We claim:

1. A genetically modified non-human animal comprising in its genome at least one modified endogenous long intergenic non-coding RNA (lincRNA) locus,

wherein the at least one modified lincRNA locus comprises an endogenous lincRNA promoter and a deletion of all or most of the sequence coding for the lincRNA,

wherein the lincRNA locus is transcriptionally active,

wherein the deletion results in a loss-of-function mutation of the lincRNA, and

wherein the non-human animal exhibits a phenotype associated with the loss-of function of the lincRNA.

2. The non-human animal of claim 1, wherein the deletion comprises

(a) a deletion starting in a second exon of the lincRNA locus;

(b) a deletion starting in a first exon of the lincRNA locus; or

(c) a deletion of an entire RNA coding region of the lincRNA locus.

3. The non-human animal of claim 2, wherein the loss-of-function mutation comprises

(a) a replacement of the lincRNA locus or a portion thereof with an insert nucleic acid;

(b) a replacement of the second exon, the last exon, and all exons between the second and last exons of the lincRNA locus, with an insert nucleic acid; or

(c) a replacement of the entire RNA coding region of the lincRNA locus with an insert nucleic acid.

4. The non-human animal of claim 3, wherein the insert nucleic acid comprises a first nucleotide sequence that encodes a reporter and/or a second nucleotide sequence that encodes a selectable marker.

5. The non-human animal of claim 4, wherein the first nucleotide sequence is and/or the second nucleotide sequence is operably linked to a promoter that drives expression of the reporter.

6. The non-human animal of claim 5, wherein the first nucleotide sequence that encodes the reporter is positioned in a lncRNA locus in operable linkage with the endogenous lncRNA promoter, wherein the endogenous lncRNA promoter drives expression of the nucleotide sequence.

7. The non-human animal of claim 6, wherein the expression of the nucleic acid sequence follows an expression pattern of the lncRNA.

8. The non-human animal of claim 4, wherein the first nucleotide sequence comprises a Kozak consensus sequence.

9. The non-human animal of claim 4, wherein the reporter is any of β-galactosidase, Green Fluorescent Protein (GFP), enhanced Green Fluorescent Protein (eGFP), mPlum, mCherry, tdTomato, mStrawberry, J-Red, DsRed, mOrange, mKO, mCitrine, Venus, YPet, enhanced yellow fluorescent protein (EYFP), Emerald, CyPet, cyan fluorescent protein (CFP), Cerulean, T-Sapphire, luciferase, alkaline phosphatase, or a combination thereof.

10. The non-human animal of claim 4, wherein the insert nucleic acid comprises site-specific recombination sites flanking a segment encoding the reporter and/or a segment encoding the selectable marker.

11. The non-human animal of claim 1, wherein the lncRNA comprises *Pint, Celrr, Crnde, Eldr, Fendrr, Halr1, Hotair, Hottip, Hoxa11os, Pantr1, Pantr2, Ptgs2os2, lincenc1, Trp53cor1, lincppara, Mannr, Haglr, Peril, Kantr, Tug1*, or a combination thereof.

12. The non-human animal of claim 1, wherein the non-human animal is homozygous for the modified lincRNA locus.

13. The non-human animal of claim 1, wherein the non-human animal is a rodent.

14. The non-human animal of claim 1, wherein the non-human animal is a rat or a mouse.

15. The non-human animal of any one of claims 1-14, wherein the rodent is a mouse.

16. The non-human animal of claim 15, wherein the lincRNA locus is *Pint, Pantr2, Fendrr, HOTTIP, Haglr, Kantr, Peril or Tug1*.

17. The non-human animal of claim 16, wherein the phenotype is

(a) a premature aging-associated phenotype;

(b) lethality;

(c) a defect in lung development;

(d) a morphological or functional malformation of skeletal and/or muscle tissue;

(e) a loss of muscle mass in one or more tissues;

(f) a defect in brain development; or

(g) a combination thereof of any of (a)-(f).

