Thomas A Bayer

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Motor deficits, neuron loss, and reduced anxiety coinciding with axonal degeneration and intraneuronal AÎ ² aggregation in the 5XFAD mouse model of Alzheimer's disease. Neurobiology of Aging, 2012, 33, 196.e29-196.e40.	1.5	421
2	Massive CA1/2 Neuronal Loss with Intraneuronal and N-Terminal Truncated Aβ42 Accumulation in a Novel Alzheimer Transgenic Model. American Journal of Pathology, 2004, 165, 1289-1300.	1.9	375
3	A modified beta-amyloid hypothesis: intraneuronal accumulation of the beta-amyloid peptide - the first step of a fatal cascade. Journal of Neurochemistry, 2004, 91, 513-520.	2.1	344
4	Dietary Cu stabilizes brain superoxide dismutase 1 activity and reduces amyloid AÂ production in APP23 transgenic mice. Proceedings of the National Academy of Sciences of the United States of America, 2003, 100, 14187-14192.	3.3	330
5	Intraneuronal AÎ ² accumulation precedes plaque formation in Î ² -amyloid precursor protein and presenilin-1 double-transgenic mice. Neuroscience Letters, 2001, 306, 116-120.	1.0	323
6	Endothelial LRP1 transports amyloid-β1–42 across the blood-brain barrier. Journal of Clinical Investigation, 2015, 126, 123-136.	3.9	299
7	Time sequence of maturation of dystrophic neurites associated with Aβ deposits in APP/PS1 transgenic mice. Experimental Neurology, 2003, 184, 247-263.	2.0	257
8	Hippocampal Neuron Loss Exceeds Amyloid Plaque Load in a Transgenic Mouse Model of Alzheimer's Disease. American Journal of Pathology, 2004, 164, 1495-1502.	1.9	233
9	Immune hyperreactivity of $A\hat{l}^2$ plaque-associated microglia in Alzheimer's disease. Neurobiology of Aging, 2017, 55, 115-122.	1.5	205
10	Pyroglutamate Amyloid-β (Aβ): A Hatchet Man in Alzheimer Disease. Journal of Biological Chemistry, 2011, 286, 38825-38832.	1.6	177
11	Intracellular accumulation of amyloid-beta – a predictor for synaptic dysfunction and neuron loss in Alzheimer's disease. Frontiers in Aging Neuroscience, 2010, 2, 8.	1.7	161
12	Key Factors in Alzheimer's Disease: βâ€amyloid Precursor Protein Processing, Metabolism and Intraneuronal Transport. Brain Pathology, 2001, 11, 1-11.	2.1	159
13	N-truncated amyloid β (Aβ) 4-42 forms stable aggregates and induces acute and long-lasting behavioral deficits. Acta Neuropathologica, 2013, 126, 189-205.	3.9	153
14	Intraneuronal pyroglutamate-Abeta 3–42 triggers neurodegeneration and lethal neurological deficits in a transgenic mouse model. Acta Neuropathologica, 2009, 118, 487-496.	3.9	151
15	Clioquinol Mediates Copper Uptake and Counteracts Copper Efflux Activities of the Amyloid Precursor Protein of Alzheimer's Disease. Journal of Biological Chemistry, 2004, 279, 51958-51964.	1.6	138
16	Focusing the amyloid cascade hypothesis on N-truncated Abeta peptides as drug targets against Alzheimer's disease. Acta Neuropathologica, 2014, 127, 787-801.	3.9	129
17	Transient intraneuronal Aβ rather than extracellular plaque pathology correlates with neuron loss in the frontal cortex of APP/PS1KI mice. Acta Neuropathologica, 2008, 116, 647-655.	3.9	116
18	Intraneuronal APP/Aβ Trafficking and Plaque Formation in βâ€Amyloid Precursor Protein and Presenilinâ€1 Transgenic Mice. Brain Pathology, 2002, 12, 275-286.	2.1	113

