Publications Stemming From

The Progeria Research Foundation Cell and Tissue Bank

The Progeria Research Foundation Cell and Tissue Bank has contributed to the following medical publications, categorized by cell line for researcher convenience:

HGADFN001

**Age-dependent loss of MMP-3 in Hutchinson-Gilford progeria syndrome.**

**The mutant form of lamin A that causes Hutchinson-Gilford progeria is a biomarker of cellular aging in human skin.**

**Hutchinson-Gilford progeria mutant lamin A primarily targets human vascular cells as detected by an anti-Lamin A G608G antibody.**

**Aggrecan expression is substantially and abnormally upregulated in Hutchinson-Gilford Progeria Syndrome dermal fibroblasts.**

**Rescue of heterochromatin organization in Hutchinson-Gilford progeria by drug treatment.**

**Recurrent de novo point mutations in lamin A cause Hutchinson-Gilford progeria syndrome.**

HGADFN003

**Sulforaphane enhances progerin clearance in Hutchinson-Gilford progeria fibroblasts.**
Depleting the methyltransferase Suv39h1 improves DNA repair and extends lifespan in a progeria mouse model.

Naïve adult stem cells from patients with Hutchinson-Gilford progeria syndrome express low levels of progerin in vivo.

Age-dependent loss of MMP-3 in Hutchinson-Gilford progeria syndrome.

Progerin and telomere dysfunction collaborate to trigger cellular senescence in normal human fibroblasts.

Defective lamin A-Rb signaling in Hutchinson-Gilford Progeria Syndrome and reversal by farnesyltransferase inhibition.

Effect of progerin on the accumulation of oxidized proteins in fibroblasts from Hutchinson Gilford progeria patients.

Ageing-related chromatin defects through loss of the NURD complex.

Lamin A-dependent misregulation of adult stem cells associated with accelerated ageing.

Perturbation of wild-type lamin A metabolism results in a progeroid phenotype.

Alterations in mitosis and cell cycle progression caused by a mutant lamin A known to accelerate human aging.

The mutant form of lamin A that causes Hutchinson-Gilford progeria is a biomarker of cellular aging in human skin.

**A lamin A protein isoform overexpressed in Hutchinson-Gilford progeria syndrome interferes with mitosis in progeria and normal cells.**


**Hutchinson-Gilford progeria mutant lamin A primarily targets human vascular cells as detected by an anti-Lamin A G608G antibody.**


**Aggrecan expression is substantially and abnormally upregulated in Hutchinson-Gilford Progeria Syndrome dermal fibroblasts.**


**Rescue of heterochromatin organization in Hutchinson-Gilford progeria by drug treatment.**


**Genomic instability in laminopathy-based premature aging.**


**Incomplete processing of mutant lamin A in Hutchinson-Gilford progeria leads to nuclear abnormalities, which are reversed by farnesyltransferase inhibition.**


**Accumulation of mutant lamin A causes progressive changes in nuclear architecture in Hutchinson-Gilford progeria syndrome.**


**Recurrent de novo point mutations in lamin A cause Hutchinson-Gilford progeria syndrome.**

Incomplete processing of mutant lamin A in Hutchinson-Gilford progeria leads to nuclear abnormalities, which are reversed by farnesyltransferase inhibition.

**HGADFN005**

Recurrent de novo point mutations in lamin A cause Hutchinson-Gilford progeria syndrome.

**HGADFN008**

Recurrent de novo point mutations in lamin A cause Hutchinson-Gilford progeria syndrome.

**HGADFN014**

Recurrent de novo point mutations in lamin A cause Hutchinson-Gilford progeria syndrome.

**HGADFN086**

Increased progerin expression associated with unusual LMNA mutations causes severe progeroid syndromes.

**HGMDFN090**

Higher-order unfolding of satellite heterochromatin is a consistent and early event in cell senescence.

Correlated alterations in genome organization, histone methylation, and DNA-lamin A/C interactions in Hutchinson-Gilford progeria syndrome.
Comparison of constitutional and replication stress-induced genome structural variation by SNP array and mate-pair sequencing.

