We claim:

1. An ex-vivo method for correcting a disease-causing mutant allele of a gene of interest in a primate cell, comprising:

a) introducing a non-naturally occurring targeted nuclease and site-specific nucleotide-binding guide that act together to introduce double-stranded breaks in the mutant allele into the primate cell, wherein:

i) the primate cell is an embryo, with the proviso that the embryo has not been cultured ex-vivo for more than 14 days;

ii) the primate cell is undergoing mitotic cell division;

iii) the primate cell comprises a genome that is heterozygous for the mutant allele, such that the genome comprises one copy of the mutant allele and one copy of a wild-type allele;

iv) single-stranded oligonucleotides homologous to the wild-type allele are not introduced into the primate cell; and

b) allowing the primate cell to activate homology-directed repair of the double-stranded DNA breaks in the mutant allele,

thereby correcting the mutant allele using the normal wild-type allele as a repair template and producing a primate cell that is homozygous for the wild-type allele.

2. The ex-vivo method of claim 1, further comprising generating the embryo prior to step (a).

3. The ex-vivo method of claim 1 or claim 2, wherein the embryo is a one-cell embryo.

4. The ex-vivo method of any one of claims 1-3, further comprising:

selecting a primate oocyte comprising a genome having a mutant allele or a wild-type allele of a gene of interest from a primate species;

fertilizing the primate oocyte with a sperm from the same primate species, wherein the sperm comprises a wild-type allele or a mutant allele of the gene of interest, respectively, thereby forming a one-cell primate embryo, wherein the primate embryo is heterozygous and comprises the one copy of the wild-type allele and the one copy of the mutant allele.

5. The ex-vivo method of claim 4, wherein the targeted nuclease and the site-specific nucleotide-binding guide are introduced into the primate oocyte simultaneously with fertilizing the primate oocyte.

6. The ex-vivo method of claim 4 or claim 5, wherein fertilizing the primate oocyte comprises intracytoplasmic sperm injection (ICSI).

7. The ex-vivo method of any one of claims 4-6, wherein the primate oocyte is at metaphase II when the targeted nuclease and the site-specific nucleotide-binding guide are introduced.

8. The ex-vivo method of any one of claims 1-3, further comprising culturing the embryo to form a multi-cell embryo *in vitro*.

9. The ex-vivo method of claim 8, wherein the multi-cell embryo is not mosaic for cells comprising the mutant allele.

10. The ex-vivo method of any one of claims 1-9, wherein the targeted nuclease is clustered regularly interspaced short palindromic repeats (CRISPR)-associated (Cas)9, zinc finger nuclease (ZFN), or transcription activator-like effector nuclease (TALEN).

11. The ex-vivo method of claim 10, wherein the targeted nuclease is CRISPR-Cas9, and the site-specific nucleotide-binding guide is a nucleic acid guide RNA.

12. The ex-vivo method of any one of claims 1-11, further comprising:

c) assaying for successful correction of the mutant allele.

13. The ex-vivo method of claim 12, wherein assaying for successful correction comprises the use of Sanger sequencing.

14. The ex-vivo method of any one of claims 1-11, further comprising assaying for off-target effects.

15. The ex-vivo method of claim 14, wherein assaying for off-target effects comprises whole-genome sequencing.

16. The ex-vivo method of any one of claims 1-15, wherein the primate is a human.

17. The ex-vivo method of any one of claims 1-16, wherein the mutant allele comprises a deletion or an insertion as compared to the wild-type allele.

18. The ex-vivo method of any one of claims 1-17, wherein the mutant allele comprises a base pair substitution as compared to the wild-type allele.

19. The ex-vivo method of any one of claims 1-18, wherein the mutant allele comprises a frame shift mutation as compared to the wild-type allele.

20. The ex-vivo method of any one of claims 1-19, wherein the gene of interest is myosin binding protein C (MYBPC3), fibroblast growth factor receptor 3 (FGFR3), serpin family A member 1 (SERPINA1), protein kinase D1 (PKD), breast cancer 1 (BRCA1), breast cancer 2 (BRCA2), glycyl-tRNA synthetase (GARS), WNT signaling pathway regulator (APC), cystic fibrosis transmembrane conductance regulator (CFTR), chimerin 1 (CHN1), dystrophin (DMD), coagulation factor V (F5), fragil X mental retardation 1 (FMR1), glucosylceramidase beta (GBA), homeostatic iron regulator (HFE), coagulation factor IX (FIX), huntingtin (HD), fibrillin 1 (FBN1), dystrophia myotonica protein kinase (DMPK), cellular nucleic acid binding protein (CNBP), protein tyrosine phosphatase, non-receptor type 11 (PTPN11), Ras/Rac guanine nucleotide exchange factor 1 (SOS1), Raf proto-oncogene serine/threonine kinase (RAF1), Kras proto-oncogene GTPase (KRAS), collagen type alpha 1 chain (COL1A1), collagen type alpha 2 chain (COL1A2), synuclein alpha (SNCA), ubiquitin C-terminal hydrolase L1 (UCHL1), leucine rich repeat kinase 2 (LRRK2), Parkinson disease 3 (PARK3), parkin RBR E3 ubiquitin protein ligase (PARK2), parkinsonism associated deglycase (PARK7), PTEN induced putative kinase 1 (PARK6), apolipoprotein B (APOB), low density lipoprotein receptor (LDLR), low density lipoprotein receptor adaptor protein 1 (LDLRAP1), proprotein convertase subtilisin/kexin type 9 (PCSK9), actin alpha cardiac muscle 1 (ACTC1), actinin alpha2 (ACTN2), calreticulin 3 (CALR3), cysteine and glycine rich protein 3 (CSRP3), junctophilin2 (JPH2), myosin heavy chain 7 (MYH7), myosin light chain 2 (MYL2), myosin light chain 3 (MyL3), myozenin 2 (MYOZ2), nexilin F-actin binding protein (NEXN), phospholamban (PLN), protein kinase AMP-activated non-catalytic

subunit gamma 2 (PRKAG2), titin-cap (TCAP), troponin I3 cardiac type (TNNI3), troponin T2 cardiac type (TNNT2), tropomyosin 1 (TPM1), titin (TTN), and/or vinculin (VCL).