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19	Axonopathy in an APP/PS1 transgenic mouse model of Alzheimer's disease. Acta Neuropathologica, 2006, 111, 312-319.	3.9	113
20	Inflammatory changes are tightly associated with neurodegeneration in the brain and spinal cord of the APP/PS1KI mouse model of Alzheimer's disease. Neurobiology of Aging, 2010, 31, 747-757.	1.5	111
21	Age-Related Loss of Synaptophysin Immunoreactive Presynaptic Boutons within the Hippocampus of APP751SL, PS1M146L, and APP751SL/PS1M146L Transgenic Mice. American Journal of Pathology, 2005, 167, 161-173.	1.9	107
22	Age-dependent axonal degeneration in an Alzheimer mouse model. Neurobiology of Aging, 2007, 28, 1689-1699.	1.5	107
23	Histone Deacetylase Inhibitor Valproic Acid Inhibits Cancer Cell Proliferation via Down-regulation of the Alzheimer Amyloid Precursor Protein. Journal of Biological Chemistry, 2010, 285, 10678-10689.	1.6	104
24	Accumulation of intraneuronal Aβ correlates with ApoE4 genotype. Acta Neuropathologica, 2010, 119, 555-566.	3.9	94
25	Intake of copper has no effect on cognition in patients with mild Alzheimer's disease: a pilot phase 2 clinical trial. Journal of Neural Transmission, 2008, 115, 1181-1187.	1.4	92
26	Identification of Low Molecular Weight Pyroglutamate Aβ Oligomers in Alzheimer Disease. Journal of Biological Chemistry, 2010, 285, 41517-41524.	1.6	91
27	Pyroglutamate Abeta pathology in APP/PS1KI mice, sporadic and familial Alzheimer's disease cases. Journal of Neural Transmission, 2010, 117, 85-96.	1.4	87
28	Prolonged Running, not Fluoxetine Treatment, Increases Neurogenesis, but does not Alter Neuropathology, in the 3xTg Mouse Model of Alzheimer's Disease. Current Topics in Behavioral Neurosciences, 2013, 15, 313-340.	0.8	85
29	Phosphorylation of the amyloid \hat{l}^2 -peptide at Ser26 stabilizes oligomeric assembly and increases neurotoxicity. Acta Neuropathologica, 2016, 131, 525-537.	3.9	84
30	Intraneuronal $A\hat{l}^2$ accumulation and neurodegeneration: Lessons from transgenic models. Life Sciences, 2012, 91, 1148-1152.	2.0	81
31	Gene Dosage Dependent Aggravation of the Neurological Phenotype in the 5XFAD Mouse Model of Alzheimer's Disease. Journal of Alzheimer's Disease, 2015, 45, 1223-1236.	1.2	80
32	Overexpression of Glutaminyl Cyclase, the Enzyme Responsible for Pyroglutamate AÎ ² Formation, Induces Behavioral Deficits, and Glutaminyl Cyclase Knock-out Rescues the Behavioral Phenotype in 5XFAD Mice. Journal of Biological Chemistry, 2011, 286, 4454-4460.	1.6	79
33	Early intraneuronal accumulation and increased aggregation of phosphorylated Abeta in a mouse model of Alzheimer's disease. Acta Neuropathologica, 2013, 125, 699-709.	3.9	79
34	Cognitive decline correlates with low plasma concentrations of copper in patients with mild to moderate Alzheimer's disease. Journal of Alzheimer's Disease, 2005, 8, 23-27.	1.2	78
35	Deciphering the Molecular Profile of Plaques, Memory Decline and Neuron Loss in Two Mouse Models for Alzheimerââ,¬â,,¢s Disease by Deep Sequencing. Frontiers in Aging Neuroscience, 2014, 6, 75.	1.7	78
