)	4 (4.8)	7 (8.4)
Discontinued due to AEs,b n (%)	3 (3.6)	3 (3.6)

### Figure 1. Proportion of MG-ADL and QMG Responders by Disease Duration AChR-Ab+ Patients, Cycle 1

Figure 2. Proportion of MG-ADL and QMG Responders by Concomitant Therapies, AChR-Ab+ Patients, Cycle 1

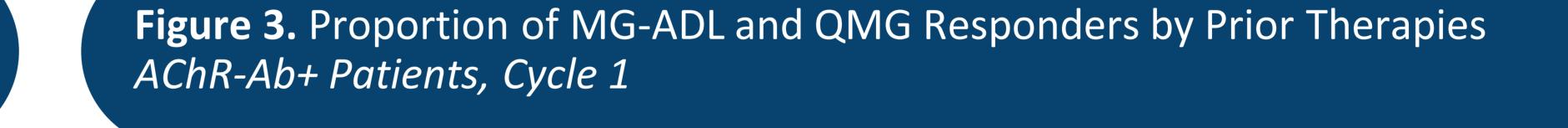
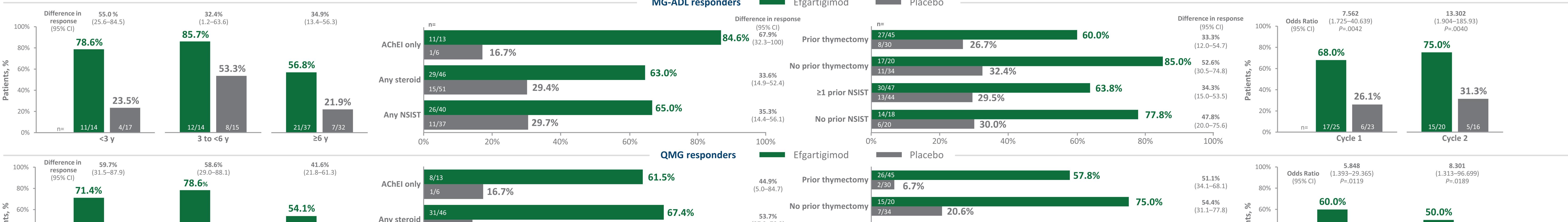
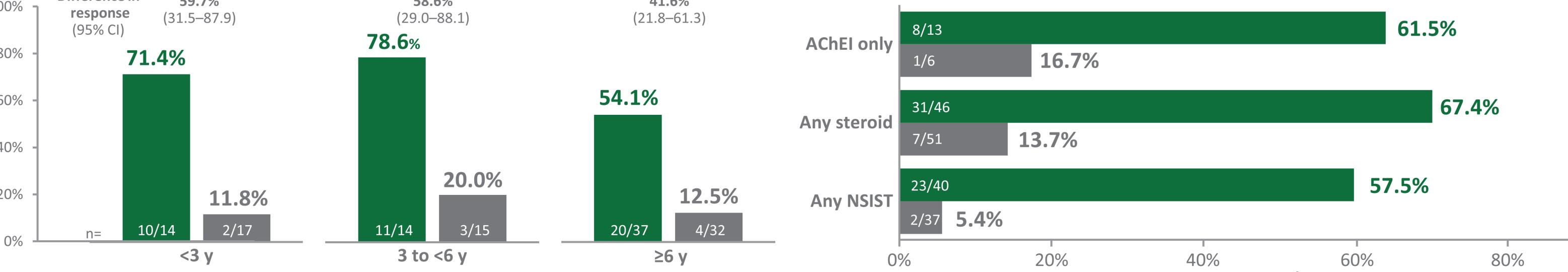


Figure 4. Proportion of MG-ADL and QMG Responders Among Patients Without Prior Treatment Failures<sup>a</sup> AChR-Ab+, Cycles 1 and 2





**53.7**% (37.2–70.2) 50.5% ≥1 prior NSIS (34.1 - 66.9)**52.1%** (35.1–69.1) 47.2% No prior NSIS (19.1 - 75.3)100%

multiple times in the

### **ABBREVIATIONS**

Steroid and NSIST

AChEI, acetylcholinesterase inhibitor; AChR-Ab+, acetylcholine receptor antibody seropositive; AE, adverse event; FcRn, neonatal Fc receptor; IgG, immunoglobulin G; IVIg, intravenous immunoglobulin G; IVIg

#### **REFERENCES** 1. Sesarman A, et al. Cell Mol Life Sci. 2010;67(15):2533-2550. 2. Ulrichts P, et al. J Clin Invest. 2015;6:176.

31 (48.4)

6 (9.4)

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