CLAIMS

1. ~~A method~~ Use of ~~treating~~an autologous genetically modified cells in the manufacture of a medicament for the treatment of a type VII collagen (C7) deficiency in a patient suffering from ~~a type VII collagen (C7)~~ the deficiency ~~comprising~~

~~obtaining~~, wherein the cells, having been obtained from the dermis or epidermis of the C7-deficient patient,

~~contacting said cells~~have been contacted with a transducing lentiviral vector comprising a nucleotide sequence encoding COL7A1 or a functional variant thereof to form ~~an~~the autologous genetically modified ~~cell~~cells having a vector copy number wherein said lentiviral vector has a transducing vector copy number in the range of 0.1 to 5.0 copies per cell~~,~~; and the said autologous genetically modified cells have been cultured; wherein an effective amount of the genetically modified cells is to be administered to the C7-deficient patient, and wherein the transducing lentiviral vector further comprises (a) a modified 5' long terminal repeat (LTR), wherein the promoter of the modified 5' LTR is a cytomegalovirus promoter, (b) at least one lentiviral central polypurine tract element, and (c) a modified 3' LTR, wherein the modified 3' LTR comprises a deletion relative to the wild-type 3' LTR, wherein a hepatitis virus post-transcriptional regulatory element (PRE) has been deleted.

~~culturing said autologous genetically modified cell,~~

~~and administering an effective amount of the genetically modified cells to the C7-deficient patient.~~

2. ~~The method~~use of claim 1, wherein the cells are selected from fibroblasts and keratinocytes.

3. ~~The method of claim 2, wherein the cells are fibroblasts.~~

4. ~~The method~~use of claim 1, wherein the C7 ~~-~~deficiency is dystrophic epidermolysis bullosa.

~~5.~~

4. The ~~method~~use of claim 4~~1~~, wherein the C7-deficiency is a dystrophic epidermolysis bullosa ~~is selected from~~, recessive dystrophic epidermolysis bullosa (RDEB~~) and~~), dominant dystrophic epidermolysis bullosa (DDEB~~).~~

~~6. The method of claim 5, wherein the C7-deficiency is RDEB.~~

~~7. The method of claim 6, wherein the RDEB is a subtype selected from the group consisting of~~), Hallopeau-Siemens, non-Hallopeau-Siemens RDEB, RDEB inversa, pretibial RDEB, acral ~~RDEV, and~~RDEB, or RDEB centripetalis.

~~8. The method of claim 5, wherein the C7-deficiency is DDEB.~~

~~9. The method~~

5. The use of claim 1, wherein said transducing lentiviral vector is in the form of a lentiviral vector particle.

~~10.~~

6. The ~~method~~use of claim ~~9~~1 or claim 2, wherein the genetically modified cells are to be administered to the patient by injection, topically, orally, or embedded in a biocompatible matrix.

7. An autologous genetically modified fibroblast from a patient suffering from a Type VII collagen deficiency transduced with a lentiviral vector particle comprising a functional *COL7A1* gene or a functional variant thereof, and expressing type VII collagen, wherein said lentiviral vector particle has a transducing vector copy number in the range of 0.1 to 5.0 copies per cell, wherein said transducing lentiviral vector particle is constructed from a transfer lentiviral vector comprising (a) a modified 5' long terminal repeat in LTR, wherein the promoter of the ~~modified~~modified 5' LTR is a cytomegalovirus promoter, (b) a functional *COL7A1* gene, (c) at least one lentiviral central polypurine tract, and (d) a modified 3' LTR, wherein the modified 3' LTR comprises a deletion relative to the wild-type 3' LTR, wherein a hepatitis virus post-transcriptional regulatory element (PRE) has been deleted, wherein the *COL7A1* gene or the functional variant thereof is incorporated into the cells to form genetically ~~modified~~modified cells having a functional *COL7A1* gene.