Hydroxyurea induces de novo copy number variants in human cells.

Progerin and telomere dysfunction collaborate to trigger cellular senescence in normal human fibroblasts.

CTP:phosphocholine cytidylyltransferase α (CCTα) and lamins alter nuclear membrane structure without affecting phosphatidylcholine synthesis.

Effect of progerin on the accumulation of oxidized proteins in fibroblasts from Hutchinson Gilford progeria patients.

Replication stress induces genome-wide copy number changes in human cells that resemble polymorphic and pathogenic variants.

A lamin A protein isoform overexpressed in Hutchinson-Gilford progeria syndrome interferes with mitosis in progeria and normal cells.

Incomplete processing of mutant lamin A in Hutchinson-Gilford progeria leads to nuclear abnormalities, which are reversed by farnesyltransferase inhibition.

Depleting the methyltransferase Suv39h1 improves DNA repair and extends lifespan in a progeria mouse model.

HGADFN122
Sulforaphane enhances progerin clearance in Hutchinson-Gilford progeria fibroblasts.

A proteomic study of Hutchinson-Gilford progeria syndrome: Application of 2D-chromatography in a premature aging disease.

Age-dependent loss of MMP-3 in Hutchinson-Gilford progeria syndrome.

CTP:phosphocholine cytidylyltransferase α (CCTα) and lamins alter nuclear membrane structure without affecting phosphatidylcholine synthesis.

Defective lamin A-Rb signaling in Hutchinson-Gilford Progeria Syndrome and reversal by farnesyltransferase inhibition.

Increased mechanosensitivity and nuclear stiffness in Hutchinson-Gilford progeria cells: effects of farnesyltransferase inhibitors.

Alterations in mitosis and cell cycle progression caused by a mutant lamin A known to accelerate human aging.

The mutant form of lamin A that causes Hutchinson-Gilford progeria is a biomarker of cellular aging in human skin.

Aggrecan expression is substantially and abnormally upregulated in Hutchinson-Gilford Progeria Syndrome dermal fibroblasts.
Hutchinson-Gilford progeria mutant lamin A primarily targets human vascular cells as detected by an anti-Lamin A G608G antibody.

Rescue of heterochromatin organization in Hutchinson-Gilford progeria by drug treatment.

Genomic instability in laminopathy-based premature aging.

Novel progerin-interactive partner proteins hnRNP E1, EGF, Mel 18, and UBC9 interact with lamin A/C.

Incomplete processing of mutant lamin A in Hutchinson-Gilford progeria leads to nuclear abnormalities, which are reversed by farnesyltransferase inhibition.

Depleting the methyltransferase Suv39h1 improves DNA repair and extends lifespan in a progeria mouse model.

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The mutant form of lamin A that causes Hutchinson-Gilford progeria is a biomarker of cellular aging in human skin.
Hutchinson-Gilford progeria mutant lamin A primarily targets human vascular cells as detected by an anti-Lamin A G608G antibody.

HGADFN155

Sulforaphane enhances progerin clearance in Hutchinson-Gilford progeria fibroblasts.

Higher-order unfolding of satellite heterochromatin is a consistent and early event in cell senescence.

Correlated alterations in genome organization, histone methylation, and DNA-lamin A/C interactions in Hutchinson-Gilford progeria syndrome.

An inhibitory role of progerin in the gene induction network of adipocyte differentiation from iPS cells.

Depleting the methyltransferase Suv39h1 improves DNA repair and extends lifespan in a progeria mouse model.

Automated image analysis of nuclear shape: what can we learn from a prematurely aged cell?

Rapamycin reverses cellular phenotypes and enhances mutant protein clearance in Hutchinson-Gilford progeria syndrome cells.

Defective lamin A-Rb signaling in Hutchinson-Gilford Progeria Syndrome and reversal by farnesyltransferase inhibition.

HGADFN164

Sulforaphane enhances progerin clearance in Hutchinson-Gilford progeria fibroblasts.
Mechanisms controlling the smooth muscle cell death in progeria via down-regulation of poly(ADP-ribose) polymerase 1.

