

# Maria Eriksson

## List of Publications by Year in descending order

Source: <https://exaly.com/author-pdf/8728521/publications.pdf>

Version: 2024-02-01

45  
papers

5,020  
citations

394421

19  
h-index

254184

43  
g-index

46  
all docs

46  
docs citations

46  
times ranked

4946  
citing authors

#	ARTICLE	IF	CITATIONS
1	Challenges of proving a causal role of somatic mutations in the aging process. <i>Aging Cell</i> , 2022, 21, e13613.	6.7	7
2	Transient expression of an adenine base editor corrects the Hutchinson-Gilford progeria syndrome mutation and improves the skin phenotype in mice. <i>Nature Communications</i> , 2022, 13, .	12.8	7
3	Reverting to old theories of ageing with new evidence for the role of somatic mutations. <i>Nature Reviews Genetics</i> , 2022, 23, 645-646.	16.3	2
4	A small-molecule ICMT inhibitor delays senescence of Hutchinson-Gilford progeria syndrome cells. <i>ELife</i> , 2021, 10, .	6.0	17
5	How can we estimate QALYs based on PHQ-9 scores? Equipercentile linking analysis of PHQ-9 and EQ-5D. <i>Evidence-Based Mental Health</i> , 2021, 24, 97-101.	4.5	11
6	Acute pulmonary hypertension and short-term outcomes in severe Covid-19 patients needing intensive care. <i>Acta Anaesthesiologica Scandinavica</i> , 2021, 65, 761-769.	1.6	30
7	Splice-inhibition therapy targets progeria. <i>Nature Medicine</i> , 2021, 27, 377-379.	30.7	1
8	Base Editing in Progeria. <i>New England Journal of Medicine</i> , 2021, 384, 1364-1366.	27.0	1
9	Targeting RAS-converting enzyme 1 overcomes senescence and improves progeria-like phenotypes of ZMPSTE24 deficiency. <i>Aging Cell</i> , 2020, 19, e13200.	6.7	5
10	Comprehensive Cardiovascular Magnetic Resonance Diastolic Dysfunction Grading Shows Very Good Agreement Compared With Echocardiography. <i>JACC: Cardiovascular Imaging</i> , 2020, 13, 2530-2542.	5.3	19
11	SO042WHOLE GENOME SEQUENCING OF HUMAN KIDNEY PROGENITORS IDENTIFIES A MUTATION-PRONE CELL TYPE IN THE PROXIMAL TUBULE. <i>Nephrology Dialysis Transplantation</i> , 2020, 35, .	0.7	0
12	Analysis of somatic mutations identifies signs of selection during in vitro aging of primary dermal fibroblasts. <i>Aging Cell</i> , 2019, 18, e13010.	6.7	6
13	The association of body mass index, weight gain and central obesity with activity-related breathlessness: the Swedish Cardiopulmonary Bioimage Study. <i>Thorax</i> , 2019, 74, 958-964.	5.6	21
14	Accumulation of Progerin Affects the Symmetry of Cell Division and Is Associated with Impaired Wnt Signaling and the Mislocalization of Nuclear Envelope Proteins. <i>Journal of Investigative Dermatology</i> , 2019, 139, 2272-2280.e12.	0.7	15
15	Healthy skeletal muscle aging: The role of satellite cells, somatic mutations and exercise. <i>International Review of Cell and Molecular Biology</i> , 2019, 346, 157-200.	3.2	10
16	Somatic mutation that affects transcription factor binding upstream of CD55 in the temporal cortex of a late-onset Alzheimer disease patient. <i>Human Molecular Genetics</i> , 2019, 28, 2675-2685.	2.9	12
17	Inhibition of DNA damage response at telomeres improves the detrimental phenotypes of Hutchinson-Gilford Progeria Syndrome. <i>Nature Communications</i> , 2019, 10, 4990.	12.8	85
18	Whole genome DNA sequencing provides an atlas of somatic mutagenesis in healthy human cells and identifies a tumor-prone cell type. <i>Genome Biology</i> , 2019, 20, 285.	8.8	46

