the polyclonal IgG can occur intranasally, subcutaneously, orally, intra-arterially or intravenously. In some embodiments, an anti-inflammatory agent is co-administered with the polyclonal IgG to the mammal. The anti-inflammatory agent may be selected from an adrenocorticotropic hormone, a corticosteroid, an interferon, glatiramer acetate, or a non-steroidal anti-inflammatory drug.

[0012] The disclosed polyclonal IgG may be administered weekly, biweekly, or monthly at a dose of about 0.05 to 5 g per kg of patient body weight or about 0.5 to 2 g per kg of patient body weight.

[0013] In one ~~aspect~~embodiment, there is provided methods of promoting myelination of a peripheral nerve cell by a Schwann cell by contacting the Schwann cell with an amount of polyclonal IgG sufficient to promote myelination of said peripheral nerve cell by the Schwann cell.

[0014] In another aspect , there is provided methods of promoting the differentiation of an immature Schwann cell into a myelinating state by contacting said Schwann cell with polyclonal IgG in an amount sufficient to induce the Schwann cell differentiation.

[0015] In yet another aspect, there is provided methods of promoting myelin production by a Schwann cell comprising contacting said Schwann cell with an amount of polyclonal IgG sufficient to upregulate MBP gene.

[0016] In a further aspect, there is provided methods of culturing mammalian nervous tissue which comprises axons by contacting the tissue in culture with an effective amount of Schwann cells and an effective amount of polyclonal IgG, whereby the contacting of Schwann cells with polyclonal IgG induces upregulation of MBP gene.

[0017] In another embodiment, there is provided methods of treating a peripheral nerve injury in a mammal by: transplanting nerve cells to a site of the peripheral nerve injury; and contacting the nerve cells with a composition comprising Schwann cells and polyclonal IgG.

[0018] In the methods described herein, the polyclonal IgG can be given through one or more routes of administration, such as intramuscularly, intradermally, subcutaneously, buccally, orally, intranasally, or intra-arterially or intravenously to an individual in need of such therapy. The individual may be a human or domesticated animal. In some embodiments, the polyclonal IgG is derived from pooled human serum.

[0019] In some embodiments, the polyclonal IgG Is co-administered with an anti-inflammatory agent to mammal in need of such therapy. The anti-inflammatory agent may

be selected from an adrenocorticotropic hormone, a corticosteroid, an interferon, glatiramer acetate, or a non-steroidal anti-inflammatory drug.

[0020] In yet another embodiment, there is provided pharmaceutical compositions comprising a pharmaceutically acceptable carrier and an effective amount of polyclonal IgG for treating a non-idiopathic, demyelinating peripheral neuropathy.

[0020a] In one aspect, there is provided the use of a therapeutically effective amount of polyclonal IgG in the manufacture of a medicament for treating a trauma-induced demyelinating peripheral neuropathy in a mammal diagnosed with said neuropathy by promoting myelination of a peripheral nerve cell by a Schwann cell~~, with the proviso that said neuropathy is not an immune-mediated or infection-mediated neuropathy and excludes Guillain-Barré syndrome, chronic demyelinating polyneuropathy and multifocal motor neuropathy~~.

[0020b] ~~In another aspect, t~~There is also disclosed ~~provided~~ the use of a therapeutically effective amount of polyclonal IgG in the manufacture of a medicament for treating peripheral nerve trauma in a mammal with peripheral nerve trauma.

[0020c] ~~In another aspect, t~~There is also ~~provided~~ disclosed the use of a therapeutically effective amount of polyclonal IgG in the manufacture of a medicament for treating toxin-induced peripheral neuropathy in a mammal diagnosed with said neuropathy, wherein said neuropathy is not infection-mediated.

[0020d] ~~In still another aspect, t~~There is also disclosed ~~provided~~ the use of a composition comprising Schwann cells and polyclonal IgG in the manufacture of a medicament for treating a peripheral nerve injury in a mammal, wherein the medicament is administered to nerve cells that have been transplanted to a site of the peripheral nerve injury.

