Karen Duff

List of Publications by Year
in descending order

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75 papers	18,164 citations	36303 51 h-index	91884 69 g-index
75 all docs	75 docs citations	75 times ranked	15385 citing authors

#	Article	IF	Citations
1	AÎ² peptide vaccination prevents memory loss in an animal model of Alzheimer's disease. Nature, 2000, 408, 982-985.	27.8	1,506
2	Increased amyloid-Î²42(43) in brains of mice expressing mutant presenilin 1. Nature, 1996, 383, 710-713.	27.8	1,480
3	Accelerated Alzheimer-type phenotype in transgenic mice carrying both mutant amyloid precursor protein and presenilin 1 transgenes. Nature Medicine, 1998, 4, 97-100.	30.7	1,288
4	Neurofibrillary tangles, amyotrophy and progressive motor disturbance in mice expressing mutant (P301L) tau protein. Nature Genetics, 2000, 25, 402-405.	21.4	1,254
5	Hypercholesterolemia Accelerates the Alzheimer's Amyloid Pathology in a Transgenic Mouse Model. Neurobiology of Disease, 2000, 7, 321-331.	4.4	964
6	Macroautophagyâ€"a novel Î²-amyloid peptide-generating pathway activated in Alzheimer's disease. Journal of Cell Biology, 2005, 171, 87-98.	5.2	891
7	Loss of Presenilin Function Causes Impairments of Memory and Synaptic Plasticity Followed by Age-Dependent Neurodegeneration. Neuron, 2004, 42, 23-36.	8.1	701
8	Hyperphosphorylation and aggregation of tau in mice expressing normal human tau isoforms. Journal of Neurochemistry, 2003, 86, 582-590.	3.9	662
9	Inhibition of glycogen synthase kinase-3 by lithium correlates with reduced tauopathy and degenerationin vivo. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 6990-6995.	7.1	649
10	Fibrillar amyloid deposition leads to local synaptic abnormalities and breakage of neuronal branches. Nature Neuroscience, 2004, 7, 1181-1183.	14.8	497
11	Cell-Cycle Reentry and Cell Death in Transgenic Mice Expressing Nonmutant Human Tau Isoforms. Journal of Neuroscience, 2005, 25, 5446-5454.	3.6	481
12	Cdk5 Is a Key Factor in Tau Aggregation and Tangle Formation In Vivo. Neuron, 2003, 38, 555-565.	8.1	474
13	Linking AÎ² and Tau in Late-Onset Alzheimer's Disease: A Dual Pathway Hypothesis. Neuron, 2008, 60, 534-542.	8.1	465
14	Novel Therapeutic Approach for the Treatment of Alzheimer's Disease by Peripheral Administration of Agents with an Affinity to Î²-Amyloid. Journal of Neuroscience, 2003, 23, 29-33.	3.6	332
15	Behavioral changes in transgenic mice expressing both amyloid precursor protein and presenilin-1 mutations: lack of association with amyloid deposits. Behavior Genetics, 1999, 29, 177-185.	2.1	320
16	Genetic dissection of Alzheimer's disease and related dementias: amyloid and its relationship to tau. Nature Neuroscience, 1998, 1, 355-358.	14.8	310
17	Age-Dependent Impairment of Cognitive and Synaptic Function in the htau Mouse Model of Tau Pathology. Journal of Neuroscience, 2009, 29, 10741-10749.	3.6	306
18	A Loss of Function Mutation of Presenilin-2 Interferes with Amyloid Î²-Peptide Production and Notch Signaling. Journal of Biological Chemistry, 1999, 274, 28669-28673.	3.4	279