~~11. The method of claim 10, wherein there are at least two lentiviral central polypurine tract elements.~~

8.

12. The method of claim 10, wherein the deleted PRE is a woodchuck hepatitis virus post-transcriptional regulatory element (WPRE).

13. The method of claim 9, wherein transducing lentiviral vector particle is constructed from a lentiviral expression plasmid vector selected from the group consisting of pSMPUW and pFUGW.

14. The method of claim 13, wherein said lentiviral expression plasmid vector is pFUGW.

15. The method of claim 9, wherein said transducing lentiviral vector particle is INXN-2004.

16. The method of claim 9, wherein said transducing lentiviral vector particle is INXN-2002.

17. The method of claim 1, wherein the genetically modified fibroblasts are administered to the patient by injection, topically, orally, or embedded in a biocompatible matrix.

18. The method of claim 17, wherein the genetically modified fibroblasts are administered by injection.

19. The method of claim 18, wherein the genetically modified cells are intradermally injected into the patient.

20. The method of claim 17, wherein the genetically modified fibroblasts are encapsulated in a polymer capsule.

21. The method of claim 17, wherein the genetically modified fibroblasts are embedded in a collagen matrix.

22. The method of claim 17, wherein the genetically modified fibroblasts are embedded in a hydrogel or mesh.

23. An autologous genetically modified fibroblast from a patient suffering from a Type VII collagen deficiency transduced with a lentiviral vector particle comprising a functional COL7A1 gene or a functional variant thereof, and expresses type VII collagen, wherein said lentiviral vector particle has a transducing vector copy number in the range of 0.1 to 5.0 copies per cell.

24. An autologous genetically modified fibroblast from a patient suffering from a recessive dystrophic epidermolysis bullosa transduced with a lentiviral vector particle comprising a functional COL7A1 gene or a functional variant thereof, and expresses type VII collagen, wherein said lentiviral vector particle has a transducing vector copy number in the range of 0.1 to 5.0 copies per cell.

~~25. An autologous genetically modified fibroblast from a patient suffering from a dominant dystrophic epidermolysis bullosa transduced with a lentiviral vector particle comprising a functional COL7A1 gene or a functional variant thereof, and expresses type VII collagen, wherein said lentiviral vector particle has a transducing vector copy number in the range of 0.1 to 5.0 copies per cell.~~

~~26.~~ A self-inactivating lentiviral vector formed from a transfer vector comprising (a) a modified 5' long terminal in LTR, wherein the promoter of the modified 5' LTR is a cytomegalovirus promoter, (b) the *COL7A1* gene or a functional variant thereof, (c) at least one lentiviral central polypurine tract element, and (d) a modified 3' LTR, wherein the modified 3' LTR comprises a deletion relative to the wild-type 3' LTR, and, wherein a hepatitis virus post-transcriptional regulatory element (PRE) has been deleted~~.~~, wherein the deleted PRE is a woodchuck post-transcriptional regulatory element PRE (WPRE).

~~27. The~~

9. A transfer vector ~~of claim 26, wherein~~comprising the ~~vector comprises~~ amino acid sequence set forth in SEQ ID NO: 34.

10. A lentiviral vector particle comprising the self-inactivating lentiviral vector of claim 8 or the transfer vector of claim 9.

11. A lentiviral vector particle constructed from a lentiviral expression plasmid comprising (a) a modified 5' long terminal repeat in LTR, wherein the promoter of the modified 5' LTR is a cytomegalovirus promoter, (b) a functional *COL7A1* gene, (c) at least ~~two~~one lentiviral central polypurine tract ~~elements.~~

~~28. The vector of claim 26, wherein the deleted PRE is a woodchuck hepatitis virus post-transcriptional regulatory element (WPRE).~~

~~29. The vector of claim 26, wherein the vector comprises (a) a modified 5' long terminal in LTR, wherein the promoter of the modified 5' LTR is a cytomegalovirus promoter, (b) the COL7A1 gene, (c) two lentiviral central polypurine tract elements, (d) further comprising a WPRE~~, and (~~e~~d) a modified 3' LTR, wherein the modified 3' LTR comprises a deletion relative to the wild-type 3' LTR, wherein a hepatitis virus post-transcriptional regulatory element (PRE) has been deleted.