36	Proteinopathies, a core concept for understanding and ultimately treating degenerative disorders?. European Neuropsychopharmacology, 2015, 25, 713-724.	0.3	78

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37	Diesel engine exhaust accelerates plaque formation in a mouse model of Alzheimer's disease. Particle and Fibre Toxicology, 2017, 14, 35.	2.8	77
38	Deficits in working memory and motor performance in the APP/PS1ki mouse model for Alzheimer's disease. Neurobiology of Aging, 2008, 29, 891-901.	1.5	75
39	APP/PS1KI bigenic mice develop early synaptic deficits and hippocampus atrophy. Acta Neuropathologica, 2009, 117, 677-685.	3.9	74
40	Pyroglutamate Amyloid β (Aβ) Aggravates Behavioral Deficits in Transgenic Amyloid Mouse Model for Alzheimer Disease. Journal of Biological Chemistry, 2012, 287, 8154-8162.	1.6	71
41	Environmental enrichment fails to rescue working memory deficits, neuron loss, and neurogenesis in APP/PS1KI mice. Neurobiology of Aging, 2012, 33, 96-107.	1.5	71
42	Intracellular Aβ triggers neuron loss in the cholinergic system of the APP/PS1KI mouse model of Alzheimer's disease. Neurobiology of Aging, 2010, 31, 1153-1163.	1.5	66
43	Neprilysin Deficiency Alters the Neuropathological and Behavioral Phenotype in the 5XFAD Mouse Model of Alzheimer's Disease. Journal of Alzheimer's Disease, 2015, 44, 1291-1302.	1.2	63
44	Amyloid Precursor Protein (APP) Mediated Regulation of Ganglioside Homeostasis Linking Alzheimer's Disease Pathology with Ganglioside Metabolism. PLoS ONE, 2012, 7, e34095.	1.1	61
45	Neuron Loss in Transgenic Mouse Models of Alzheimer's Disease. International Journal of Alzheimer's Disease, 2010, 2010, 1-6.	1.1	57
46	Accelerated tau pathology with synaptic and neuronal loss in a novel triple transgenic mouse model of Alzheimer's disease. Neurobiology of Aging, 2013, 34, 2564-2573.	1.5	55
47	Abeta targets of the biosimilar antibodies of Bapineuzumab, Crenezumab, Solanezumab in comparison to an antibody against N-truncated Abeta in sporadic Alzheimer disease cases and mouse models. Acta Neuropathologica, 2015, 130, 713-729.	3.9	53
48	Effect of copper intake on CSF parameters in patients with mild Alzheimer's disease: a pilot phaseÂ2 clinical trial. Journal of Neural Transmission, 2008, 115, 1651-1659.	1.4	52
49	18F-FDG-PET Detects Drastic Changes in Brain Metabolism in the Tg4–42 Model of Alzheimer's Disease. Frontiers in Aging Neuroscience, 2018, 10, 425.	1.7	49
50	Copper and clioquinol treatment in young APP transgenic and wild-type mice: effects on life expectancy, body weight, and metal-ion levels. Journal of Molecular Medicine, 2007, 85, 405-413.	1.7	42
51	Analysis of Motor Function in the Tg4-42 Mouse Model of Alzheimer's Disease. Frontiers in Behavioral Neuroscience, 2019, 13, 107.	1.0	41
52	The Arctic AβPP mutation leads to Alzheimer's disease pathology with highly variable topographic deposition of differentially truncated Aβ. Acta Neuropathologica Communications, 2013, 1, 60.	2.4	38
53	No alterations of hippocampal neuronal number and synaptic bouton number in a transgenic mouse model expressing the β-cleaved C-terminal APP fragment. Neurobiology of Disease, 2003, 12, 110-120.	2.1	37