Correlated alterations in genome organization, histone methylation, and DNA-lamin A/C interactions in Hutchinson-Gilford progeria syndrome.

An inhibitory role of progerin in the gene induction network of adipocyte differentiation from iPS cells.

Depleting the methyltransferase Suv39h1 improves DNA repair and extends lifespan in a progeria mouse model.

Naïve adult stem cells from patients with Hutchinson-Gilford progeria syndrome express low levels of progerin in vivo.

Defective lamin A-Rb signaling in Hutchinson-Gilford Progeria Syndrome and reversal by farnesyltransferase inhibition.

**HGADFN167**

Phenotype-Dependent Coexpression Gene Clusters: Application to Normal and Premature Ageing.

Mechanisms controlling the smooth muscle cell death in progeria via down-regulation of poly(ADP-ribose) polymerase 1.

Higher-order unfolding of satellite heterochromatin is a consistent and early event in cell senescence.
Correlated alterations in genome organization, histone methylation, and DNA-lamin A/C interactions in Hutchinson-Gilford progeria syndrome.

Progeria: translational insights from cell biology.

Automated image analysis of nuclear shape: what can we learn from a prematurely aged cell?

Computational image analysis of nuclear morphology associated with various nuclear-specific aging disorders.

Rapamycin reverses cellular phenotypes and enhances mutant protein clearance in Hutchinson-Gilford progeria syndrome cells.

Progerin and telomere dysfunction collaborate to trigger cellular senescence in normal human fibroblasts.

CTP:phosphocholine cytidylyltransferase α (CCTα) and lamins alter nuclear membrane structure without affecting phosphatidylcholine synthesis.

Effect of progerin on the accumulation of oxidized proteins in fibroblasts from Hutchinson Gilford progeria patients.

A lamin A protein isoform overexpressed in Hutchinson-Gilford progeria syndrome interferes with mitosis in progeria and normal cells.

HGADFN168
Phenotype-Dependent Coexpression Gene Clusters: Application to Normal and Premature Ageing.

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Rapamycin reverses cellular phenotypes and enhances mutant protein clearance in Hutchinson-Gilford progeria syndrome cells.

Rapamycin reverses cellular phenotypes and enhances mutant protein clearance in Hutchinson-Gilford progeria syndrome cells.

Stem cell depletion in Hutchinson-Gilford progeria syndrome.

Low and high expressing alleles of the LMNA gene: implications for laminopathy disease development.

Recurrent de novo point mutations in lamin A cause Hutchinson-Gilford progeria syndrome.
HGALBV011

Low and high expressing alleles of the LMNA gene: implications for laminopathy disease development.

HGMLBV013

Recurrent de novo point mutations in lamin A cause Hutchinson-Gilford progeria syndrome.

HGFLBV021

Stem cell depletion in Hutchinson-Gilford progeria syndrome.

HGMLBV023

Stem cell depletion in Hutchinson-Gilford progeria syndrome.

HGFLBV031
**Stem cell depletion in Hutchinson-Gilford progeria syndrome.**

**Recurrent de novo point mutations in lamin A cause Hutchinson-Gilford progeria syndrome.**
**HGMLBV066**

**Stem cell depletion in Hutchinson-Gilford progeria syndrome.**

**Recurrent de novo point mutations in lamin A cause Hutchinson-Gilford progeria syndrome.**

**HGFLBV067**

**Stem cell depletion in Hutchinson-Gilford progeria syndrome.**

**Recurrent de novo point mutations in lamin A cause Hutchinson-Gilford progeria syndrome.**

**HGALBV071**

**Recurrent de novo point mutations in lamin A cause Hutchinson-Gilford progeria syndrome.**

**HGMLBV081**

**Recurrent de novo point mutations in lamin A cause Hutchinson-Gilford progeria syndrome.**

**HGFLBV082**

**Recurrent de novo point mutations in lamin A cause Hutchinson-Gilford progeria syndrome.**