12.30. The vector of claim 26, wherein the vector is designated INXN-2004.

31. The vector of claim 26, wherein the vector is designated INXN-2002.

32. A transfer vector designated IGE-308.

33. A lentiviral vector particle designated INXN-2004.

34. A lentiviral vector particle designated INXN-2002.

35. A stable virus packaging cell line producing the INXN-2002 lentiviral vector particle.

36. A stable virus packaging cell line producing the INXN-2004 lentiviral vector particle of claim 10 or claim 11.

37.

13. A pharmaceutical composition comprising a fibroblastfibroblast obtained from a patient suffering from RDEB transduced with a lentiviral vector designated INXN-2004 having the sequence of IGE-308.

38. A pharmaceutical composition comprising a fibroblast obtained from a patient suffering from RDEB transduced with a lentiviral vector designated INXN-2002.

39. A pharmaceutical composition comprising a fibroblast obtained from a patient suffering fromor DDEB transduced with athe lentiviral vector designated INXN-2004 having the sequence of IGE-308claim 10 or claim 11.

14.40. A pharmaceutical composition comprising a fibroblast obtained from a patient suffering from DDEB transduced with a lentiviral vector designated INXN-2002.

41. A cell transduced *in vitro* or *ex vivo* with the vector of claim 3310 or claim 11.

15.42. A cell transduced in vitro or ex vivo with the vector of claim 34.

43. A method of An autologous population of cells for use in treating a patient suffering from pseudosyndactyly comprising administering , wherein said autologous population of cells is to said patient an autologous population of cells be obtained from said patient and transduced with a lentiviral vector particle comprising a functional *COL7A1* gene or a functional variant thereof, and expressing type VII collagen, wherein said lentiviral vector particle has a transducing vector copy number in the range of 0.1 to 5.0 copies

per cell, wherein said transducing lentiviral vector particle is constructed from a transfer lentiviral vector comprising (a) a modified 5' long terminal repeat in LTR, wherein the promoter of the modified 5' LTR is a cytomegalovirus promoter, (b) a functional *COL7A1* gene, (c) at least one lentiviral central polypurine tract, and (d) a modified 3' LTR, wherein the modified 3' LTR comprises a deletion relative to the wild-type 3' LTR, wherein a hepatitis virus post-transcriptional regulatory element (PRE) has been deleted, wherein the *COL7A1* gene or the functional variant thereof is incorporated into the cells to form genetically modified cells having a functional *COL7A1* gene.

16.~~44. The method of claim 43, wherein the autologous population of cells is selected from keratinocytes and fibroblasts.~~

~~45. The method of claim 43, wherein the autologous population of cells is fibroblasts. 46. The method of claim 43, wherein administration of said cells to the patient is by injection, topically, orally, or embedded in a biocompatible matrix.~~

~~47. A method of treating, inhibiting or reducing the blistering of a dystrophic epidermolysis bullosa (DEB) patient comprising administering to said patient an autologous population of cells obtained from said patient transduced with a lentiviral vector particle comprising a functional COL7A1 gene or a functional variant thereof, and expressing type VII collagen, wherein said lentiviral vector particle has a transducing vector copy number in the range of 0.1 to 5.0 copies per cell.~~

~~48. The method of claim 47, wherein the autologous population of cells is selected from keratinocytes and fibroblasts.~~

~~49. The method of claim 48, wherein the autologous population of cells is fibroblasts. 50. The method of claim 47, wherein administration of said cells to the patient is by injection, topically, orally, or embedded in a biocompatible matrix.~~

~~51. The method of claim 47, wherein the patient suffers from recessive dystrophic epidermolysis bullosa.~~

~~52. The method of claim 47, wherein the patient suffers from dominant dystrophic epidermolysis bullosa.~~

53. A method of treating lesions in patients suffering from recessive dystrophic epidermolysis bullosa comprising administering to said patient an autologous population of cells obtained from said patient transduced with a lentiviral vector particle comprising a functional COL7A1 gene or a functional variant thereof, and expressing type VII collagen, wherein said lentiviral vector particle has a transducing vector copy number in the range of 0.1 to 5.0 copies per cell.