54	N-truncated Abeta starting with position four: early intraneuronal accumulation and rescue of toxicity using NT4X-167, a novel monoclonal antibody. Acta Neuropathologica Communications, 2013, 1, 56.	2.4	36

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55	Pyroglutamate Aβ cascade as drug target in Alzheimer's disease. Molecular Psychiatry, 2022, 27, 1880-1885.	4.1	36
56	Expression of the Alzheimer's Disease Mutations AβPP695sw and PSEN1M146I in Double-Transgenic GA¶ttingen Minipigs. Journal of Alzheimer's Disease, 2016, 53, 1617-1630.	1.2	35
57	Concomitant detection of β-amyloid peptides with N-terminal truncation and different C-terminal endings in cortical plaques from cases with Alzheimer's disease, senile monkeys and triple transgenic mice. Journal of Chemical Neuroanatomy, 2010, 40, 82-92.	1.0	34
58	Alzheimer therapy with an antibody against N-terminal Abeta 4-X and pyroglutamate Abeta 3-X. Scientific Reports, 2015, 5, 17338.	1.6	34
59	Loss of Munc18-1 long splice variant in GABAergic terminals is associated with cognitive decline and increased risk of dementia in a community sample. Molecular Neurodegeneration, 2015, 10, 65.	4.4	34
60	N-truncated Aβ4–x peptides in sporadic Alzheimer's disease cases and transgenic Alzheimer mouse models. Alzheimer's Research and Therapy, 2017, 9, 80.	3.0	34
61	Cellular Copper Import by Nanocarrier Systems, Intracellular Availability, and Effects on Amyloid β Peptide Secretion. Biochemistry, 2009, 48, 4273-4284.	1.2	33
62	Intraneuronal $A\hat{l}^2$ as a trigger for neuron loss: can this be translated into human pathology?. Biochemical Society Transactions, 2011, 39, 857-861.	1.6	33
63	AβPP Accumulation and/or Intraneuronal Amyloid-β Accumulation? The 3xTg-AD Mouse Model Revisited. Journal of Alzheimer's Disease, 2012, 28, 897-904.	1.2	33
64	No improvement after chronic ibuprofen treatment in the 5XFAD mouse model of Alzheimer's disease. Neurobiology of Aging, 2012, 33, 833.e39-833.e50.	1.5	32
65	Formic acid is essential for immunohistochemical detection of aggregated intraneuronal Aβ peptides in mouse models of Alzheimer's disease. Brain Research, 2009, 1301, 116-125.	1.1	31
66	Deposition of C-terminally truncated Aβ species Aβ37 and Aβ39 in Alzheimer's disease and transgenic mouse models. Acta Neuropathologica Communications, 2016, 4, 24.	2.4	29
67	Disturbed Copper Bioavailability in Alzheimer's Disease. International Journal of Alzheimer's Disease, 2011, 2011, 1-5.	1.1	28
68	Frontotemporal dysregulation of the SNARE protein interactome is associated with faster cognitive decline in old age. Neurobiology of Disease, 2018, 114, 31-44.	2.1	27
69	Aβ38 in the Brains of Patients with Sporadic and Familial Alzheimer's Disease and Transgenic Mouse Models. Journal of Alzheimer's Disease, 2014, 39, 871-881.	1.2	25
70	Reduced levels of IgM autoantibodies against N-truncated pyroglutamate Aβ in plasma of patients with Alzheimer's disease. Neurobiology of Aging, 2011, 32, 1379-1387.	1.5	23
71	In vivo Imaging With 18F-FDG- and 18F-Florbetaben-PET/MRI Detects Pathological Changes in the Brain of the Commonly Used 5XFAD Mouse Model of Alzheimer's Disease. Frontiers in Medicine, 2020, 7, 529.	1.2	23