18. The non-human animal of claim 15, wherein the lncRNA is *Pint*, and the loss of-function mutation results in the mouse exhibiting:

(a) a slower growth rate than that of a wild type control;

(b) a decline in muscle strength;

(c) fibrosis;

(d) a lower body fat content than that of the wild type control;

(e) a lower femur bone mineral density and bone mass than that of the wild type control;

(f) a decreased muscle mass as compared with that of the wild type control;

(g) a decrease in median longevity;

(h) lordokyphosis;

(i) organ atrophy;

(j) a premature-aging phenotype;

(k) a defect in brain development; or

(l) a combination thereof of any of (a)-(k).

19. The non-human animal of claim 18, wherein the loss-of-function mutation comprises a deletion of a genomic sequence of the *Pint* locus between genomic coordinates Chromosome 6: 31166026-31197846.

20. The non-human animal of claim 18, wherein the loss-of-function mutation further comprises

(a) a replacement of the second exon, last exon, and all exons between the second and the last exons of the *Pint* locus with an insert nucleic acid; or

(b) a replacement of an entire RNA coding region of the *Pint* locus with an insert nucleic acid.

21. The non-human animal of claim 20, wherein the insert nucleic acid replaces the genomic sequence between genomic coordinates Chromosome 6: 31166026-31197846.

22. The non-human animal of claim 15, wherein the lincRNA is *Pantr2* and the loss-of-function mutation results in the mouse exhibiting a defect in brain development and/or a premature aging associated phenotype.

23. The non-human animal of claim 22, wherein the loss-of-function mutation comprises a deletion of a genomic sequence of the *Pantr2* locus between genomic coordinates Chromosome 1: 42707143-42713698.

24. The non-human animal of claim 22, wherein the loss-of-function mutation further comprises

(a) a replacement of the second exon, last exon, and all exons between the second and the last exons of the *Pantr2* locus with an insert nucleic acid; or

(b) a replacement of an entire RNA coding region of the *Pantr2*_locus with an insert nucleic acid.

25. The non-human animal of claim 24, wherein the insert nucleic acid replaces the genomic sequence between genomic coordinates Chromosome 1: 42707143-42713698.

26. The non-human animal of claim 15, wherein the lincRNA is *Fendrr* and the loss-of-function mutation results in the mouse exhibiting lethality and/or a defect in lung development.

27. The non-human animal of claim 26, wherein the loss-of-function mutation comprises a deletion of a genomic sequence of a *Fendrr* locus between genomic coordinates Chromosome 8: 121054882-121074065.

28. The non-human animal of claim 26, wherein the loss-of-function mutation further comprises

(a) a replacement of the second exon, last exon, and all exons between the second and the last exons of the *Fendrr* locus with an insert nucleic acid; or

(b) a replacement of an entire RNA coding region of the *Fendrr* locus with an insert nucleic acid.

29. The non-human animal of claim 28, wherein the insert nucleic acid replaces the genomic sequence between genomic coordinates Chromosome 8: 121054882 121074065.

30. The non-human animal of claim 15, wherein the lincRNA is *HOTTIP* and the loss-of-function mutation results in the mouse exhibiting at least one morphological or functional defect in skeleton and/or muscle.

31. The non-human animal of claim 30, wherein the loss-of-function mutation comprises a deletion of a genomic sequence of a *HOTTIP* locus between genomic coordinates Chromosome 6: 52262834-52267603 or Chromosome 6: 52265374-52267603.

32. The non-human animal of claim 30, wherein the loss-of-function mutation further comprises

(a) a replacement of the second exon, last exon, and all exons between the second and the last exons of the *HOTTIP* locus with an insert nucleic acid; or

(b) a replacement of an entire RNA coding region of the *HOTTIP* locus with an insert nucleic acid.

33. The non-human animal of claim 32, wherein the insert nucleic acid replaces the genomic sequence between genomic coordinates Chromosome 6: 52262834-52267603 or Chromosome 6: 52265374-52267603.

34. The non-human animal of claim 15, wherein the lncRNA is *Haglr* and the loss-of-function mutation results in the mouse exhibiting lethality.