54. The method of claim 53, wherein the autologous population of cells is selected from keratinocytes and fibroblasts.

55. The method of claim 53, wherein the autologous population of cells is fibroblasts. 56. The method of claim 53, wherein administration of said cells to the patient is by injection, topically, orally, or embedded in a biocompatible matrix.

57. The method of claim 53, wherein the lesions are present in oral mucosa.

58. The method of claim 53, wherein the lesions are present in the gastrointestinal tract.

59. An isolated population of genetically modified fibroblasts autologous to a patient suffering from a Type VII collagen deficiency transduced with a lentiviral vector particle comprising a functional COL7A1 gene or a functional variant thereof, and expresses type VII collagen, wherein said lentiviral vector particle has a transducing vector copy number in the range of 0.1 to 5.0 copies per cell.

60. The population of claim 59, wherein the lentiviral vector particle is INXN-2002. 61. The population of claim 59, wherein the lentiviral vector particle is INXN-2004. 62. An isolated population of genetically modified fibroblast autologous to a patient suffering from a recessive dystrophic epidermolysis bullosa transduced with a lentiviral vector particle comprising a functional COL7A1 gene or a functional variant thereof, and expresses type VII collagen, wherein said lentiviral vector particle has a transducing vector copy number in the range of 0.1 to 5.0 copies per cell.

63. The population of claim 62, wherein the lentiviral vector particle is INXN-2002. 64. The population of claim 62, wherein the lentiviral vector particle is INXN-2004. 65. An isolated population of genetically modified fibroblasts autologous to a patient suffering from a dominant dystrophic epidermolysis bullase transduced with a lentiviral vector particle comprising a functional COL7A1 gene or a functional variant thereof, and expresses type

~~VII collagen, wherein said lentiviral vector particle has a transducing vector copy number in the range of 0.1 to 5.0 copies per cell.~~

~~66. The population of claim 65, wherein the lentiviral vector particle is INXN-2002. 67. The population of claim 65, wherein the lentiviral vector particle is INXN-2004. 68.~~ A method of making an isolated population of Type VII collagen (C7) expressing genetically modified cells autologous to a patient suffering from C7 deficiency comprising ~~harvesting~~: contacting cells, obtained from the dermis or epidermis of the C7-~~-~~deficient patient, ~~contacting said cells~~ with a transducing lentiviral vector particle comprising the *COL7A1* gene or a functional variant thereof to form ~~an~~ autologous genetically modified ~~cell~~cells comprising the *COL7A1* gene having a vector copy number of wherein said lentiviral vector particle has a transducing vector copy number in the range of 0.1 to 5.0 copies per cell, and culturing said autologous genetically modified ~~cell to obtain an isolated population of C7 expressing genetically modified cells~~cells to obtain an isolated population of C7 expressing genetically modified cells, wherein said transducing lentiviral vector particle is constructed from a transfer lentiviral vector comprising (a) a modified 5' long terminal repeat in LTR, wherein the promoter of the modified 5' LTR is a cytomegalovirus promoter, (b) a functional *COL7A1* gene, (c) at least one lentiviral central polypurine tract, and (d) a modified 3' LTR, wherein the modified 3' LTR comprises a deletion relative to the wild-type 3' LTR, wherein a hepatitis virus post-transcriptional regulatory element (PRE) has been deleted, wherein the *COL7A1* gene or the functional variant thereof is incorporated into the cells to form genetically modified cells having a functional *COL7A1* gene.

