Mathias Jucker

List of Publications by Year
in descending order

121 papers	19,808 citations	20817 60 h-index	18647 119 g-index
132 all docs	132 docs citations	132 times ranked	20141 citing authors

#	Article	IF	Citations
1	Biomarker clustering in autosomal dominant Alzheimer's disease. Alzheimer's and Dementia, 2023, 19, 274-284.	0.8	2
2	Different rates of cognitive decline in autosomal dominant and lateâ€onset Alzheimer disease. Alzheimer's and Dementia, 2022, 18, 1754-1764.	0.8	4
3	Association ofBDNFVal66Met With Tau Hyperphosphorylation and Cognition in Dominantly Inherited Alzheimer Disease. JAMA Neurology, 2022, 79, 261.	9.0	15
4	Soluble TAM receptors sAXL and sTyro3 predict structural and functional protection in Alzheimerâ€™s disease. Neuron, 2022, 110, 1009-1022.e4.	8.1	27
5	CSF p-tau increase in response to AÎ²-type and Danish-type cerebral amyloidosis and in the absence of neurofibrillary tangles. Acta Neuropathologica, 2022, 143, 287-290.	7.7	25
6	Soluble TREM2 in CSF and its association with other biomarkers and cognition in autosomal-dominant Alzheimer's disease: a longitudinal observational study. Lancet Neurology, The, 2022, 21, 329-341.	10.2	72
7	Signatures of glial activity can be detected in the CSF proteome. Proceedings of the National Academy of Sciences of the United States of America, 2022, 119, .	7.1	12
8	A neuronal blood marker is associated with mortality in old age. Nature Aging, 2021, 1, 218-225.	11.6	30
9	LAG3 is not expressed in human and murine neurons and does not modulate Î±â€synucleinopathies. EMBO Molecular Medicine, 2021, 13, e14745.	6.9	44
10	Comparison of CSF biomarkers in Down syndrome and autosomal dominant Alzheimer's disease: a cross-sectional study. Lancet Neurology, The, 2021, 20, 615-626.	10.2	26
11	Microglial inclusions and neurofilament light chain release follow neuronal Î±-synuclein lesions in long-term brain slice cultures. Molecular Neurodegeneration, 2021, 16, 54.	10.8	20
12	Accelerated functional brain aging in pre-clinical familial Alzheimerâ€™s disease. Nature Communications, 2021, 12, 5346.	12.8	43
13	Autosomal dominantly inherited alzheimer disease: Analysis of genetic subgroups by machine learning. Information Fusion, 2020, 58, 153-167.	19.1	17
14	Risk of Transmissibility From Neurodegenerative Disease-Associated Proteins: Experimental Knowns and Unknowns. Journal of Neuropathology and Experimental Neurology, 2020, 79, 1141-1146.	1.7	24
15	Acute targeting of pre-amyloid seeds in transgenic mice reduces Alzheimer-like pathology later in life. Nature Neuroscience, 2020, 23, 1580-1588.	14.8	53
16	Sequence of Alzheimer disease biomarker changes in cognitively normal adults. Neurology, 2020, 95, e3104-e3116.	1.1	35
17	Potential human transmission of amyloid Î² pathology: surveillance and risks. Lancet Neurology, The, 2020, 19, 872-878.	10.2	46
18	Medin aggregation causes cerebrovascular dysfunction in aging wild-type mice. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 23925-23931.	7.1	20