35. The non-human animal of claim 34, wherein the loss-of-function mutation comprises a deletion of a genomic sequence of a *Haglr* locus between genomic coordinates Chromosome 2: 74750433-74762886.

36. The non-human animal of claim 34, wherein the loss-of-function mutation further comprises

(a) a replacement of the second exon, last exon, and all exons between the second and the last exons of the *Haglr* locus with an insert nucleic acid; or

(b) a replacement of an entire RNA coding region of the *Haglr* locus with an insert nucleic acid.

37. The non-human animal of claim 36, wherein the insert nucleic acid replaces the genomic sequence between genomic coordinates Chromosome 2: 74750433-74762886.

38. The non-human animal of claim 15, wherein the lincRNA is *Kantr* and the loss-of-function mutation results in the mouse exhibiting a defect in brain development and/or a premature aging associated phenotype.

39. The non-human animal of claim 38, wherein the loss-of-function mutation comprises a deletion of a genomic sequence of a *Kantr* locus between genomic coordinates Chromosome X: 152298544-152327475.

40. The non-human animal of claim 38, wherein the loss-of-function mutation further comprises

(a) a replacement of the second exon, last exon, and all exons between the second and the last exons of the *Kantr* locus with an insert nucleic acid; or

(b) a replacement of an entire RNA coding region of the *Kantr* locus with an insert nucleic acid.

41. The non-human animal of claim 40, wherein the insert nucleic acid replaces the genomic sequence between genomic coordinates Chromosome X: 152298544 152327475.

42. The non-human animal of claim 15, wherein the lincRNA is *Peril* and the loss-of-function mutation results in the mouse exhibiting a defect in brain development, a premature aging associated phenotype, and/or perinatal lethality.

43. The non-human animal of claim 42, wherein the loss-of-function mutation comprises a deletion of a genomic sequence of a *Peril* locus between genomic coordinates Chromosome 3: 34767849-34782292.

44. The non-human animal of claim 43, wherein the loss-of-function mutation further comprises

(a) a replacement of the second exon, last exon, and all exons between the second and the last exons of the *Peril* locus with an insert nucleic acid; or

(b) a replacement of an entire RNA coding region of the *Peril* locus with an insert nucleic acid.

45. The non-human animal of claim 44, wherein the insert nucleic acid replaces the genomic sequence between genomic coordinates Chromosome 3: 34767849-34782292.

46. The non-human animal of claim 15, wherein the lincRNA is *Tug1* and the loss-of-function mutation results in the mouse exhibiting a defect in brain development and/or a premature aging-associated phenotype.

47. The non-human animal of claim 46, wherein the loss-of-function mutation comprises a deletion of a genomic sequence of a *Tug1* locus between genomic coordinates Chromosome 11: 3639794-3648758 or Chromosome 11: 3639794-3645518.

48. The non-human animal of claim 46, wherein the loss-of-function mutation further comprises

(a) a replacement of the second exon, last exon, and all exons between the second and the last exons of the *Tug1* locus with an insert nucleic acid; or

(b) a replacement of an entire RNA coding region of the *Tug1* locus with an insert nucleic acid.

49. The non-human animal of claim 48, wherein the insert nucleic acid replaces the genomic sequence between genomic coordinates Chromosome 11: 3639794-3648758 or Chromosome 11: 3639794-3645518.

50. A cell, tissue, or embryo derived from the non-human animal of any one of claims 1-14 and 16-49.

51. A targeting vector, comprising an insert nucleic acid flanked by 5' and 3' homology arms that can undergo homologous recombination with a lincRNA locus of interest, wherein following the homologous recombination with the lincRNA locus of interest, the insert nucleic acid is operably linked to an endogenous lincRNA promoter and replaces all or most of the sequence coding for the lincRNA.

52. The targeting vector of claim 51, wherein the insert nucleic acid comprises a first nucleic acid sequence that encodes a reporter.

53. The targeting vector of claim 53, wherein following the homologous recombination with the lncRNA locus of interest, the first nucleic acid sequence that encodes the reporter is operably linked to the endogenous promoter that drives expression of an lncRNA at the lncRNA locus.