17.~~69. The method of claim 68, wherein the cells are selected from fibroblasts and keratinocytes.~~

~~70. The method of claim 69, wherein the cells are fibroblasts.~~

~~71. The method of claim 68, wherein the C7-deficiency is dystrophic epidermolysis bullosa.~~

~~72. The method of claim 71, wherein the dystrophic epidermolysis bullosa is selected from recessive dystrophic epidermolysis bullosa (RDEB) and dominant dystrophic epidermolysis bullosa (DDEB).~~

~~73. The method of claim 71, wherein the C7-deficiency is RDEB.~~

~~74. The method of claim 73, wherein the RDEB is a subtype selected from the group consisting of Hallopeau-Siemens, non-Hallopeau-Siemens RDEB, RDEB inversa, pretibial RDEB, acral RDEV, and RDEB centripetalis.~~

~~75. The method of claim 71, wherein the C7-deficiency is DDEB.~~

~~76. The method of claim 68, wherein the transducing lentiviral vector particle is INXN-2002.~~

~~77. The method of claim 68, wherein the transducing lentiviral vector particle is INXN-2004.~~

~~78.~~ An isolated population of Type VII collagen (C7)-expressing genetically modified cells produced by the method of claim ~~68~~16.

18.~~79. An isolated population of Type VII collagen (C7)-expressing genetically modified cells produced by the method of claim 76.~~

~~80. An isolated population of Type VII collagen (C7)-expressing genetically modified cells produced by the method of claim 77.~~

~~81.~~ A pharmaceutical formulation comprising the isolated population of claim ~~78.~~ 17.

19.~~82. A pharmaceutical formulation comprising the isolated population of claim 79.~~

~~83. A pharmaceutical formulation comprising the isolated population of claim 80.~~

~~84.~~ A method of increasing the integrated transgene copy number per cell in genetically modified human dermal fibroblasts or keratinocytes comprising

(a) contacting a transducing lentiviral vector comprising a nucleotide sequence encoding a *COL7A1* gene or a functional variant thereof with a human dermal ~~fibroblast or keratinocyte obtained from a C7-deficient patient to form a transduction composition, and subjecting said transduction composition to spinoculation to form transduced human dermal fibroblast or keratinocyte, wherein the integrated transgene copy number of the transduced human dermal fibroblasts or keratinocytes is higher relative to a transduction composition not subjected to spinoculation.~~

~~85. The method of claim 84, wherein the transduced human dermal fibroblasts are contacted to a second transducing lentiviral vector to form a second tranduscrion composition.~~

~~86. The method of claim 85, wherein the second transduction composition is subjected to spinoculation.~~

~~87. The method of claim 84, wherein the transduced human dermal fibroblasts have at least a 1-fold greater integrated transgene copy number per cell relative to a transduction composition not subjected to spinoculation.~~

~~88. The method of claim 84, wherein the transduced human dermal fibroblasts have at least a 2-fold greater integrated transgene copy number per cell relative to a transduction composition not subjected to spinoculation.~~

~~88. The method of claim 86, wherein the transduced human dermal fibroblasts have at least a 5-fold greater integrated transgene copy number per cell relative to a transduction composition not subjected to spinoculation or a second transduction.~~

~~88. The method of claim 86, wherein the transduced human dermal fibroblasts have at least a 10-fold greater integrated transgene copy number per cell relative to a transduction composition not subjected to spinoculation or a second transduction.~~

~~88. The method of claim 86, wherein the transduced human dermal fibroblasts have at least a 20-fold greater integrated transgene copy number per cell relative to a transduction composition not subjected to spinoculation or a second transduction.~~

~~88. The method of claim 86, wherein the transduced human dermal fibroblasts have at least a 27-fold greater integrated transgene copy number per cell relative to a transduction composition not subjected to spinoculation or a second transduction.~~

