variable region (VL) comprising the amino acid of SEQ ID NO: 2, wherein the antibody is administered intravenously at a dose of from about 250 mg to about 350 mg every 6 months, thereby treating the MG. In examples, two weeks prior to the administering, an initial dose of the antibody is administered to the subject, wherein the initial dose is from about 250 mg to about 350 mg. In examples, a second dose of the antibody of about 300 mg is administered two weeks after the administering. In examples, the administering is effective in (i) depleting at least about 90% of circulating CD20+ B cells for at least six months; (ii) does not increase incidence of an infection in the subject; or (iii) (i) and (ii). In examples, the administering reduces a level of peripheral blood $CD20^-$ plasmablasts and plasma cells within about 8 days following the administering. In examples, the dose of a provided composition is about 300 mg. In examples, the administering of a provided composition is effective in reducing MG-related disability. In examples, the administering of a provided composition is effective in reducing frequency of MG exacerbations. In examples, a subject is acetylcholine receptor antibody positive (AChR-Ab+). In examples, a subject is muscle-specific kinase-antibody positive (MuSK-Ab+). In examples, a subject is acetylcholine receptor antibody positive (AChR-Ab+) and muscle-specific kinase-antibody positive (MuSK-Ab+). In examples, the frequency of MG exacerbations is reduced by at least about 1-fold as compared to an otherwise comparable subject lacking the administering. In examples, an administering is effective in reducing fatigue in a subject as determined by Neuro-QoL Fatigue score. subject is further administered one or more additional therapies. In examples, one or more additional therapies comprise one or more standard-of-care therapies. In examples, one or more standard-of-care therapies comprises a corticosteroid, a nonsteroidal immunosuppressive therapy, or both. In examples, the one or more standard-of-care therapies comprises the corticosteroid, and wherein the corticosteroid comprises prednisone. In examples, the one or more additional therapies are one or more of azathioprine, mycophenolate mofetil, mycophenolic acid, or tacrolimus. In examples, an antibody is Inebilizumab. In examples, an antibody is administered at a dose of about 300 mg. In examples, an administration, treats MG as determined by: a) a reduction in MG activities of daily living score; b) a reduction of a MG score; c) an increase in a quality-of-life score; and/or d) a reduction in incidence of exacerbations. In examples, MG is refractory MG. In examples, a subject in need of treatment for MG has uncontrolled MG, on a standard-of-care therapy, as determined by one or more of: a) a MG Foundation of America classification of II, III, or IV; b) an MG activities of daily living (MG-ADL) score ≥6, with >50% of this score being attributed to non-ocular items; or c) a quantitative MG (QMG) score ≥11. In examples, when a subject in need of treatment for MG is receiving a dosage of prednisone over 5 mg/day or equivalent, the subject will undergo tapering

of the prednisone dosage to 5 mg/day. In examples, a maximum dosage of: a) tacrolimus ≤3 mg/day; b) azathioprine ≤3 mg/kg/day; c) mycophenolate mofetil ≤3 g/day; and/or d) mycophenolic acid ≤1440 mg/day. In examples, a subject in need of treatment for MG has uncontrolled MG, on a standard-of-care therapy, as determined by the MG-ADL score of ≥6, with >50% of the score being attributed to non-ocular items, and wherein the MG-ADK score is reduced after the administering. In examples, the reduction is of at least about 2 points. In examples, a method comprises determining: a) a B-cell subset phenotype; b) a B-cell receptor repertoire; c) a B-cell gene expression profiling; or d) any combination thereof. In examples, an administration is effective in reducing or eliminating mature plasma cells in the subject. In examples, an administration results in longer-lasting B-cell reduction or elimination as compared to an otherwise comparable method comprising administration of an anti-CD20 therapy.