54. The targeting vector of any one of claims 52 or 53, wherein the reporter is any of β-galactosidase, Green Fluorescent Protein (GFP), enhanced Green Fluorescent Protein (eGFP), mPlum, mCherry, tdTomato, mStrawberry, J-Red, DsRed, mOrange, mKO, mCitrine, Venus, YPet, enhanced yellow fluorescent protein (EYFP), Emerald, CyPet, cyan fluorescent protein (CFP), Cerulean, T-Sapphire, luciferase, alkaline phosphatase, or a combination thereof.

55. The targeting vector of any one of claims 51-54, wherein the insert nucleic acid further comprises a second nucleic acid sequence that encodes a selectable marker, wherein the second nucleic acid is operably linked to a promoter.

56. The targeting vector of claim 55, further comprising site-specific recombination sites flanking a segment encoding the reporter and/or a segment encoding the selectable marker nucleic acid.

57. The targeting vector of any one of claims 52-56, wherein the first and/or the second nucleic acid sequence further comprises a Kozak consensus sequence.

58. The targeting vector of any one of claims 51-57, wherein the insert nucleic acid further comprises a promoter that drives expression of the reporter.

59. A method for making a non-human animal of claim 1, the method comprising:

(a) contacting a pluripotent cell with a targeting construct comprising an insert nucleic acid flanked by 5' and 3' homology arms; wherein the targeting construct undergoes homologous recombination with the lincRNA locus in a genome of the cell to form a modified pluripotent cell comprising a modified lincRNA locus;

(b) introducing the modified pluripotent cell into a host embryo; and

(c) gestating the host embryo in a surrogate mother, wherein the surrogate mother produces progeny comprising the modified lncRNA locus,

wherein the modified lincRNA locus comprises an endogenous lincRNA promoter and a deletion of all or most of the sequence coding for the lincRNA,

wherein the lincRNA locus is transcriptionally active,

wherein the deletion results in a loss-of-function mutation of the lincRNA, and

wherein said genetic modification results in a loss-of-function mutation of the at least one lncRNA such that the progeny exhibits a phenotype associated with the loss-of-function mutation.

60. The method of claim 59, wherein the loss-of-function mutation comprises a deletion of at least a second exon, a last exon, and all exons between the second and the last exons of the lincRNA locus.

61. The method of any one of claims 59-60, wherein the lncRNA comprises *Pint, Celrr, Crnde, Eldr, Fendrr, Halr1, Hotair, Hottip, Hoxa11os, Pantr1, Pantr2, Ptgs2os2, lincenc1, Trp53cor1, lincppara, Mannr, Haglr, Peril, Kantr, Tug1*, or a combination thereof.

62. A method for modifying a lincRNA locus in a pluripotent cell, comprising

(a) introducing into the pluripotent cell a targeting construct comprising an insert nucleic acid flanked with 5' and 3' homology arms that can undergo homologous recombination with the lincRNA locus; and

(b) identifying a modified pluripotent cell comprising a targeted genetic modification at the lincRNA locus,

wherein the modified lincRNA locus comprises an endogenous lincRNA promoter and a deletion of all or most of the sequence coding for the lincRNA,

wherein the lincRNA locus is transcriptionally active,

wherein the deletion results in loss-of-function of the lincRNA, and

wherein a non-human animal generated from the pluripotent cell exhibits a phenotype associated with the loss-of-function mutation.

63. The method of claim 62, wherein the pluripotent cell is a human iPS cell.

64. The method of claim 62, wherein the pluripotent cell is a mouse or a rat embryonic stem (ES) cell.

65. The method of any one of claims 62-64, wherein the lncRNA comprises *Pint, Celrr, Crnde, Eldr, Fendrr, Halr1, Hotair, Hottip, Hoxa11os, Pantr1, Pantr2, Ptgs2os2, lincenc1, Trp53cor1, lincppara, Mannr, Haglr, Peril, Kantr, Tug1*, or a combination thereof.

66. A method of obtaining a cell, tissue, or embryo comprising deriving a cell, tissue or embryo from a non-human animal of 1-14 and 16-49.