David R Borchelt

List of Publications by Year
in descending order

192 papers	25,241 citations	11651 70 h-index	6836 155 g-index
198 all docs	198 docs citations	198 times ranked	19623 citing authors

#	Article	IF	Citations
1	Blood-based biomarkers of inflammation in amyotrophic lateral sclerosis. Molecular Neurodegeneration, 2022, 17, 11.	10.8	35
2	TAPPing into the potential of inducible tau/APP transgenic mice. Neuropathology and Applied Neurobiology, 2022, 48, .	3.2	3
3	Impact of APOE genotype on prion-type propagation of tauopathy. Acta Neuropathologica Communications, 2022, 10, 57.	5.2	4
4	Pathogenic tau recruits wild-type tau into brain inclusions and induces gut degeneration in transgenic SPAM mice. Communications Biology, 2022, 5, 446.	4.4	4
5	Soluble brain homogenates from diverse human and mouse sources preferentially seed diffuse AÎ² plaque pathology when injected into newborn mouse hosts.. Free Neuropathology, 2022, 3, .	3.0	2
6	Modeling the Competition between Misfolded AÎ² Conformers That Produce Distinct Types of Amyloid Pathology in Alzheimerâ€™s Disease. Biomolecules, 2022, 12, 886.	4.0	2
7	Building a Case for Withaferin A as a Treatment for FTD/ALS Syndromes. Neurotherapeutics, 2021, 18, 284-285.	4.4	1
8	Novel SOD1 monoclonal antibodies against the electrostatic loop preferentially detect misfolded SOD1 aggregates. Neuroscience Letters, 2021, 742, 135553.	2.1	1
9	Reactive astrocytes as treatment targets in Alzheimer's diseaseâ€”Systematic review of studies using the <scp>APPswePS1dE9</scp> mouse model. Glia, 2021, 69, 1852-1881.	4.9	37
10	Remodeling Alzheimer-amyloidosis models by seeding. Molecular Neurodegeneration, 2021, 16, 8.	10.8	7
11	Supercharging Prions via Amyloidâ€Selective Lysine Acetylation. Angewandte Chemie, 2021, 133, 15196-15206.	2.0	0
12	Variation in the vulnerability of mice expressing human superoxide dismutase 1 to prion-like seeding: a study of the influence of primary amino acid sequence. Acta Neuropathologica Communications, 2021, 9, 92.	5.2	5
13	Supercharging Prions via Amyloidâ€Selective Lysine Acetylation. Angewandte Chemie - International Edition, 2021, 60, 15069-15079.	13.8	2
14	Astrocytic apoE4 and tau: Deadly combination for neurons. Cell Reports Medicine, 2021, 2, 100316.	6.5	1
15	Collusion of Î±-Synuclein and AÎ² aggravating co-morbidities in a novel prion-type mouse model. Molecular Neurodegeneration, 2021, 16, 63.	10.8	12
16	Intracerebral Expression of AAV-APOE4 Is Not Sufficient to Alter Tau Burden in Two Distinct Models of Tauopathy. Molecular Neurobiology, 2020, 57, 1986-2001.	4.0	9
17	Phenotypic diversity in ALS and the role of poly-conformational protein misfolding. Acta Neuropathologica, 2020, 142, 41-55.	7.7	9
18	IL-10 based immunomodulation initiated at birth extends lifespan in a familial mouse model of amyotrophic lateral sclerosis. Scientific Reports, 2020, 10, 20862.	3.3	5