#	Article	IF	Citations
19	Prominent microglial inclusions in transgenic mouse models of Î±-synucleinopathy that are distinct from neuronal lesions. Acta Neuropathologica Communications, 2020, 8, 133.	5.2	20
20	Serum neurofilament light chain levels are associated with white matter integrity in autosomal dominant Alzheimer's disease. Neurobiology of Disease, 2020, 142, 104960.	4.4	31
21	A soluble phosphorylated tau signature links tau, amyloid and the evolution of stages of dominantly inherited Alzheimerâ€™s disease. Nature Medicine, 2020, 26, 398-407.	30.7	351
22	Early AÎ² reduction prevents progression of cerebral amyloid angiopathy. Annals of Neurology, 2019, 86, 561-571.	5.3	18
23	Prion-like spreading of Alzheimerâ€™s disease within the brainâ€™s connectome. Journal of the Royal Society Interface, 2019, 16, 20190356.	3.4	71
24	Cryo-EM structure and polymorphism of AÎ² amyloid fibrils purified from Alzheimerâ€™s brain tissue. Nature Communications, 2019, 10, 4760.	12.8	411
25	Serum neurofilament dynamics predicts neurodegeneration and clinical progression in presymptomatic Alzheimerâ€™s disease. Nature Medicine, 2019, 25, 277-283.	30.7	610
26	Clinical, pathophysiological and genetic features of motor symptoms in autosomal dominant Alzheimerâ€™s disease. Brain, 2019, 142, 1429-1440.	7.6	36
27	Emerging cerebrospinal fluid biomarkers in autosomal dominant Alzheimer's disease. Alzheimer's and Dementia, 2019, 15, 655-665.	0.8	72
28	Association of Longitudinal Changes in Cerebrospinal Fluid Total Tau and Phosphorylated Tau 181 and Brain Atrophy With Disease Progression in Patients With Alzheimer Disease. JAMA Network Open, 2019, 2, e1917126.	5.9	23
29	A physics-based model explains the prion-like features of neurodegeneration in Alzheimerâ€™s disease, Parkinsonâ€™s disease, and amyotrophic lateral sclerosis. Journal of the Mechanics and Physics of Solids, 2019, 124, 264-281.	4.8	83
30	Left frontal hub connectivity delays cognitive impairment in autosomal-dominant and sporadic Alzheimerâ€™s disease. Brain, 2018, 141, 1186-1200.	7.6	83
31	Infectious prions do not induce AÎ² deposition in an in vivo seeding model. Acta Neuropathologica, 2018, 135, 965-967.	7.7	8
32	Innate immune memory in the brain shapes neurological disease hallmarks. Nature, 2018, 556, 332-338.	27.8	605
33	<scp>CSF</scp> progranulin increases in the course of Alzheimer's disease and is associated with <scp>sTREM</scp> 2, neurodegeneration and cognitive decline. EMBO Molecular Medicine, 2018, 10, .	6.9	64
34	Relationship between physical activity, cognition, and Alzheimer pathology in autosomal dominant Alzheimer's disease. Alzheimer's and Dementia, 2018, 14, 1427-1437.	0.8	51
35	Propagation and spread of pathogenic protein assemblies in neurodegenerative diseases. Nature Neuroscience, 2018, 21, 1341-1349.	14.8	289
36	Longitudinal cognitive and biomarker changes in dominantly inherited Alzheimer disease. Neurology, 2018, 91, e1295-e1306.	1.1	193

#	Article	IF	Citations
37	The Exceptional Vulnerability of Humans to Alzheimerâ€™s Disease. Trends in Molecular Medicine, 2017, 23, 534-545.	6.7	74
38	Microglia turnover with aging and in an Alzheimer's model via long-term in vivo single-cell imaging. Nature Neuroscience, 2017, 20, 1371-1376.	14.8	277
39	Amyloid polymorphisms constitute distinct clouds of conformational variants in different etiological subtypes of Alzheimerâ€™s disease. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, 13018-13023.	7.1	170
40	AÎ² seeding potency peaks in the early stages of cerebral Î²â€amyloidosis. EMBO Reports, 2017, 18, 1536-1544.	4.5	38
41	AÎ² seeds and prions: How close the fit?. Prion, 2017, 11, 215-225.	1.8	29
42	Prevention of tau increase in cerebrospinal fluid of APP transgenic mice suggests downstream effect of BACE1 inhibition. Alzheimer's and Dementia, 2017, 13, 701-709.	0.8	35
43	Pharmacological BACE1 and BACE2 inhibition induces hair depigmentation by inhibiting PMEL17 processing in mice. Scientific Reports, 2016, 6, 21917.	3.3	56
44	Highly potent intracellular membrane-associated AÎ² seeds. Scientific Reports, 2016, 6, 28125.	3.3	18
45	Conversion of Synthetic AÎ² toIn VivoActive Seeds and Amyloid Plaque Formation in a Hippocampal Slice Culture Model. Journal of Neuroscience, 2016, 36, 5084-5093.	3.6	41
46	Immune receptor for pathogenic Î±-synuclein. Science, 2016, 353, 1498-1499.	12.6	5
47	Neurofilament Light Chain in Blood and CSF as Marker of Disease Progression in Mouse Models and in Neurodegenerative Diseases. Neuron, 2016, 91, 56-66.	8.1	289
48	The Prion-Like Properties of Amyloid-Î² Assemblies: Implications for Alzheimer's Disease. Cold Spring Harbor Perspectives in Medicine, 2016, 6, a024398.	6.2	71
49	The Malignant Protein Puzzle. Cerebrum: the Dana Forum on Brain Science, 2016, 2016, .	0.1	0
50	Distinct Spacing Between Anionic Groups: An Essential Chemical Determinant for Achieving Thiopheneâ€Based Ligands to Distinguish Î²â€Amyloid or Tau Polymorphic Aggregates. Chemistry - A European Journal, 2015, 21, 9072-9082.	3.3	44
51	Increased <scp>CSF</scp> AÎ² during the very early phase of cerebral AÎ² deposition in mouse models. EMBO Molecular Medicine, 2015, 7, 895-903.	6.9	42
52	No Dopamine Cell Loss or Changes in Cytoskeleton Function in Transgenic Mice Expressing Physiological Levels of Wild Type or G2019S Mutant LRRK2 and in Human Fibroblasts. PLoS ONE, 2015, 10, e0118947.	2.5	24
53	Cerebral Î²-Amyloidosis in Mice Investigated by Ultramicroscopy. PLoS ONE, 2015, 10, e0125418.	2.5	26
54	Diagnostic Value of Subjective Memory Complaints Assessed with a Single Item in Dominantly Inherited Alzheimerâ€™s Disease: Results of the DIAN Study. BioMed Research International, 2015, 2015, 1-7.	1.9	7