[0020e] Another aspect relates to the use of a therapeutically effective amount of polyclonal IgG in the manufacture of a medicament for treating a trauma-induced demyelinating peripheral neuropathy by promoting myelination of a peripheral nerve cell by a Schwann cell and by inducing Schwann cell differentiation, wherein the polyclonal IgG is to be administered to a mammal diagnosed with said neuropathy.

[0020f] Another aspect relates to the use of a therapeutically effective amount of polyclonal IgG in the manufacture of a medicament for treating a trauma-induced demyelinating peripheral neuropathy by promoting myelination of a peripheral nerve cell by a Schwann cell and by inducing Schwann cell differentiation and by upregulating the myelin basic protein (MBP) gene in a Schwann cell, wherein the polyclonal IgG is to be administered to a mammal diagnosed with said neuropathy.

BRIEF DESCRIPTION OF THE DRAWINGS

[0021] More particular descriptions of the invention are made by reference to certain exemplary embodiments thereof which are illustrated in the appended Figures. These Figures form a part of the specification. It is to be noted, however, that the appended Figures illustrate exemplary embodiments of the invention and therefore are not to be considered limiting in their scope.

[0022] FIG. 1 shows the relative proliferation rates of immature Schwann cells that were exposed to nondialysed (FIG. 1A) and dialysed (FIG. 1B) IVIG/buffer formulations after 2 days as measured by BrdU incorporation assays. These relative proliferation rates were generated based on the number of cells positive for of 5-bromo-2'-deoxyuridine (BrdU) incorporated into cellular DNA during cell proliferation.

[0023] FIG. 2 shows the relative proliferative rates of immature Schwann cells that were exposed to nondialysed (FIG. 2A) and dialysed (FIG. 2B) IVIG/buffer formulations after 2 days as measured using Ki-67 assays. These relative proliferation rates were generated based on the number of cells positive for Ki-67 expression during cell proliferation.

[0024] FIG. 3 shows the levels of P0 (FIG. 3A) and MBP (FIG. 3B) gene expression in immature Schwann cells that were exposed to dialysed IVIG/buffer formulations at 1 day and 3 day time-points.

[0025] FIG. 4 shows the levels of P0 (FIG. 4A) and MBP (FIG. 4B) gene expression in p57kip2 suppressed Schwann cells that were exposed to dialysed IVIG/buffer formulations at the 7 day time-point (9 days suppression).

[0026] FIG. 5 shows the expression levels of CD64 Fc receptor in p57kip2 suppressed Schwann cells as compared to control transfected Schwann cells (without p57kip2 suppression). Neither group of Schwann cells were exposed to IVIG/buffer formulations.

[0027] FIG. 6 shows fluorescent images of p57kip2 suppressed Schwann cells (FIG. 6B) and control transfected cells (FIG. 6A) after stimulation with 20 mg dialyzed IVIG/buffer formulations. The location and length of cellular processes are indicated by the arrows superimposed onto the fluorescent images.

CLAIMS

1. Use of a therapeutically effective amount of polyclonal IgG in the manufacture of a medicament for treating a trauma-induced demyelinating peripheral neuropathy in a mammal diagnosed with said neuropathy by promoting myelination of a peripheral nerve cell by a Schwann cell.

2. The use of claim 1, wherein the mammal is human.

3. The use of any of claims 1-2, wherein the polyclonal IgG is administered locally.

4. The use of claim 3, wherein the polyclonal IgG is administered intramuscularly or intradermally.

5. The use of any of claims 1-2, wherein the polyclonal IgG is administered systemically.

6. The use of claim 5, wherein the polyclonal IgG is administered intranasally, subcutaneously, orally, intra-arterially or intravenously.

7. The use of any of claims 1-6, wherein an anti-inflammatory agent is co-administered with the polyclonal IgG to the mammal.

8. The use of claim 7, wherein the anti-inflammatory agent is adrenocorticotropic hormone, a corticosteroid, an interferon, glatiramer acetate, or a non-steroidal anti-inflammatory drug.

9. The use of any of claims 1-8, wherein the polyclonal IgG is administered weekly.

10. The use of any of claims 1-8, wherein the polyclonal IgG is administered biweekly.

11. The use of any of claims 1-8, wherein the polyclonal IgG is administered monthly.

12. The use of any of claims 1-11, wherein the polyclonal IgG is administered to the mammal at a dose of about 0.05 to 5 g per kg of patient body weight.

