

Resource Summary Report

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Progeria Research Foundation

RRID:SCR_012786

Type: Tool

Proper Citation

Progeria Research Foundation (RRID:SCR_012786)

Resource Information

URL: <http://www.progeriaresearch.org/index.html>

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Description: The mission of The Progeria Research Foundation is to discover treatments and the cure for Progeria, and its aging related disorders. Progeria is a rare and fatal genetic disease characterized by an appearance of accelerated aging in children. Without the discovery of new treatments, all children with Progeria will die of heart disease at an average age of 13 years. The Progeria Research Foundation (PRF) was founded in 1999 in response to the complete lack of progress being made to help children with Progeria. We have filled a void, taking these children out of the background where they had been for over 100 years and putting them and Progeria at the forefront of scientific efforts. In just 11.5 years, we have achieved extraordinary progress towards our mission: the Progeria gene discovery in 2003, first-ever clinical drug trials initiated in 2007, extensive global awareness of the disease and PRF's work, and discovery of critical biological links between Progeria, heart disease and aging we all experience.

Abbreviations: PRF

Resource Type: institution

Keywords: progeria

Related Condition: Aging

Resource Name: Progeria Research Foundation

Resource ID: SCR_012786

Alternate IDs: Crossref funder ID: 100002287, grid.423381.f, nlx_53243

Alternate URLs: <https://ror.org/03qs03z89>

Record Creation Time: 20220129T080312+0000

Ratings and Alerts

No rating or validation information has been found for Progeria Research Foundation.

No alerts have been found for Progeria Research Foundation.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 40 mentions in open access literature.

Listed below are recent publications. The full list is available at [NIF](#) .

Yamada L, et al. (2026) Senescence-inhibitory ?133p53? counteracts accelerated ageing and mortality. bioRxiv : the preprint server for biology.

Yamada L, et al. (2026) The senescence-inhibitory p53 isoform ?133p53? represses the proinflammatory chemokine CXCL10 in progeria model mice and naturally aged mice. bioRxiv : the preprint server for biology.

Fenzl FQ, et al. (2025) Deregulated miR-145 and miR-27b in Hutchinson-Gilford progeria syndrome: implications for adipogenesis. Aging, 17(9), 2278.

Joruiz SM, et al. (2025) Pharmacologic activation of ?133p53? reduces cellular senescence in progeria patients-derived cells. bioRxiv : the preprint server for biology.

Muela-Zarzuela I, et al. (2024) The NLRP3 inhibitor Dapansutride improves the therapeutic action of lonafarnib on progeroid mice. Aging cell, 23(9), e14272.

Olsen FJ, et al. (2024) Abnormal Myocardial Deformation Despite Normal Ejection Fraction in Hutchinson-Gilford Progeria Syndrome. Journal of the American Heart Association, 13(3), e031470.

Lattanzi G, et al. (2024) Aging research from bench to bedside and beyond: What we learned from Sammy Basso. Aging cell, 23(12), e14414.

Krüger P, et al. (2024) Navigating Lipodystrophy: Insights from Laminopathies and Beyond. International journal of molecular sciences, 25(15).

Krüger P, et al. (2024) Inflammation and Fibrosis in Progeria: Organ-Specific Responses in an HGPS Mouse Model. International journal of molecular sciences, 25(17).

Macías Á, et al. (2024) Coronary and carotid artery dysfunction and KV7 overexpression in a mouse model of Hutchinson-Gilford progeria syndrome. GeroScience, 46(1), 867.

Blouin S, et al. (2024) Normal Bone Matrix Mineralization but Altered Growth Plate Morphology in the LmnaG609G/G609G Mouse Model of Progeria. *Aging and disease*, 16(5), 3204.

Odinammadu KO, et al. (2023) The farnesyl transferase inhibitor (FTI) lonafarnib improves nuclear morphology in ZMPSTE24-deficient fibroblasts from patients with the progeroid disorder MAD-B. *Nucleus (Austin, Tex.)*, 14(1), 2288476.

Murtada SI, et al. (2023) Lonafarnib improves cardiovascular function and survival in a mouse model of Hutchinson-Gilford progeria syndrome. *eLife*, 12.

Gordon LB, et al. (2023) Plasma Progerin in Patients With Hutchinson-Gilford Progeria Syndrome: Immunoassay Development and Clinical Evaluation. *Circulation*, 147(23), 1734.

Zhang N, et al. (2023) Unique progerin C-terminal peptide ameliorates Hutchinson-Gilford progeria syndrome phenotype by rescuing BUBR1. *Nature aging*, 3(2), 185.

Macías Á, et al. (2022) Paclitaxel mitigates structural alterations and cardiac conduction system defects in a mouse model of Hutchinson-Gilford progeria syndrome. *Cardiovascular research*, 118(2), 503.

Vehns E, et al. (2022) Impact of MnTBAP and Baricitinib Treatment on Hutchinson-Gilford Progeria Fibroblasts. *Pharmaceuticals (Basel, Switzerland)*, 15(8).

Röhl JM, et al. (2021) Nuclear Pore Complexes Cluster in Dysmorphic Nuclei of Normal and Progeria Cells during Replicative Senescence. *Cells*, 10(1).

Sánchez-López A, et al. (2021) Cardiovascular Progerin Suppression and Lamin A Restoration Rescue Hutchinson-Gilford Progeria Syndrome. *Circulation*, 144(22), 1777.

Gordon LB, et al. (2021) The progeria research foundation 10th international scientific workshop; researching possibilities, EXTENDING lives - webinar version scientific summary. *Aging*, 13(6), 9143.