[0010] Provided are methods of treating myasthenia gravis (MG), comprising: (a) administering a standard-of-care therapy in an amount sufficient to treat MG in a subject in need thereof; and (b) administering a pharmaceutical composition that comprises an antibody that comprises a heavy chain variable region (VH) comprising the amino acid of SEQ ID NO:1, and a light chain variable region (VL) comprising the amino acid of SEQ ID NO: 2 at a dose of from about 250 mg to about 350 mg. In examples, the standard-of-care therapy comprises a corticosteroid. In examples, the standard-of-care therapy comprises a nonsteroidal immunosuppressive therapy. In examples, the standard-of-care therapy comprises a corticosteroid and a nonsteroidal immunosuppressive therapy. In examples, the corticosteroid comprises prednisone. In examples, the antibody is Inebilizumab. In examples, the dose is from about 275 mg to about 325 mg, from about 290 mg to about 310 mg, or 205 mg to about 305 mg. In examples, the dose is about 300 mg. In examples, the subject has a CD19+ B-cell count ≥ 40 cells/μL prior to administration of the antibody. In examples, the subject has a myasthenia gravis foundation of America (MGFA) clinical classification selected from class II, class III, or class IV. In examples, the subject has anti-AChR antibodies or anti-MuSK antibodies, or both. In examples, the subject does not have an immunodeficiency disorder.

[0011] Provided are methods of treating myasthenia gravis (MG), comprising: administering Inebilizumab to a subject in need of treatment for MG, wherein the Inebilizumab is administered intravenously at a dose of about 300 mg every 6 months. In examples, two weeks prior to administering the 300 mg Inebilizumab every 6 months, an initial 300 mg Inebilizumab dose is administered to the subject. In examples, provided can be a method comprising: administering Inebilizumab to a subject diagnosed with MG, wherein the Inebilizumab is

administered intravenously at a first dose of 300 mg, a second dose of 300 mg two weeks after the first dose, and subsequent doses of 300 mg every 6 months following the first dose.

[0012] Provided are methods of treating a subject diagnosed with MG, the methods comprising: administering Inebilizumab to a subject diagnosed with MG, wherein the Inebilizumab is administered at a dose that: (i) depletes at least 90% of circulating CD20+ B cells for at least six months; and (ii) does not increase incidence of an infection in the subject. In examples, the Inebilizumab further depletes peripheral blood $CD20^-$ plasmablasts and plasma cells within about 8 days following the administering. In examples, the dose is 300 mg.

[0013] Provided are methods of reducing MG-related disability, comprising: administering Inebilizumab to a subject in need of treatment for MG, wherein the Inebilizumab is administered intravenously at a first dose of 300 mg, a second dose of 300 mg two weeks after the first dose, and subsequent doses of 300 mg every 6 months following the first initial dose.

[0014] Provided are methods of reducing the frequency of MG exacerbations, comprising: administering Inebilizumab to a subject in need of treatment for MG, wherein the Inebilizumab is administered intravenously with at a first dose of 300 mg, a second dose of 300 mg two weeks after the first dose, and subsequent doses of 300 mg every 6 months following the first dose.

[0015] In examples of any of the methods, a subject is acetylcholine receptor antibody positive (AChR-Ab+). In examples, the subject is muscle-specific kinase-antibody positive (MuSK-Ab+). In examples, the subject is acetylcholine receptor antibody positive (AChR-Ab+) and muscle-specific kinase-antibody positive (MuSK-Ab+). In examples, administration of the Inebilizumab reduces the frequency of MG exacerbations. In examples, administration of the Inebilizumab reduces fatigue as determined by Neuro-QoL Fatigue score. In examples, the subject is further administered one or more additional therapies. In examples, one or more additional therapies comprise one or more standard-of-care therapies. In examples, standard-of-care therapy comprises a corticosteroid, a nonsteroidal immunosuppressive therapy, or both. In examples, the corticosteroid is prednisone. In examples, the one or more additional therapies are one or more of azathioprine, mycophenolate mofetil, mycophenolic acid, or tacrolimus. In examples, the Inebilizumab comprises a heavy chain variable region (VH) comprising the amino acid of SEQ ID NO:1, and a light chain variable region (VL) comprising the amino acid of SEQ ID NO: 2. In examples, the Inebilizumab is administered intravenously. In examples, administration of Inebilizumab treats the MG as determined by: a) a reduction in MG activities