#	Article	IF	Citations
55	Formaldehyde-fixed brain tissue from spontaneously ill Î±-synuclein transgenic mice induces fatal Î±-synucleinopathy in transgenic hosts. Acta Neuropathologica, 2015, 129, 157-159.	7.7	34
56	Progression of Seedâ€induced <scp>A</scp>Î² Deposition within the Limbic Connectome. Brain Pathology, 2015, 25, 743-752.	4.1	45
57	Endogenous murine AÎ² increases amyloid deposition in APP23 but not in APPPS1 transgenic mice. Neurobiology of Aging, 2015, 36, 2241-2247.	3.1	9
58	Neurodegenerative Diseases: Expanding the Prion Concept. Annual Review of Neuroscience, 2015, 38, 87-103.	10.7	278
59	Cerebral amyloidosis associated with cognitive decline in autosomal dominant Alzheimer disease. Neurology, 2015, 85, 790-798.	1.1	27
60	Replacement of brain-resident myeloid cells does not alter cerebral amyloid-Î² deposition in mouse models of Alzheimerâ€™s disease. Journal of Experimental Medicine, 2015, 212, 1803-1809.	8.5	81
61	Amyloid-Î² pathology induced in humans. Nature, 2015, 525, 193-194.	27.8	43
62	Persistence of AÎ² seeds in APP null mouse brain. Nature Neuroscience, 2015, 18, 1559-1561.	14.8	51
63	Blood Platelets in the Progression of Alzheimerâ€™s Disease. PLoS ONE, 2014, 9, e90523.	2.5	111
64	AÎ² seeds resist inactivation by formaldehyde. Acta Neuropathologica, 2014, 128, 477-484.	7.7	58
65	Multiple Factors Contribute to the Peripheral Induction of Cerebral Â-Amyloidosis. Journal of Neuroscience, 2014, 34, 10264-10273.	3.6	76
66	Longitudinal PET-MRI reveals Î²-amyloid deposition and rCBF dynamics and connects vascular amyloidosis to quantitative loss of perfusion. Nature Medicine, 2014, 20, 1485-1492.	30.7	108
67	Highly potent soluble amyloid-Î² seeds in human Alzheimer brain but not cerebrospinal fluid. Brain, 2014, 137, 2909-2915.	7.6	61
68	Homeostatic and injuryâ€induced microglia behavior in the aging brain. Aging Cell, 2014, 13, 60-69.	6.7	259
69	Self-propagation of pathogenic protein aggregates in neurodegenerative diseases. Nature, 2013, 501, 45-51.	27.8	1,331
70	Evidence for Age-Dependent in Vivo Conformational Rearrangement within AÎ² Amyloid Deposits. ACS Chemical Biology, 2013, 8, 1128-1133.	3.4	93
71	Changes in Amyloid-Î² and Tau in the Cerebrospinal Fluid of Transgenic Mice Overexpressing Amyloid Precursor Protein. Science Translational Medicine, 2013, 5, 194re2.	12.4	166
72	Immunization targeting a minor plaque constituent clears Î²-amyloid and rescues behavioral deficits in an Alzheimer's disease mouse model. Neurobiology of Aging, 2013, 34, 137-145.	3.1	33

