WHAT IS CLAIMED IS:

1. Use of an adeno-associated virus (AAV) expression vector for treating Fabry disease in a human subject in need thereof, wherein the subject has anti-α-GalA neutralizing antibodies prior to the use of the AAV expression vector, wherein the vector comprises an α galactosidase A (α-Gal A) expression cassette, which comprises an apolipoprotein E (APOE) enhancer operably linked to an alpha 1-antitrypsin (hAAT) promoter, a human hemoglobin beta (HBB)-IGG intron, a sequence encoding a signal peptide, an α galactosidase A (α-Gal A) transgene encoding the at least one α-Gal A protein, and a bovine growth hormone poly A signal sequence, and wherein the AAV expression vector is used at a dose of 5×10^{12} vector genomes per kilogram of body weight (vg/kg) to 5×10^{13} vg/kg.

2. Use of an adeno-associated virus (AAV) expression vector for treating Fabry disease in a human subject in need thereof, wherein the subject has anti-α-GalA neutralizing antibodies prior to the use of the AAV expression vector, wherein the vector comprises an α galactosidase A (α-Gal A) expression cassette, which comprises an α galactosidase A (α-Gal A) transgene encoding the at least one α-Gal A protein, wherein the α-GalA transgene comprises the nucleotide sequence as set forth in SEQ ID NO: 5, and wherein the AAV expression vector is used at a dose of 5×10^{12} vector genomes per kilogram of body weight (vg/kg) to 5×10^{13} vg/kg.

3. The use of any one of claims 1 or 2, wherein the α-Gal A expression cassette further comprises a mutated Woodchuck Hepatitis Virus (WHV) Posttranscriptional Regulatory Element (WPRE) sequence, optionally wherein the mutated WPRE sequence comprises a mut6 mutated WPRE sequence.

4. The use of claim 1, wherein the transgene comprises a wild-type α-Gal A sequence or a codon-optimized α-Gal A sequence.

5. The use of claim 1, wherein the signal peptide is an α-GalA signal peptide.

6. The use of claim 3, wherein the APOE enhancer comprises the nucleotide sequence as set forth in SEQ ID NO: 2, the hAAT promotor comprises the nucleotide sequence as set forth in SEQ ID NO: 3, the HBB-IGG intron comprises the nucleotide sequence as set forth in SEQ ID NO: 4, the α-GalA transgene comprises the nucleotide sequence as set forth in SEQ ID NO: 5, the mutated WPRE sequence comprises the nucleotide sequence as set forth in SEQ ID NO: 6, and the bovine growth hormone poly A signal sequence comprises the nucleotide sequence as set forth in SEQ ID NO: 7.

7. The use of claim 3, wherein the APOE enhancer comprises the nucleotide sequence as set forth in SEQ ID NO: 2, the hAAT promotor comprises the nucleotide sequence as set forth in SEQ ID NO: 3, the HBB-IGG intron comprises the nucleotide sequence as set forth in SEQ ID NO: 4, the mutated WPRE sequence comprises the nucleotide sequence as set forth in SEQ ID NO: 6, and the bovine growth hormone poly A signal sequence comprises the nucleotide sequence as set forth in SEQ ID NO: 7.

8. The use of any one of claims 1 to 7, wherein the α-Gal A expression cassette comprises the nucleotide sequence as set forth in SEQ ID NO: 9.

9. The use of any one of claims 1 to 8, wherein the AAV expression vector serotype is AAV2/6.

10. The use of any one of claims 1 to 9, wherein the subject has one or more of the following symptoms: globotriaosylceramide (Gb3) levels above normal, globotriaosylsphingosine (lyso-Gb3) levels above normal, renal disease, cardiac disease, anhidrosis, acroparesthesia, angiokeratoma, gastrointestinal (GI) tract pain, corneal and lenticular opacities, or cerebrovascular disease.

11. The use of any one of claims 1 to 10, wherein the presence of the anti-α-GalA neutralizing antibodies is determined by an enzyme-linked immunosorbent assay (ELISA).

12. The use of any one of claims 1 to 11, wherein a biological sample obtained from the subject exhibits greater than 9.6 % inhibition of α-Galactosidase A activity as measured by a fluorescent enzyme inhibition assay.

13. The use of any one of claims 1 to 12, wherein the α-Gal A protein expressed from the transgene decreases the amount of glycosphingolipids in the subject by at least 2 fold as compared to the amount of glycosphingolipids in the subject prior to the use.

14. The use of any one of claims 1 to 12, wherein the α-Gal A protein expressed from the transgene decreases the amount of glycosphingolipids in the subject by about 10 percent (%), about 11%, about 12%, about 13%, about 14%, about 15%, about 16%, about 17%, about 18%, about 19%, about 20%, about 21%, about 22%, about 23%, about 24%, about 25%, about 26%, about 27%, about 28%, about 29%, about 30%, about 31%, about 32%, about 33%, about 34%, about 35%, about 36%, about 37%, about 38%, about 39%, about 40%, about 41%, about 42%, about 43%, about 44%, about 45%, about 46%, about 47%, about 48%, about 49%, about 50%, about 51%, about 52%, about 53%, about 54%, about 55%, about 56%, about 57%, about 58%, about 59%, about 60%, about 61%, about 62%, about 63%, about 64%, about 65%, about 66%, about 67%, about 68%, about 69%, about 70%, about 71%, about 72%, about 73%, about 74%, about 75%, about 76%, about 77%, about 78%, about 79%, about 80%, about 81%, about 82%, about 83%, about 84%, about 85%, about 86%, about 87%, about 88%, about 89%, about 90%, about 91%, about 92%, about 93%, about 94%, about 95%, about 96%, about 97%, about 98%, about 99%, or about 100 % as compared to the amount of glycosphingolipids in the subject prior to the use.

15. The use of any one of claims 1 to 14, wherein the α-Gal A protein expressed from the transgene decreases the amount of glycosphingolipids in one or more of plasma, liver, heart, kidney, urine, skin, or spleen.

16. The use of any one of claims 1 to 15, wherein the subject is to be administered an immunosuppressant prior to and/or during administration of the AAV expression vector.

17. The use of any one of claims 1 to 15, wherein the subject has not been administered an immunosuppressant prior to and/or during the use of the AAV expression vector.

18. The use of any one of claims 1 to 17, wherein the subject has not been administered a preconditioning treatment prior to the use of the AAV expression vector.

19. The use of any one of claims 1 to 18, wherein the expression of the at least one α-Gal A protein is sustained for at least 3 months, at least 4 months, at least 5 months, at least 6 months, at least 7 months, at least 8 months, at least 9 months, at least 10 months, at least 11 months, at least 12 months, at least 13 months, at least 14 months, at least 15 months, at least 16 months, at least 17 months, at least 18 months, at least 19 months, at least 20 months, at least 21 months, at least 22 months, at least 23 months, or at least 24 months.

20. The use of any one of claims 1 to 19, wherein the subject has been administered with an enzyme replacement therapy (ERT) for Fabry disease prior to the use of the AAV expression vector ("pre-treatment").