of daily living score; b) a reduction of a MG score; c) an increase in a quality-of-life score; and/or d) a reduction in incidence of exacerbations. In examples, the MG is refractory MG. In examples, the subject in need of treatment for MG has uncontrolled MG, on a standard-of-care therapy, as determined by one or more of: a) a MG Foundation of America classification of II, III, or IV; b) an MG activities of daily living (MG-ADL) score ≥6, with >50% of this score being attributed to non-ocular items; or c) a quantitative MG (QMG) score ≥11. In examples, if the subject in need of treatment for MG is receiving a dosage of prednisone over 5 mg/day or equivalent, the subject will undergo tapering of the prednisone dosage to 5 mg/day. In examples, a maximum dosage of: a) tacrolimus is about ≤3 mg/day; b) azathioprine is about ≤3 mg/kg/day; c) mycophenolate mofetil is about ≤3 g/day; and/or d) mycophenolic acid is about ≤1440 mg/day. In examples, the MG-ADK score is reduced after the administration of Inebilizumab. In examples, the reduction is of at least about 2 points. In examples, a method comprises determining: a) B-cell subset phenotype; b) B-cell receptor repertoire; c) B-cell gene expression profiling; or d) any combination thereof. In examples, administering of a composition provided herein is effective in reducing or eliminating mature plasma cells in the subject. In examples, administration results in longer-lasting B-cell reduction or elimination as compared to an otherwise comparable method comprising administration of an anti-CD20 therapy.

[0016] The description provides for methods of treating MG in a subject in need of treatment for MG, treating a subject diagnosed with MG, reducing MG-related disability in a subject need of treatment for MG and reducing the frequency of MG exacerbations in a subject need of treatment for MG.

[0017] In examples, the disclosure provides a method of treating MG. In the method, VIB551 is administered to a subject in need of treatment for MG. The VIB551 is administered intravenously at a dose of 300 mg every 6 months. In examples of the method, an initial 300 mg dose VIB551 is administered two weeks prior to the 300 mg VIB551 dose being administered every 6 months.

[0018] In examples, the disclosure provides a method of treating a subject diagnosed with MG. In one such method, VIB551 is administered to a subject diagnosed with MG. The VIB551 is administered intravenously with a first initial dose of 300 mg, a second initial dose of 300 mg two weeks after the first initial dose, and subsequent doses of 300 mg every 6 months following the first initial dose. In another such method of treating a subject diagnosed with MG in which VIB551 is administered to a subject diagnosed with MG, the VIB551 is

administered at a dose that: (i) depletes at least 90% of circulating CD20+ B cells for at least six months, and (ii) does not increase risk of infections in the subject. In examples of the methods of treating a subject diagnosed with MG, the VIB551 further depletes peripheral blood CD20 plasmablasts and plasma cells within 8 days following the administering. In yet other examples, the dose VIB551 is 300 mg. In further yet examples, the VIB551 is administered intravenously.

[0019] In examples, the disclosure provides a method of reducing MG-related disability. In the method, VIB551 is administered to a subject in need of treatment for MG. The VIB551 is administered intravenously with a first initial dose of 300 mg, a second initial dose of 300 mg two weeks after the first initial dose, and subsequent doses of 300 mg every 6 months following the first initial dose.

[0020] In examples, the disclosure provides a method of reducing the frequency of MG exacerbations. In the method, VIB551 is administered to a subject in need of treatment for MG. The VIB551 is administered intravenously with a first initial dose of 300 mg, a second initial dose of 300 mg two weeks after the first initial dose, and subsequent doses of 300 mg every 6 months following the first initial dose

[0021] In examples of any of the methods provided herein, the subject is acetylcholine receptor antibody positive (AChR-Ab+). In examples of any of the methods provided herein, the subject is muscle-specific kinase-antibody positive (MuSK-Ab+). In examples of any of the methods provided herein, the subject is acetylcholine receptor antibody positive (AChR-Ab+) and muscle-specific kinase-antibody positive (MuSK-Ab+). In examples of any of the methods provided herein, administration of the VIB551 reduces the frequency of MG exacerbations. In examples of any of the methods provided herein, administration of the VIB551 reduces fatigue.

