CLAIMS

1. A method of regulating expression of a gene located on an episomal vector in a subject in need thereof, comprising providing an editing agent to the subject, wherein the editing agent effects an alteration in a region of an mRNA transcript of the gene to thereby regulate the expression of the gene.
2. The method of claim 1, wherein the editing agent effects a base alteration in the region of the mRNA transcript of the gene.
3. The method of claim 1, wherein the alteration in a region of an mRNA transcript of the gene alters the stability of the mRNA transcript, the initiation or level of the translation of the mRNA transcript, the stability and/or activity of the translated protein.
4. The method of claim 2, wherein the region of the mRNA transcript comprises a premature termination codon convertible to an amino acid codon via the base alteration, or an amino acid codon convertible to a premature termination codon via the base alteration.
5. The method of claim 2, wherein the base alteration (a) is within a microRNA targeting site or a toe-hold switch site, or (b) induces a ribosomal frameshift or alters a codon encoding an amino acid residue critical to function and/or structure of an encoded protein.
6. The method of any one of claims 2-5, wherein the editing agent comprises a targeting ribonucleic acid complementary to the region of the mRNA transcript.
7. The method of claim 6, wherein the targeting ribonucleic acid is linear or circular.
8. The method of claim 6 or claim 7, wherein the targeting ribonucleic acid effects the base alteration via binding to an endogenous adenosine deaminase domain.
9. The method of claim 8, wherein the adenosine deaminase is selected from the group consisting of adenosine deaminase acting on RNA 1 (ADAR1), ADAR2, and ADAR3.
10. The method of any one of claims 6-9, wherein the editing agent further comprises:

    a base editor comprising a polynucleotide programmable RNA binding domain and an adenosine deaminase domain or a cytidine deaminase domain, or a nucleic acid encoding the based editor,

    wherein the polynucleotide programmable RNA binding domain in conjunction with the targeting ribonucleic acid effects the base alteration.
11. The method of claim 10, wherein the polynucleotide programmable RNA binding domain comprises a nuclease inactive variant of Cas13 or a nickase variant of Cas13.

12. The method of claim 11, wherein the Cas13 is Cas13a or Cas13b.
13. The method of claim 11 or claim12, wherein (i) the cytidine deaminase domain is selected from the group consisting of the apolipoprotein B mRNA-editing enzyme, catalytic polypeptide-like (APOBEC) family of deaminases, such as APOBEC1, APOBEC2, APOBEC3A, APOBEC3B, APOBEC3C, APOBEC3D/E, APOBEC3F, APOBEC3G, APOBEC3H, or APOBEC4; activation-induced cytidine deaminase (AID), such as activation induced cytidine deaminase (AICDA); cytosine deaminase 1 (CDA1) or CDA2; or cytosine deaminase acting on tRNA (CDAT), and (ii) the adenosine deaminase is selected from the group consisting of adenosine deaminase acting on RNA 1 (ADAR1), ADAR2, ADAR3; adenosine deaminase acting on tRNA 1 (ADAT1), ADAT2, ADAT3; and naturally occurring or engineered tRNA-specific adenosine deaminase (TadA).
14. The method of any one of claims 6-13, wherein the targeting ribonucleic acid is a guide RNA or trigger RNA.
15. The method of any of claims 10-13, wherein the base editor or the targeting ribonucleic acid is encoded by one or more nucleic acid molecules to be administered to the subject, preferably the base editor is encoded by one or more RNA molecules, such as one or more mRNA molecules, to be administered to the subject.
16. The method of claim 15, wherein the targeting ribonucleic acid and/or the one or more nucleic acid molecules, such as the one or more mRNA molecules, are to be administered to the subject with a lipid nanoparticle (LNP), a peptide cage, or a polymer nanoparticle.
17. The method of any one of claims 2-16, wherein the base alteration results in:
    (a) a conversion of an amino acid codon to a premature stop codon, preferably upstream of a splice junction, to thereby down-regulate the expression of the gene; or
    (b) a conversion of a premature stop codon to an amino acid codon to thereby up-regulate the expression of the gene.
18. The method of claim 17, wherein the base alteration results in the conversion of a CGA, CAG, or TGG codon to a premature TGA, TAG, or TAA stop codon, respectively, and the base editor comprises the cytidine deaminase domain, preferably, the CAG codon is located in proximity to the 5'-end of the gene.

19. The method of claim 17, wherein the base alteration results in the conversion of a premature UAG, UAA, or UGA stop codon to a CAG, CAA, or CGA, respectively, and the base editor comprises the adenosine deaminase domain, preferably, the premature UAG stop codon is located in proximity to the 5'-end of the gene.
20. The method of any one of the foregoing claims, wherein varying amounts of the editing agent, such as varying amounts of the targeting ribonucleic acid are to be administered to the subject to obtain varying expression levels of the gene.
21. The method of any one of the foregoing claims, further providing the episomal vector comprising the gene to the subject.
22. The method of any one of the foregoing claims, wherein the episomal vector is a nonviral vector, such as a plasmid, or a viral vector, such as an adeno-associated virus (AAV) vector or an adenovirus vector.
23. The method of any one of the foregoing claims, wherein the subject is a human, such as a human subject suffering from a disease selected from the group consisting of Hereditary angioedema, Pompe disease, hemophilia A, hemophilia B, Fabry disease, Huntington disease, Parkinson's disease, Alzheimer's disease, a synucleinopathy, epilepsy, neuropathic pain, wet macular degeneration, Usher 1F, Usher 1B, glaucoma, Leber congenital amaurosis, and Stargardt disease.