72	Circulating immune complexes of Aβ and IgM in plasma of patients with Alzheimer's disease. Journal of Neural Transmission, 2009, 116, 913-920.	1.4	22

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73	Synaptic Alterations in Mouse Models for Alzheimer Disease—A Special Focus on N-Truncated Abeta 4-42. Molecules, 2018, 23, 718.	1.7	20
74	Nâ€ŧerminal heterogeneity of parenchymal and vascular amyloidâ€Î² deposits in Alzheimer's disease. Neuropathology and Applied Neurobiology, 2020, 46, 673-685.	1.8	20
75	Shotgun Brain Proteomics Reveals Early Molecular Signature in Presymptomatic Mouse Model of Alzheimer's Disease. Journal of Alzheimer's Disease, 2013, 37, 297-308.	1.2	19
76	Abundance of Aβ5-xlike immunoreactivity in transgenic 5XFAD, APP/PS1KI and 3xTG mice, sporadic and familial Alzheimer's disease. Molecular Neurodegeneration, 2014, 9, 13.	4.4	19
77	Age-dependent loss of dentate gyrus granule cells in APP/PS1KI mice. Brain Research, 2008, 1222, 207-213.	1.1	18
78	Intraneuronal β-Amyloid Is a Major Risk Factor – Novel Evidence from the APP/PS1KI Mouse Model. Neurodegenerative Diseases, 2008, 5, 140-142.	0.8	18
79	Oligomeric Pyroglutamate Amyloid-β is Present in Microglia and a Subfraction of Vessels in Patients with Alzheimer's Disease: Implications for Immunotherapy. Journal of Alzheimer's Disease, 2013, 35, 741-749.	1.2	18
80	Antibody 9D5 Recognizes Oligomeric Pyroglutamate Amyloid-β in a Fraction of Amyloid-β Deposits in Alzheimer's Disease without Cross-Reactivity with other Protein Aggregates. Journal of Alzheimer's Disease, 2012, 29, 361-371.	1.2	17
81	Decreased cortical FADD protein is associated with clinical dementia and cognitive decline in an elderly community sample. Molecular Neurodegeneration, 2017, 12, 26.	4.4	17
82	<scp>SUMO</scp> 1 onjugation is altered during normal aging but not by increased amyloid burden. Aging Cell, 2018, 17, e12760.	3.0	15
83	I716F AβPP Mutation Associates with the Deposition of Oligomeric Pyroglutamate Amyloid-β and α-Synucleinopathy with Lewy Bodies. Journal of Alzheimer's Disease, 2015, 44, 103-114.	1.2	13
84	Amyloid Precursor Protein Is a Biomarker for Transformed Human Pluripotent Stem Cells. American Journal of Pathology, 2012, 180, 1636-1652.	1.9	12
85	Gene Expression Profiling in the APP/PS1KI Mouse Model of Familial Alzheimer's Disease. Journal of Alzheimer's Disease, 2016, 50, 397-409.	1.2	12
86	Super-Resolution Microscopy of Cerebrospinal Fluid Biomarkers as a Tool forÂAlzheimer's Disease Diagnostics. Journal of Alzheimer's Disease, 2015, 46, 1007-1020.	1.2	12
87	Neuron Loss and Behavioral Deficits in the TBA42 Mouse Model Expressing N-Truncated Pyroglutamate Amyloid-β3–42. Journal of Alzheimer's Disease, 2015, 45, 471-482.	1.2	12
88	Synergistic Effect on Neurodegeneration by N-Truncated Aβ4â^'42 and Pyroglutamate Aβ3â^'42 in a Mouse Model of Alzheimer's Disease. Frontiers in Aging Neuroscience, 2018, 10, 64.	1.7	11
89	Discovery of a novel pseudo β-hairpin structure of N-truncated amyloid-β for use as a vaccine against Alzheimer's disease. Molecular Psychiatry, 2021, , .	4.1	11
90	N-Terminal Truncated Aβ4-42 Is a Substrate for Neprilysin Degradation in vitro and in vivo. Journal of Alzheimer's Disease, 2019, 67, 849-858.	1.2	10