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19	Subcellular diversion of cholesterol by gainâ€•and lossâ€•ofâ€function mutations in <scp>PMP22</scp>. Glia, 2020, 68, 2300-2315.	4.9	11
20	Tryptophan residue 32 in human Cu-Zn superoxide dismutase modulates prion-like propagation and strain selection. PLoS ONE, 2020, 15, e0227655.	2.5	22
21	Therapeutic approaches targeting Apolipoprotein E function in Alzheimerâ€™s disease. Molecular Neurodegeneration, 2020, 15, 8.	10.8	89
22	Diversity in AÎ² deposit morphology and secondary proteome insolubility across models of Alzheimer-typeÂamyloidosis. Acta Neuropathologica Communications, 2020, 8, 43.	5.2	16
23	Comparative analyses of the in vivo induction and transmission of Î±-synuclein pathology in transgenic mice by MSA brain lysate and recombinant Î±-synuclein fibrils. Acta Neuropathologica Communications, 2019, 7, 80.	5.2	30
24	PMP22 Regulates Cholesterol Trafficking and ABCA1-Mediated Cholesterol Efflux. Journal of Neuroscience, 2019, 39, 5404-5418.	3.6	29
25	N-terminal sequences in matrin 3 mediate phase separation into droplet-like structures that recruit TDP43 variants lacking RNA binding elements. Laboratory Investigation, 2019, 99, 1030-1040.	3.7	30
26	ALS-Linked SOD1 Mutants Enhance Neurite Outgrowth and Branching in Adult Motor Neurons. IScience, 2019, 11, 294-304.	4.1	28
27	Experimental Mutations in Superoxide Dismutase 1 Provide Insight into Potential Mechanisms Involved in Aberrant Aggregation in Familial Amyotrophic Lateral Sclerosis. G3: Genes, Genomes, Genetics, 2019, 9, 719-728.	1.8	13
28	Aberrant accrual of BIN1 near Alzheimerâ€™s disease amyloid deposits in transgenic models. Brain Pathology, 2019, 29, 485-501.	4.1	25
29	Characterization of gene regulation and protein interaction networks for Matrin 3 encoding mutations linked to amyotrophic lateral sclerosis and myopathy. Scientific Reports, 2018, 8, 4049.	3.3	30
30	Prion-like Spreading in Tauopathies. Biological Psychiatry, 2018, 83, 337-346.	1.3	70
31	Short AÎ²2 peptides attenuate AÎ²42 toxicity in vivo. Journal of Experimental Medicine, 2018, 215, 283-301.	8.5	56
32	Analysis of spinal and muscle pathology in transgenic mice overexpressing wild-type and ALS-linked mutant MATR3. Acta Neuropathologica Communications, 2018, 6, 137.	5.2	20
33	Loss of charge mutations in solvent exposed Lys residues of superoxide dismutase 1 do not induce inclusion formation in cultured cell models. PLoS ONE, 2018, 13, e0206751.	2.5	7
34	Differential induction of mutant SOD1 misfolding and aggregation by tau and Î±-synuclein pathology. Molecular Neurodegeneration, 2018, 13, 23.	10.8	3
35	Targeting the Neuromuscular Junction in ALS. Neurotherapeutics, 2018, 15, 713-714.	4.4	0
36	Changes in proteome solubility indicate widespread proteostatic disruption in mouse models of neurodegenerative disease. Acta Neuropathologica, 2018, 136, 919-938.	7.7	27

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37	Targeting the accomplice to thwart the culprit: a new target for the prevention of amyloid deposition. Journal of Clinical Investigation, 2018, 128, 1734-1736.	8.2	3
38	Quantitative Comparison of Dense-Core Amyloid Plaque Accumulation in Amyloid-Î² Protein Precursor Transgenic Mice. Journal of Alzheimer's Disease, 2017, 56, 743-761.	2.6	39
39	Relationship between mutant Cu/Zn superoxide dismutase 1 maturation and inclusion formation in cell models. Journal of Neurochemistry, 2017, 140, 140-150.	3.9	15
40	Heterogeneity of Matrin 3 in the developing and aging murine central nervous system. Journal of Comparative Neurology, 2016, 524, 2740-2752.	1.6	14
41	Vulnerability of newly synthesized proteins to proteostasis stress. Journal of Cell Science, 2016, 129, 1892-901.	2.0	24
42	C9orf72 BAC Mouse Model with Motor Deficits and Neurodegenerative Features of ALS/FTD. Neuron, 2016, 90, 521-534.	8.1	294
43	Sex-related dimorphism in dentate gyrus atrophy and behavioral phenotypes in an inducible tTa:APPsi transgenic model of Alzheimer's disease. Neurobiology of Disease, 2016, 96, 171-185.	4.4	19
44	Distinct conformers of transmissible misfolded SOD1 distinguish human SOD1-FALS from other forms of familial and sporadic ALS. Acta Neuropathologica, 2016, 132, 827-840.	7.7	42
45	Generation of a new transgenic mouse model for assessment of tau gene silencing therapies. Alzheimer's Research and Therapy, 2016, 8, 36.	6.2	1
46	Prion-like propagation of mutant SOD1 misfolding and motor neuron disease spread along neuroanatomical pathways. Acta Neuropathologica, 2016, 131, 103-114.	7.7	117
47	Non-prion-type transmission in A53T Î±-synuclein transgenic mice: a normal component of spinal homogenates from naÃ¯ve non-transgenic mice induces robust Î±-synuclein pathology. Acta Neuropathologica, 2016, 131, 151-154.	7.7	19
48	Substantially elevating the levels of Î±Bâ€crystallin in spinal motor neurons of mutant <scp>SOD</scp>1 mice does not significantly delay paralysis or attenuate mutant protein aggregation. Journal of Neurochemistry, 2015, 133, 452-464.	3.9	19
49	Murine AÎ² over-production produces diffuse and compact Alzheimer-type amyloid deposits. Acta Neuropathologica Communications, 2015, 3, 72.	5.2	46
50	Subcellular Localization of Matrin 3 Containing Mutations Associated with ALS and Distal Myopathy. PLoS ONE, 2015, 10, e0142144.	2.5	43
51	Direct and indirect mechanisms for wild-type SOD1 to enhance the toxicity of mutant SOD1 in bigenic transgenic mice. Human Molecular Genetics, 2015, 24, 1019-1035.	2.9	15
52	Behavioral abnormalities in APPSwe/PS1dE9 mouse model of AD-like pathology: comparative analysis across multiple behavioral domains. Neurobiology of Aging, 2015, 36, 2519-2532.	3.1	72
53	Widespread and Efficient Transduction of Spinal Cord and Brain Following Neonatal AAV Injection and Potential Disease Modifying Effect in ALS Mice. Molecular Therapy, 2015, 23, 53-62.	8.2	50
54	Characterization of Protein Structural Changes in Living Cells Using Time-Lapsed FTIR Imaging. Analytical Chemistry, 2015, 87, 6025-6031.	6.5	35