~~89. The method of claim 84, wherein the transduced human dermal fibroblasts have an integrated transgene copy number per cell of at least 0.05.~~

~~90. The method of claim 84, wherein the transduced human dermal fibroblasts have an integrated transgene copy number per cell of at least 0.09.~~

~~91. The method of claim 85, wherein the transduced human dermal fibroblasts have an integrated transgene copy number per cell of at least 0.41.~~

~~92. The method of claim 86, wherein the transduced human dermal fibroblasts have an integrated transgene copy number per cell of at least 0.74.~~

~~93. The method of claim 84, wherein the transduced human dermal fibroblasts have an integrated transgene copy number per cell of at between about 0.1 to about 5.~~

~~94. The method of claim 84, wherein the transduced human dermal fibroblasts have an integrated transgene copy number per cell of between about 0.1 to about 1.~~

~~95. The method of claim 84, wherein the transduced human dermal fibroblasts have an integrated transgene copy number per cell of between about 0.4 to about 1.~~

~~96. The method of claim 84, wherein the transduced human dermal fibroblasts have an integrated transgene copy number per cell of between about 0.4 to about 0.75.~~

~~100. The method of claim 84, wherein the transducing lentiviral vector is in the form of a lentiviral vector particle.~~

~~101. The methd of claim 100, wherein the transducing lentiviral vector particle is INXN-2002.~~

~~102. The method of claim 100, wherein the transducing lentiviral vector particle is INXN-2004.~~

~~103. The method of claim 86, wherein the transduced human dermal fibroblasts or keratinocytes have an increased expression of C7 relative to transduced human dermal fibroblasts or keratinocytes not subjected to spinoculation or a second transduction. 104. The method of claim 86, wherein the transduced human dermal fibroblasts or keratinocytes have at least a 100-fold increased expression of C7 relative to transduced human dermal fibroblasts or keratinocytes not subjected to spinoculation or a second transduction.~~

~~105. The method of claim 86, wherein the transduced human dermal fibroblasts or keratinocytes have at least a 200-fold increased expression relative to transduced human dermal fibroblasts or keratinocytes not subjected to spinoculation or a second transduction.~~

~~106. A method of increasing the integrated transgene copy number per cell in genetically modified human dermal fibroblasts or keratinocytes comprising~~

~~(a) contacting a transducing lentiviral vector comprising a nucleotide sequence encoding a COL7A1 gene or a functional variant thereof with a human dermal fibroblast~~fibroblast or keratinocyte obtained from a C7-deficient patient to form a first transduction composition~~,~~;

(b) ~~-~~ subjecting said first transduction composition to spinoculation to form transduced human dermal ~~fibroblast~~fibroblast or keratinocyte~~,~~;

(c) ~~-~~ contacting the transduced human dermal ~~fibroblasts~~fibroblasts or keratinocytes of step (b) with a second transducing lentiviral vector to form a second transduction composition; and

(d) ~~-~~ subjecting said ~~first~~first transduction composition to spinoculation to form transduced human dermal ~~fibroblast~~fibroblast or keratinocyte,

wherein the integrated transgene copy number of the transduced human dermal ~~fibroblasts~~fibroblasts or keratinocytes is at least 5, 10, 15, 20, 25, 27, 28, 29, 30, 35, 40, 45, or 50- fold higher relative to transduced human dermal ~~fibroblasts~~fibroblasts or keratinocytes not subjected to spinoculation or a second transduction step~~.~~

~~107. The method of claim 106,~~ , and wherein the ~~first and second tranducing~~transducing lentiviral vector ~~are INXN-2004.~~

~~108. The method of claim 106,~~further comprises (a) a modified 5' long terminal repeat (LTR), wherein the ~~cells are human dermal fibroblasts.~~