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19	Inflammatory Responses to Amyloidosis in a Transgenic Mouse Model of Alzheimerâ€™s Disease. American Journal of Pathology, 2001, 158, 1345-1354.	3.8	275
20	Detection of Alzheimer's amyloid in transgenic mice using magnetic resonance microimaging. Magnetic Resonance in Medicine, 2003, 50, 293-302.	3.0	267
21	5â€² Splice Site Mutations in tau Associated with the Inherited Dementia FTDP-17 Affect a Stem-Loop Structure That Regulates Alternative Splicing of Exon 10. Journal of Biological Chemistry, 1999, 274, 15134-15143.	3.4	266
22	A mutation in AlzheimerÊ¼s disease destroying a splice acceptor site in the presenilin-1 gene. NeuroReport, 1995, 7, 297-301.	1.2	262
23	Retromer deficiency observed in Alzheimer's disease causes hippocampal dysfunction, neurodegeneration, and AÎ² accumulation. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 7327-7332.	7.1	230
24	Age-Related Amyloid Î² Deposition in Transgenic Mice Overexpressing Both Alzheimer Mutant Presenilin 1 and Amyloid Î² Precursor Protein Swedish Mutant Is Not Associated with Global Neuronal Loss. American Journal of Pathology, 2000, 157, 331-339.	3.8	222
25	A Technique for Serial Collection of Cerebrospinal Fluid from the Cisterna Magna in Mouse. Journal of Visualized Experiments, 2008, , .	0.3	207
26	Reorganization of Cholinergic Terminals in the Cerebral Cortex and Hippocampus in Transgenic Mice Carrying Mutated Presenilin-1 and Amyloid Precursor Protein Transgenes. Journal of Neuroscience, 1999, 19, 2706-2716.	3.6	193
27	Contrasting Pathology of the Stress Granule Proteins TIA-1 and G3BP in Tauopathies. Journal of Neuroscience, 2012, 32, 8270-8283.	3.6	186
28	Presenilin Redistribution Associated with Aberrant Cholesterol Transport Enhances Î²-Amyloid ProductionIn Vivo. Journal of Neuroscience, 2003, 23, 5645-5649.	3.6	170
29	Dense-Core Plaques in Tg2576 and PSAPP Mouse Models of Alzheimer's Disease Are Centered on Vessel Walls. American Journal of Pathology, 2005, 167, 527-543.	3.8	168
30	The phospholipase D1 pathway modulates macroautophagy. Nature Communications, 2010, 1, 142.	12.8	161
31	Collapsin response mediator proteinâ€2 hyperphosphorylation is an early event in Alzheimerâ€™s disease progression. Journal of Neurochemistry, 2007, 103, 1132-1144.	3.9	158
32	Statin therapy for Alzheimerâ€™s disease. Journal of Molecular Neuroscience, 2002, 19, 155-161.	2.3	152
33	Altered Calcium Homeostasis and Mitochondrial Dysfunction in Cortical Synaptic Compartments of Presenilin-1 Mutant Mice. Journal of Neurochemistry, 2008, 72, 1030-1039.	3.9	144
34	Presenilin Mutations in Familial Alzheimer Disease and Transgenic Mouse Models Accelerate Neuronal Lysosomal Pathology. Journal of Neuropathology and Experimental Neurology, 2004, 63, 821-830.	1.7	139
35	The amyloid pathology progresses in a neurotransmitter-specific manner. Neurobiology of Aging, 2006, 27, 1644-1657.	3.1	129
36	Increased AÎ²42(43) from cell lines expressing presenilin 1 mutations. Annals of Neurology, 1998, 43, 256-258.	5.3	117

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37	Cholesterol in Alzheimer's Disease and Tauopathy. Annals of the New York Academy of Sciences, 2002, 977, 367-375.	3.8	116
38	Amyloid-Î² Deposition Is Associated with Decreased Hippocampal Glucose Metabolism and Spatial Memory Impairment in APP/PS1 Mice. Journal of Neuropathology and Experimental Neurology, 2004, 63, 418-428.	1.7	111
39	Permeability of Proteins at the Bloodâ€“Brain Barrier in the Normal Adult Mouse and Double Transgenic Mouse Model of Alzheimer's Disease. Neurobiology of Disease, 2001, 8, 555-567.	4.4	110
40	Co-localization of cholesterol, apolipoprotein E and fibrillar AÎ² in amyloid plaques. Molecular Brain Research, 2003, 110, 119-125.	2.3	108
41	MRI assessment of neuropathology in a transgenic mouse model of Alzheimer's disease. Magnetic Resonance in Medicine, 2004, 51, 794-798.	3.0	108
42	Visualization of ?-amyloid plaques in a transgenic mouse model of Alzheimer's disease using MR microscopy without contrast reagents. Magnetic Resonance in Medicine, 2004, 52, 538-544.	3.0	99
43	Optical visualization of Alzheimerâ€™s pathology via multiphoton-excited intrinsic fluorescence and second harmonic generation. Optics Express, 2009, 17, 3679.	3.4	94
44	A transgenic rat that develops Alzheimer's disease-like amyloid pathology, deficits in synaptic plasticity and cognitive impairment. Neurobiology of Disease, 2008, 31, 46-57.	4.4	92
45	Transgenic mouse models of Alzheimer's disease: How useful have they been for therapeutic development?. Briefings in Functional Genomics & Proteomics, 2004, 3, 47-59.	3.8	90
46	Links Between the Pathology of Alzheimer's Disease and Vascular Dementia. Neurochemical Research, 2004, 29, 1257-1266.	3.3	89
47	Plaque-Induced Abnormalities in Neurite Geometry in Transgenic Models of Alzheimer Disease: Implications for Neural System Disruption. Journal of Neuropathology and Experimental Neurology, 2001, 60, 753-758.	1.7	88
48	Rat transgenic models with a phenotype of intracellular AÎ² accumulation in hippocampus and cortex. Journal of Alzheimer's Disease, 2004, 6, 209-219.	2.6	70
49	Functional Phenotype in Transgenic Mice Expressing Mutant Human Presenilin-1. Neurobiology of Disease, 2000, 7, 119-126.	4.4	68
50	Survival and plasticity of basal forebrain cholinergic systems in mice transgenic for presenilin-1 and amyloid precursor protein mutant genes. NeuroReport, 2001, 12, 1377-1384.	1.2	65
51	Non-invasive, Focused Ultrasound-Facilitated Gene Delivery for Optogenetics. Scientific Reports, 2017, 7, 39955.	3.3	53
52	Cholesterol Distribution, Not Total Levels, Correlate With Altered Amyloid Precursor Protein Processing in Statin-Treated Mice. NeuroMolecular Medicine, 2006, 8, 319-328.	3.4	52
53	Changes in Apolipoprotein E Expression in Response to Dietary and Pharmacological Modulation of Cholesterol. Journal of Molecular Neuroscience, 2003, 20, 395-406.	2.3	47
54	Rapid Neurofibrillary Tangle Formation after Localized Gene Transfer of Mutated Tau. American Journal of Pathology, 2004, 164, 347-353.	3.8	44