#	ARTICLE	IF	CITATIONS
19	Somatic mutagenesis in satellite cells associates with human skeletal muscle aging. <i>Nature Communications</i> , 2018, 9, 800.	12.8	94
20	Expression of the Hutchinson-Gilford Progeria Mutation Leads to Aberrant Dentin Formation. <i>Scientific Reports</i> , 2018, 8, 15368.	3.3	9
21	Endothelial progerin expression causes cardiovascular pathology through an impaired mechanoresponse. <i>Journal of Clinical Investigation</i> , 2018, 129, 531-545.	8.2	75
22	The role of epigenetics in renal ageing. <i>Nature Reviews Nephrology</i> , 2017, 13, 471-482.	9.6	86
23	Emerging candidate treatment strategies for Hutchinson-Gilford progeria syndrome. <i>Biochemical Society Transactions</i> , 2017, 45, 1279-1293.	3.4	18
24	Rare progerin-expressing preadipocytes and adipocytes contribute to tissue depletion over time. <i>Scientific Reports</i> , 2017, 7, 4405.	3.3	24
25	Global genome splicing analysis reveals an increased number of alternatively spliced genes with aging. <i>Aging Cell</i> , 2016, 15, 267-278.	6.7	79
26	Expression of progerin in aging mouse brains reveals structural nuclear abnormalities without detectable significant alterations in gene expression, hippocampal stem cells or behavior. <i>Human Molecular Genetics</i> , 2015, 24, 1305-1321.	2.9	30
27	Transgene silencing of the Hutchinson-Gilford progeria syndrome mutation results in a reversible bone phenotype, whereas resveratrol treatment does not show overall beneficial effects. <i>FASEB Journal</i> , 2015, 29, 3193-3205.	0.5	21
28	Skin Disease in Laminopathy-Associated Premature Aging. <i>Journal of Investigative Dermatology</i> , 2015, 135, 2577-2583.	0.7	10
29	Overexpression of Lamin B Receptor Results in Impaired Skin Differentiation. <i>PLoS ONE</i> , 2015, 10, e0128917.	2.5	7
30	Comparison Between the Montgomery-Asberg Depression Rating Scaleâ€“Self and the Beck Depression Inventory II in Primary Care. <i>primary care companion for CNS disorders, The</i> , 2015, 17, .	0.6	17
31	Low Levels of the Reverse Transactivator Fail to Induce Target Transgene Expression in Vascular Smooth Muscle Cells. <i>PLoS ONE</i> , 2014, 9, e104098.	2.5	1
32	Embryonic expression of the common progeroid lamin A splice mutation arrests postnatal skin development. <i>Aging Cell</i> , 2014, 13, 292-302.	6.7	36
33	Expression of the Hutchinson-Gilford Progeria Mutation during Osteoblast Development Results in Loss of Osteocytes, Irregular Mineralization, and Poor Biomechanical Properties. <i>Journal of Biological Chemistry</i> , 2012, 287, 33512-33522.	3.4	39
34	Stem cell depletion in Hutchinsonâ€“Gilford progeria syndrome. <i>Aging Cell</i> , 2011, 10, 1011-1020.	6.7	84
35	A previously functional tetracycline-regulated transactivator fails to target gene expression to the bone. <i>BMC Research Notes</i> , 2011, 4, 282.	1.4	4
36	Low and High Expressing Alleles of the LMNA Gene: Implications for Laminopathy Disease Development. <i>PLoS ONE</i> , 2011, 6, e25472.	2.5	16

#	ARTICLE	IF	CITATIONS
37	Evidence for the Involvement of Lamins in Aging. <i>Current Aging Science</i> , 2010, 3, 81-89.	1.2	18
38	Increased expression of the Hutchinsonâ€“Gilford progeria syndrome truncated lamin A transcript during cell aging. <i>European Journal of Human Genetics</i> , 2009, 17, 928-937.	2.8	113
39	Differential Expression of A-Type and B-Type Lamins during Hair Cycling. <i>PLoS ONE</i> , 2009, 4, e4114.	2.5	13
40	Targeted transgenic expression of the mutation causing Hutchinson-Gilford progeria syndrome leads to proliferative and degenerative epidermal disease. <i>Journal of Cell Science</i> , 2008, 121, 969-978.	2.0	76
41	Progressive vascular smooth muscle cell defects in a mouse model of Hutchinson-Gilford progeria syndrome. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2006, 103, 3250-3255.	7.1	255
42	Mutant nuclear lamin A leads to progressive alterations of epigenetic control in premature aging. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2006, 103, 8703-8708.	7.1	685
43	Real-Time RT-PCR for CTG Repeat-Containing Genes. , 2004, 277, 077-084.		2
44	Accumulation of mutant lamin A causes progressive changes in nuclear architecture in Hutchinsonâ€“Gilford progeria syndrome. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2004, 101, 8963-8968.	7.1	988
45	Recurrent de novo point mutations in lamin A cause Hutchinsonâ€“Gilford progeria syndrome. <i>Nature</i> , 2003, 423, 293-298.	27.8	1,925