~~109. The method of claim 106, wherein the integrated transgene copy number of the transduced human dermal fibroblasts or keratinocytes~~promoter of the modified 5' LTR is a cytomegalovirus promoter, (b) at least ~~5-fold higher~~one lentiviral central polypurine tract element, and (c) a modified 3' LTR, wherein the modified 3' LTR comprises a deletion relative to ~~transduced human dermal fibroblasts or keratinocytes not subjected to spinoculation or a second transduction step.~~

~~110. The method of claim 106~~the wild-type 3' LTR, wherein ~~the integrated transgene copy number of the transduced human dermal fibroblasts or keratinocytes is at least 10-fold higher relative to transduced human dermal fibroblasts or keratinocytes not subjected to spinoculation or a second transduction step~~a hepatitis virus post-transcriptional regulatory element (PRE) has been deleted.

20. Autologous genetically modified cells for use in a method for treating a patient suffering from a type VII collagen (C7) deficiency, wherein said method comprises:

(i) contacting cells, obtained from the dermis or epidermis of the C7-deficient patient, with a transducing lentiviral vector comprising a nucleotide sequence encoding COL7A1 or a functional variant thereof to form the autologous genetically modified cells having a vector copy number wherein said lentiviral vector has a transducing vector copy number in the range of 0.1 to 5.0 copies per cell; and

(ii) culturing said autologous genetically modified cells,

wherein an effective amount of the genetically modified cells to the C7-deficient patient are to be administered to the patient, and wherein the transducing lentiviral vector further comprises (a) a modified 5' long terminal repeat (LTR), wherein the promoter of the modified 5' LTR is a cytomegalovirus promoter, (b) at least one lentiviral central polypurine tract element, and (c) a modified 3' LTR, wherein the modified 3' LTR comprises a deletion relative to the wild-type 3' LTR, wherein a hepatitis virus post-transcriptional regulatory element (PRE) has been deleted.

21. ~~111. The method of claim 106, wherein the integrated transgene copy number of the transduced human dermal fibroblasts or keratinocytes is at least 27-fold higher relative to transduced human dermal fibroblasts or keratinocytes not subjected to spinoculation or a second transduction step.~~

~~112. The method of claim 106, wherein the integrated transgene copy number of the transduced human dermal fibroblasts or keratinocytes is about 5-fold higher relative to~~

~~transduced human dermal fibroblasts or keratinocytes not subjected to spinoculation or a second transduction step.~~

~~113. The method of claim 106, wherein the integrated transgene copy number of the transduced human dermal fibroblasts or keratinocytes is about 10-fold higher relative to transduced human dermal fibroblasts or keratinocytes not subjected to spinoculation or a second transduction step.~~

~~114. The method of claim 106, wherein the integrated transgene copy number of the transduced human dermal fibroblasts or keratinocytes is about 27-fold higher relative to transduced human dermal fibroblasts or keratinocytes not subjected to spinoculation or a second transduction step.~~

Use of an autologous population of cells in the manufacture of a medicament for the treatment of a patient suffering from pseudosyndactyly, wherein said autologous population of cells is to be obtained from said patient and transduced with a lentiviral vector particle comprising a functional *COL7A1* gene or a functional variant thereof, and expressing type VII collagen, wherein said lentiviral vector particle has a transducing vector copy number in the range of 0.1 to 5.0 copies per cell, wherein said transducing lentiviral vector particle is constructed from a transfer lentiviral vector comprising (a) a modified 5' long terminal repeat in LTR, wherein the promoter of the modified 5' LTR is a cytomegalovirus promoter, (b) a functional *COL7A1* gene, (c) at least one lentiviral central polypurine tract, and (d) a modified 3' LTR, wherein the modified 3' LTR comprises a deletion relative to the wild-type 3' LTR, wherein a hepatitis virus post-transcriptional regulatory element (PRE) has been deleted, wherein the *COL7A1* gene or the functional variant thereof is incorporated into the cells to form genetically modified cells having a functional *COL7A1* gene.