#	Article	IF	Citations
73	CSF biomarker variability in the Alzheimer's Association quality control program. Alzheimer's and Dementia, 2013, 9, 251-261.	0.8	344
74	The Prion-Like Aspect of Alzheimer Pathology. Research and Perspectives in Alzheimer's Disease, 2013, , 61-69.	0.1	2
75	Membrane-Anchored AÎ² Accelerates Amyloid Formation and Exacerbates Amyloid-Associated Toxicity in Mice. Journal of Neuroscience, 2013, 33, 19284-19294.	3.6	30
76	Comment on â€œApoE-Directed Therapeutics Rapidly Clear Î²-Amyloid and Reverse Deficits in AD Mouse Modelsâ€. Science, 2013, 340, 924-924.	12.6	136
77	Seeded strainâ€like transmission of Î²â€amyloid morphotypes in APP transgenic mice. EMBO Reports, 2013, 14, 1017-1022.	4.5	118
78	Microglial repopulation model reveals a robust homeostatic process for replacing CNS myeloid cells. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 18150-18155.	7.1	210
79	Spectral Discrimination of Cerebral Amyloid Lesions after Peripheral Application of Luminescent Conjugated Oligothiophenes. American Journal of Pathology, 2012, 181, 1953-1960.	3.8	36
80	The Amyloid State of Proteins in Human Diseases. Cell, 2012, 148, 1188-1203.	28.9	1,496
81	Repeatable target localization for long-term in vivo imaging of mice with 2-photon microscopy. Journal of Neuroscience Methods, 2012, 205, 357-363.	2.5	29
82	Exogenous seeding of cerebral Î²â€amyloid deposition in Î²APPâ€transgenic rats. Journal of Neurochemistry, 2012, 120, 660-666.	3.9	111
83	The presence of AÎ² seeds, and not age per se, is critical to the initiation of AÎ² deposition in the brain. Acta Neuropathologica, 2012, 123, 31-37.	7.7	91
84	BRI2 Protein Regulates Î²-Amyloid Degradation by Increasing Levels of Secreted Insulin-degrading Enzyme (IDE). Journal of Biological Chemistry, 2011, 286, 37446-37457.	3.4	37
85	Soluble AÎ² Seeds Are Potent Inducers of Cerebral Î²-Amyloid Deposition. Journal of Neuroscience, 2011, 31, 14488-14495.	3.6	203
86	Early onset amyloid lesions lead to severe neuritic abnormalities and local, but not global neuron loss in APPPS1 transgenic mice. Neurobiology of Aging, 2011, 32, 2324.e1-2324.e6.	3.1	67
87	Amyloid by default. Nature Neuroscience, 2011, 14, 669-670.	14.8	28
88	The Alzheimer's Association external quality control program for cerebrospinal fluid biomarkers. Alzheimer's and Dementia, 2011, 7, 386.	0.8	354
89	Pathogenic protein seeding in alzheimer disease and other neurodegenerative disorders. Annals of Neurology, 2011, 70, 532-540.	5.3	536
90	Long-TermIn VivoImaging of Î²-Amyloid Plaque Appearance and Growth in a Mouse Model of Cerebral Î²-Amyloidosis. Journal of Neuroscience, 2011, 31, 624-629.	3.6	126