[0022] In examples, the subject is further administered one or more additional therapies. In examples in which the subject is further administered one or more additional therapies, the one or more additional therapies are one or more standard of care therapies. In examples, examples in which the one or more additional therapies are one or more standard of care therapies, the additional therapy is a corticosteroid, which may be prednisone. In examples in which the subject is further administered one or more additional therapies, the one or more additional therapies are one or more of azathioprine, mycophenolate mofetil, mycophenolic acid, or tacrolimus.

[0023] In examples of any of the methods provided herein, the VIB551 has a heavy chain variable region that includes the amino acid sequence of SEQ ID NO:1 and a light chain

variable region that includes the amino acid of SEQ ID NO:2. In examples of any of the methods provided herein, the administration of the VIB551 treats the MG. In examples of any of the methods provided herein, the MG is refractory MG.

[0023a] In one aspect, the disclosure provides use of an antibody in the manufacture of a medicament for the treatment of myasthenia gravis (MG) in a subject in need of treatment for MG, the antibody comprising: Complementarity-Determining Regions (CDRs) HCDR1, HCDR2, HCDR3, LCDR1, LCDR2, and LCDR3, wherein the HCDR1, HCDR2, HCDR3, LCDR1, LCDR2, and LCDR3 comprise the amino acid sequences of SEQ ID NOs: 3, 4, 5, 6, 7 , and 8, respectively, and wherein the antibody is to be administered intravenously at a dose of from about 250 mg to about 350 mg every 6 months.

[0023b] In another aspect, the disclosure provides use of an antibody in the manufacture of a medicament for the treatment of myasthenia gravis (MG), the antibody comprising: a heavy chain variable region (VH) comprising the amino acid of SEQ ID NO:1, and a light chain variable region (VL) comprising the amino acid of SEQ ID NO: 2, wherein the antibody is to be administered intravenously at a dose of 300 mg every 6 months, thereby treating the MG.

BRIEF DESCRIPTION OF FIGURES

[0024] The novel features of the disclosure are set forth with particularity in the appended claims. A better understanding of the features and advantages of the present disclosure will be obtained by reference to the following detailed description that sets forth illustrative embodiments, in which the principles of the disclosure are utilized, and the accompanying drawings of which:

[0025] **FIG. 1** depicts an exemplary study flow diagram.

[0026] **FIG. 2** depicts the design scheme for AChR-Ab+ population. AChR-Ab+ = the population of subjects who are antibody positive for the acetylcholine receptor; CS = corticosteroid; D = Day; IV = intravenous(ly); MG = Myasthenia Gravis; MG-ADL = Myasthenia Gravis Activity of Daily Living score; MGFA = Myasthenia Gravis Foundation of America; OLP = Open-Label Period; QMG = Quantitative Myasthenia Gravis scale; RCP = Randomized Controlled Period; SoC = Standard of Care.

[0027] **FIG. 3** depicts the design scheme for MuSK-Ab+ population. CS = corticosteroid; D = Day; IV = intravenous(ly); MG = Myasthenia Gravis; MG-ADL = Myasthenia Gravis Activity of Daily Living score; MGFA = Myasthenia Gravis Foundation of America classification; MuSK-Ab+ = the population of subjects who are antibody positive for muscle-specific kinase; OLP = Open-Label Period; QMG = Quantitative Myasthenia Gravis scale; RCP = Randomized Controlled Period; SoC = standard of care.

[0028] **FIG. 4** depicts the VH (SEQ ID NO:1) and VL (SEQ ID NO:2) amino acid sequences of the VIB551 antibody.

[0029] **FIG. 5A** - **FIG. 5C** are graphics illustrating a possible Inebilizumab mechanism of action in MG. **FIG. 5A** shows that Inebilizumab targets the CD19 receptor, consequently inhibiting autoantibody production and depleting circulating B cells. **FIG. 5B** shows that autoantibodies disrupt the normal architecture and functioning of the neuromuscular junction in MG. **FIG. 5C** shows that Anti-AChR autoantibodies drive MG by: 1) complement activation and muscle membrane damage; 2) AChR crosslinking and internalization; and 3) direct