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55	Untangling memory deficits. Nature Medicine, 2005, 11, 826-827.	30.7	42
56	Organotypic Slice Cultures from Transgenic Mice as Disease Model Systems. Journal of Molecular Neuroscience, 2002, 19, 317-320.	2.3	32
57	Brain Damage Results in Down-Regulation of N-Acetylaspartate as a Neuronal Osmolyte. NeuroMolecular Medicine, 2003, 3, 95-104.	3.4	32
58	Mostly Separate Distributions of CLAC- versus AÎ²40- or Thioflavin S-Reactivities in Senile Plaques Reveal Two Distinct Subpopulations of Î²-Amyloid Deposits. American Journal of Pathology, 2004, 165, 273-281.	3.8	30
59	New directions for frontotemporal dementia drug discovery. , 2008, 4, 89-93.		29
60	Alzheimer transgenic mouse models come of age. Trends in Neurosciences, 1997, 20, 279-280.	8.6	28
61	Transgenic mouse models of Alzheimer's disease: phenotype and mechanisms of pathogenesis. Biochemical Society Symposia, 2001, 67, 195-202.	2.7	24
62	Using proteomics and network analysis to elucidate the consequences of synaptic protein oxidation in a PS1+AÎ²PP mouse model of Alzheimer's disease. Journal of Alzheimer's Disease, 2005, 8, 227-241.	2.6	24
63	Recent work on Alzheimer's disease transgenics. Current Opinion in Biotechnology, 1998, 9, 561-564.	6.6	21
64	Progress in the modeling of neurodegenerative diseases in transgenic mice. Current Opinion in Neurology, 2001, 14, 441-447.	3.6	21
65	Use of in vivo models to study the role of cholesterol in the etiology of Alzheimer's disease. Neurochemical Research, 2003, 28, 979-986.	3.3	21
66	Antibody against C-terminal Abeta selectively elevates plasma Abeta. NeuroReport, 2007, 18, 293-296.	1.2	17
67	Global Axonal Transport Rates are Unaltered in Htau Mice in vivo. Journal of Alzheimer's Disease, 2013, 37, 579-586.	2.6	14
68	An Aβ Sequestration Approach Using Non-Antibody Aβ Binding Agents. Current Alzheimer Research, 2005, 2, 265-268.	1.4	9
69	Normal and Abnormal Tau Neurobiology. Alzheimer Disease and Associated Disorders, 2006, 20, 202-205.	1.3	6
70	In Vivo Magnetic Resonance of Amyloid Plaques in Alzheimerâ€™s Disease Model Mice. , 2004, , 47-59.		3
71	Chapter 5.8 Transgenic mouse models of Alzheimer's disease. Handbook of Behavioral Neuroscience, 1999, , 880-894.	0.0	1
72	In Vivo Perturbation of Lysosomal Function Promotes Neurodegeneration in the PS1M146V/APPK670N,M671L Mouse Model of Alzheimer's Disease Pathology. , 0, , 687-695.		1

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73	Transgenic Mice Overexpressing Presenilin cDNAs. , 2000, , 123-128.		0
74	Cdk5 and Neuregulin-1 Signaling. , 2008, , 139-144.		0
75	Animal Models of Tauopathy. , 0, , 215-236.		0