#	Article	IF	Citations
91	The benefits and limitations of animal models for translational research in neurodegenerative diseases. Nature Medicine, 2010, 16, 1210-1214.	30.7	301
92	Modeling familial Danish dementia in mice supports the concept of the amyloid hypothesis of Alzheimer's disease. Proceedings of the National Academy of Sciences of the United States of America, 2010, 107, 7969-7974.	7.1	65
93	Peripherally Applied AÎ²-Containing Inoculates Induce Cerebral Î²-Amyloidosis. Science, 2010, 330, 980-982.	12.6	519
94	Induction of cerebral Î²-amyloidosis: Intracerebral versus systemic AÎ² inoculation. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 12926-12931.	7.1	249
95	Transmission and spreading of tauopathy in transgenic mouse brain. Nature Cell Biology, 2009, 11, 909-913.	10.3	1,515
96	Formation and maintenance of Alzheimer's disease Î²-amyloid plaques in the absence of microglia. Nature Neuroscience, 2009, 12, 1361-1363.	14.8	390
97	Novel Pentameric Thiophene Derivatives for in Vitro and in Vivo Optical Imaging of a Plethora of Protein Aggregates in Cerebral Amyloidoses. ACS Chemical Biology, 2009, 4, 673-684.	3.4	290
98	E22Q-Mutant AÎ² Peptide (AÎ²Dutch) Increases Vascular but Reduces Parenchymal AÎ² Deposition. American Journal of Pathology, 2009, 174, 722-726.	3.8	15
99	Independent Effects of Intra- and Extracellular AÎ² on Learning-Related Gene Expression. American Journal of Pathology, 2009, 175, 271-282.	3.8	35
100	The value of incomplete mouse models of Alzheimerâ€™s disease. European Journal of Nuclear Medicine and Molecular Imaging, 2008, 35, 70-74.	6.4	79
101	Cerebral and Peripheral Amyloid Phagocytesâ€” an Old Liaison with a New Twist. Neuron, 2008, 59, 8-10.	8.1	29
102	BACE1 and Mutated Presenilin-1 Differently Modulate AÎ²40 and AÎ²42 Levels and Cerebral Amyloidosis in APPDutch Transgenic Mice. Neurodegenerative Diseases, 2007, 4, 127-135.	1.4	19
103	Vessel ultrastructure in APP23 transgenic mice after passive anti-AÎ² immunotherapy and subsequent intracerebral hemorrhage. Neurobiology of Aging, 2007, 28, 202-212.	3.1	34
104	Induction of Tau Pathology by Intracerebral Infusion of Amyloid-Î²-Containing Brain Extract and by Amyloid-Î² Deposition in APP Ã— Tau Transgenic Mice. American Journal of Pathology, 2007, 171, 2012-2020.	3.8	239
105	Cystatin C modulates cerebral Î²-amyloidosis. Nature Genetics, 2007, 39, 1437-1439.	21.4	151
106	Exogenous Induction of Cerebral ÃŸ-Amyloidogenesis Is Governed by Agent and Host. Science, 2006, 313, 1781-1784.	12.6	875
107	Inducible proteopathies. Trends in Neurosciences, 2006, 29, 438-443.	8.6	92
108	Mechanism of Cerebral Î²-Amyloid Angiopathy: Murine and Cellular Models. Brain Pathology, 2006, 16, 40-54.	4.1	150

#	Article	IF	Citations
109	AÎ²42â€driven cerebral amyloidosis in transgenic mice reveals early and robust pathology. EMBO Reports, 2006, 7, 940-946.	4.5	832
110	Kochâ€™s postulates and infectious proteins. Acta Neuropathologica, 2006, 112, 1-4.	7.7	69
111	Invasion of Hematopoietic Cells into the Brain of Amyloid Precursor Protein Transgenic Mice. Journal of Neuroscience, 2005, 25, 11125-11132.	3.6	164
112	AÎ² is targeted to the vasculature in a mouse model of hereditary cerebral hemorrhage with amyloidosis. Nature Neuroscience, 2004, 7, 954-960.	14.8	367
113	Extracellular amyloid formation and associated pathology in neural grafts. Nature Neuroscience, 2003, 6, 370-377.	14.8	115
114	Amyloid-Associated Neuron Loss and Gliogenesis in the Neocortex of Amyloid Precursor Protein Transgenic Mice. Journal of Neuroscience, 2002, 22, 515-522.	3.6	199
115	Spontaneous Hemorrhagic Stroke in a Mouse Model of Cerebral Amyloid Angiopathy. Journal of Neuroscience, 2001, 21, 1619-1627.	3.6	281
116	Mechanisms of Cerebrovascular Amyloid Deposition: Lessons from Mouse Models. Annals of the New York Academy of Sciences, 2000, 903, 307-316.	3.8	62
117	Cerebral Amyloid Induces Aberrant Axonal Sprouting and Ectopic Terminal Formation in Amyloid Precursor Protein Transgenic Mice. Journal of Neuroscience, 1999, 19, 8552-8559.	3.6	183
118	Association of Microglia with Amyloid Plaques in Brains of APP23 Transgenic Mice. American Journal of Pathology, 1999, 154, 1673-1684.	3.8	304
119	Neuron loss in APP transgenic mice. Nature, 1998, 395, 755-756.	27.8	474
120	Murine models of brain aging and age-related neurodegenerative diseases. Behavioural Brain Research, 1997, 85, 1-25.	2.2	102
121	Alzheimer: Wie man nach einer kausalen Therapie sucht. , 0, , .		0