13. The use of claim 12, wherein the polyclonal IgG is administered to the mammal at a dose of about 0.5 to 2 g per kg of patient body weight.

14. Use of a therapeutically effective amount of polyclonal IgG in the manufacture of a medicament for treating a trauma-induced demyelinating peripheral neuropathy by promoting myelination of a peripheral nerve cell by a Schwann cell and by inducing Schwann cell differentiation, wherein the polyclonal IgG is to be administered to a mammal diagnosed with said neuropathy.

15. Use of a therapeutically effective amount of polyclonal IgG in the manufacture of a medicament for treating a trauma-induced demyelinating peripheral neuropathy by promoting myelination of a peripheral nerve cell by a Schwann cell and by inducing Schwann cell differentiation and by upregulating the myelin basic protein (MBP) gene in a Schwann cell, wherein the polyclonal IgG is to be administered to a mammal diagnosed with said neuropathy.

16. The use of any of claims 14-15, wherein the mammal is human.

17. The use of any of claims 14-16, wherein the polyclonal IgG is to be administered weekly.

18. The use of any of claims 14-16, wherein the polyclonal IgG is to be administered biweekly.

19. The use of any of claims 14-16, wherein the polyclonal IgG is to be administered monthly.

20. The use of any of claims 14-16, wherein the polyclonal IgG is to be administered to the mammal at a dose of about 0.05 to 5 g per kg of patient body weight.

21. The use of claim 20, wherein the polyclonal IgG is to be administered to the mammal at a dose of about 0.5 to 2 g per kg of patient body weight.

1. Use of a therapeutically effective amount of polyclonal IgG in the manufacture of a medicament for treating a demyelinating peripheral neuropathy in a mammal diagnosed with said neuropathy, with the proviso that said neuropathy is not an immune-mediated or infection-mediated neuropathy and excludes Guillain-Barré syndrome, chronic demyelinating polyneuropathy and multifocal motor neuropathy.

2. The use of claim 1, wherein the mammal is human.

3. The use of any of claims 1-2, wherein the polyclonal IgG is administered locally.

4. The use of claim 3, wherein the polyclonal IgG is administered intramuscularly or intradermally.

5. The use of any of claims 1-2, wherein the polyclonal IgG is administered systemically.

6. The use of claim 5, wherein the polyclonal IgG is administered intranasally, subcutaneously, orally, intra-arterially or intravenously.

7. The use of any of claims 1-6, wherein an anti-inflammatory agent is co-administered with the polyclonal IgG to the mammal.

8. The use of claim 7, wherein the anti-inflammatory agent is adrenocorticotropic hormone, a corticosteroid, an interferon, glatiramer acetate, or a non-steroidal anti-inflammatory drug.

9. The use of any of claims 1-8, wherein the demyelinating peripheral neuropathy is selected from a trauma-induced neuropathy, a toxin-induced neuropathy, an inherited neuropathy, and a neuropathy induced by a metabolic disease.

10. The use of claim 9, wherein the peripheral neuropathy is a trauma-induced neuropathy.

11. The use of claim 9, wherein the peripheral neuropathy is a toxin-induced neuropathy.

12. The use of claim 9, wherein the peripheral neuropathy is an inherited neuropathy.

13. The use of claim 9, wherein the peripheral neuropathy is induced by a metabolic disease.

~~14. The use of claim 13, wherein the peripheral neuropathy is diabetic neuropathy.~~

15. The use of any of claims 1-8, wherein the demyelinating peripheral neuropathy is motor neuropathy.

16. The use of any of claims 1-8, wherein the demyelinating peripheral neuropathy is sensory neuropathy.

17. The use of any of claims 1-8, wherein the demyelinating peripheral neuropathy is sensorimotor neuropathy.

18. The use of any of claims 1-8, wherein the demyelinating peripheral neuropathy is autonomic neuron neuropathy.

19. The use of any of claims 1-18, wherein the polyclonal IgG is administered weekly.

20. The use of any of claims 1-18, wherein the polyclonal IgG is administered biweekly.

21. The use of any of claims 1-18, wherein the polyclonal IgG is administered monthly.

22. The use of any of claims 1-21, wherein the polyclonal IgG is administered to the mammal at a dose of about 0.05 to 5 g per kg of patient body weight.

