

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 34 mentions in open access literature.

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

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

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.

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

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

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.

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

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

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.

Hayashi K, et al. (2021) Long-term survival in a patient with Hutchinson-Gilford progeria syndrome and osteosarcoma: A case report. *World journal of clinical cases*, 9(4), 854.

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

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.

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

Arnold R, et al. (2021) Baricitinib, a JAK-STAT Inhibitor, Reduces the Cellular Toxicity of the Farnesyltransferase Inhibitor Lonafarnib in Progeria Cells. *International journal of molecular sciences*, 22(14).

Coppedè F, et al. (2021) Mutations Involved in Premature-Ageing Syndromes. The application of clinical genetics, 14, 279.

González-Dominguez A, et al. (2021) Inhibition of the NLRP3 inflammasome improves lifespan in animal murine model of Hutchinson-Gilford Progeria. *EMBO molecular medicine*, 13(10), e14012.

Hamczyk MR, et al. (2019) Progerin accelerates atherosclerosis by inducing endoplasmic reticulum stress in vascular smooth muscle cells. *EMBO molecular medicine*, 11(4).

Liu C, et al. (2019) Inhibition of JAK-STAT Signaling with Baricitinib Reduces Inflammation and Improves Cellular Homeostasis in Progeria Cells. *Cells*, 8(10).