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91	Search strategy analysis of Tg4-42 Alzheimer Mice in the Morris Water Maze reveals early spatial navigation deficits. Scientific Reports, 2022, 12, 5451.	1.6	10
92	N-Truncated Aβ2-X Starting with Position Two in Sporadic Alzheimer's Disease Cases and Two Alzheimer Mouse Models. Journal of Alzheimer's Disease, 2015, 49, 101-110.	1.2	9
93	Reduced Acoustic Startle Response and Prepulse Inhibition in the Tg4-42 Model of Alzheimer's Disease. Journal of Alzheimer's Disease Reports, 2019, 3, 269-278.	1.2	9
94	N-Truncated Aβ Starting at Position Four—Biochemical Features, Preclinical Models, and Potential as Drug Target in Alzheimer's Disease. Frontiers in Aging Neuroscience, 2021, 13, 710579.	1.7	9
95	Donanemab detects a minor fraction of amyloid-β plaques in post-mortem brain tissue of patients with Alzheimer's disease and Down syndrome. Acta Neuropathologica, 2022, 143, 601-603.	3.9	9
96	Altered cholesterol metabolism in APP695-transfected neuroblastoma cells. Brain Research, 2007, 1152, 209-214.	1.1	6
97	miRNA Alterations Elicit Pathways Involved in Memory Decline and Synaptic Function in the Hippocampus of Aged Tg4-42 Mice. Frontiers in Neuroscience, 2020, 14, 580524.	1.4	5
98	Transgene integration causes RARB downregulation in homozygous Tg4–42 mice. Scientific Reports, 2020, 10, 6377.	1.6	5
99	Metabolic, Phenotypic, and Neuropathological Characterization of the Tg4-42 Mouse Model for Alzheimer's Disease. Journal of Alzheimer's Disease, 2021, 80, 1151-1168.	1.2	5
100	Immunotherapy Against N-Truncated Amyloid-β Oligomers. Methods in Pharmacology and Toxicology, 2016, , 37-50.	0.1	3
101	Small RNA Sequencing in the Tg4–42 Mouse Model Suggests the Involvement of snoRNAs in the Etiology of Alzheimer's Disease. Journal of Alzheimer's Disease, 2022, 87, 1671-1681.	1.2	2
102	New insights into Alzheimer's disease: â€~modeling neurodegeneration - causes and consequences'. Genes, Brain and Behavior, 2008, 7, iv-iv.	1.1	1
103	Die modifizierte Amyloid-Hypothese der Alzheimer-Demenz – intraneuronales Abeta induziert Neurodegeneration. E-Neuroforum, 2009, 15, 76-83.	0.2	0
104	Problems During Aging (Alzheimer's and Others). , 2013, , 2953-2969.		0
105	P1-422: THE IMPACT OF PASSSIVE IMMUNIZATION AGAINST N-TERMINALLY TRUNCATED AB SPECIES: A COMPARATIVE STUDY IN THE 5XFAD ALZHEIMER'S MODEL. , 2014, 10, P468-P468.		0
106	O4â€11â€05: Endothelialâ€LRP1 Clears Major Amounts of Abeta 1â€42 Across the Bloodâ€Brain Barrier. Alzheimer's and Dementia, 2016, 12, P361.	0.4	0
107	P1â€156: Abeta Plaqueâ€Associated Microglia Priming in Alzheimer's Disease. Alzheimer's and Dementia, 2016, 12, P462.	0.4	0
108	[P4–393]: COEXPRESSION OF THE TWO Nâ€TRUNCATED PEPTIDES Aβ _{3(PE)â€42} AND Aβ _{4–42} AGGRAVATES THE BEHAVIORAL PHENOTYPE IN TRANSGENIC AMYLOID MOUSE MODELS FOR ALZHEIMER'S DISEASE. Alzheimer's and Dementia, 2017, 13, P1478.	0.4	0

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109	Effects of a WIN 55,212â€2â€based therapy in two mouse models of Alzheimer's disease. Alzheimer's and Dementia, 2020, 16, e043211.	0.4	ο