23. The use of claim 22, wherein the polyclonal IgG is administered to the mammal at a dose of about 0.5 to 2 g per kg of patient body weight.

24. Use of a therapeutically effective amount of polyclonal IgG in the manufacture of a medicament for treating peripheral nerve trauma in a mammal with peripheral nerve trauma.

25. The use of claim 24, wherein the polyclonal IgG is administered locally.

26. The use of claim 23, wherein the polyclonal IgG is administered intramuscularly or intradermally.

27. The use of claim 24, wherein the polyclonal IgG is administered systemically.

28. The use of claim 27, wherein the polyclonal IgG is administered intranasally, orally, intra-arterially, subcutaneously, or intravenously.

29. The use of any of claims 24-28, wherein the polyclonal IgG is administered to the mammal at a dose of about 0.05 to 5 g per kg of patient body weight.

30. The use of claim 29, wherein the polyclonal IgG is administered to the mammal at a dose of about 0.5 to 2 g per kg of patient body weight.

31. The use of any of claims 24-30, wherein an anti-inflammatory agent is co-administered with the polyclonal IgG to the mammal.

32. The use of claim 31, wherein the anti-inflammatory agent is adrenocorticotropic hormone, a corticosteroid, an interferon, glatiramer acetate, or a non-steroidal anti-inflammatory drug.

33. The use of any of claims 24-32, wherein the mammal is human.

34. Use of a therapeutically effective amount of polyclonal IgG in the manufacture of a medicament for treating toxin-induced peripheral neuropathy in a mammal diagnosed with said neuropathy, wherein said neuropathy is not infection-mediated.

35. The use of claim 34, wherein the polyclonal IgG is administered locally.

36. The use of claim 35, wherein the polyclonal IgG is administered intramuscularly or intradermally.

37. The use of claim 34, wherein the polyclonal IgG is administered systemically.

38. The use of claim 37, wherein the polyclonal IgG is administered intranasally, orally, intra-arterially, subcutaneously, or intravenously.

39. The use of any of claims 34-38, wherein the polyclonal IgG is administered to the mammal at a dose of about 0.05 to 5 g per kg of patient body weight.

40. The use of claim 39, wherein the polyclonal IgG is administered to the mammal at a dose of about 0.5 to 2 g per kg of patient body weight.

41. The use of any of claims 34-40, wherein an anti-inflammatory agent is co-administered with the polyclonal IgG to the mammal.

42. The use of claim 41, wherein the anti-inflammatory agent is adrenocorticotropic hormone, a corticosteroid, an interferon, glatiramer acetate, or a non-steroidal anti-inflammatory drug.

43. The use of any of claims 34-42, wherein the mammal is human.

44. ~~A method of promoting myelination of a peripheral nerve cell by a Schwann cell~~ comprising contacting said Schwann cell with an amount of polyclonal IgG sufficient to promote myelination of said peripheral nerve cell by the Schwann cell.

45. A method of promoting the differentiation of an immature Schwann cell into a myelinating state comprising contacting said Schwann cell with polyclonal IgG in an amount sufficient to induce the Schwann cell differentiation.

46. A method of promoting the production of myelin by a Schwann cell comprising contacting said Schwann cell with an amount of polyclonal IgG sufficient to upregulate MBP genes.

47. A method of culturing mammalian nervous tissue which comprises axons, said method comprising contacting the tissue in culture with an effective amount of Schwann cells and an effective amount of polyclonal IgG, whereby the contacting of Schwann cells with polyclonal IgG induces upregulation of MBP genes.

48. Use of a composition comprising Schwann cells and polyclonal IgG in the manufacture of a medicament for treating a peripheral nerve injury in a mammal wherein

the medicament is administered to nerve cells that have been transplanted to a site of the peripheral nerve injury.

49. The use of claim 48, wherein the mammal is human.