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55	RAN Translation in Huntington Disease. Neuron, 2015, 88, 667-677.	8.1	275
56	Analysis of Mutant SOD1 Electrophoretic Mobility by Blue Native Gel Electrophoresis; Evidence for Soluble Multimeric Assemblies. PLoS ONE, 2014, 9, e104583.	2.5	7
57	Metal-deficient aggregates and diminished copper found in cells expressing SOD1 mutations that cause ALS. Frontiers in Aging Neuroscience, 2014, 6, 110.	3.4	52
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73	Capsid Serotype and Timing of Injection Determines AAV Transduction in the Neonatal Mice Brain. PLoS ONE, 2013, 8, e67680.	2.5	149
74	An Analysis of Interactions between Fluorescently-Tagged Mutant and Wild-Type SOD1 in Intracellular Inclusions. PLoS ONE, 2013, 8, e83981.	2.5	7
75	Thinking laterally about neurodegenerative proteinopathies. Journal of Clinical Investigation, 2013, 123, 1847-1855.	8.2	98
76	A novel variant of human superoxide dismutase 1 harboring amyotrophic lateral sclerosisâ€associated and experimental mutations in metalâ€binding residues and free cysteines lacks toxicity in vivo. Journal of Neurochemistry, 2012, 121, 475-485.	3.9	20
77	Reduction of low-density lipoprotein receptor-related protein (LRP1) in hippocampal neurons does not proportionately reduce, or otherwise alter, amyloid deposition in APPswe/PS1dE9 transgenic mice. Alzheimer's Research and Therapy, 2012, 4, 12.	6.2	16
78	A Preclinical Assessment of Neural Stem Cells as Delivery Vehicles for Anti-Amyloid Therapeutics. PLoS ONE, 2012, 7, e34097.	2.5	24
79	Abnormal SDSâ€PAGE migration of cytosolic proteins can identify domains and mechanisms that control surfactant binding. Protein Science, 2012, 21, 1197-1209.	7.6	111
80	Role of Disulfide Cross-Linking of Mutant SOD1 in the Formation of Inclusion-Body-Like Structures. PLoS ONE, 2012, 7, e47838.	2.5	23
81	Identification of Proteins Sensitive to Thermal Stress in Human Neuroblastoma and Glioma Cell Lines. PLoS ONE, 2012, 7, e49021.	2.5	27
82	Analysis of Proteolytic Processes and Enzymatic Activities in the Generation of Huntingtin N-Terminal Fragments in an HEK293 Cell Model. PLoS ONE, 2012, 7, e50750.	2.5	22
83	Cellular fusion for gene delivery to SCA1 affected Purkinje neurons. Molecular and Cellular Neurosciences, 2011, 47, 61-70.	2.2	33
84	Passive (Amyloid-Î²) Immunotherapy Attenuates Monoaminergic Axonal Degeneration in the AÎ²PPswe/PS1dE9 Mice. Journal of Alzheimer's Disease, 2011, 23, 271-279.	2.6	16
85	Superoxide dismutase 1 encoding mutations linked to ALS adopts a spectrum of misfolded states. Molecular Neurodegeneration, 2011, 6, 77.	10.8	49
86	Transgenic mice expressing caspase-6-derived N-terminal fragments of mutant huntingtin develop neurologic abnormalities with predominant cytoplasmic inclusion pathology composed largely of a smaller proteolytic derivative. Human Molecular Genetics, 2011, 20, 2770-2782.	2.9	39
87	Premature death and neurologic abnormalities in transgenic mice expressing a mutant huntingtin exon-2 fragment. Human Molecular Genetics, 2011, 20, 1633-1642.	2.9	22
88	Copper and Zinc Metallation Status of Copper-Zinc Superoxide Dismutase from Amyotrophic Lateral Sclerosis Transgenic Mice. Journal of Biological Chemistry, 2011, 286, 2795-2806.	3.4	112
89	Partial Depletion of CREB-Binding Protein Reduces Life Expectancy in a Mouse Model of Huntington Disease. Journal of Neuropathology and Experimental Neurology, 2010, 69, 396-404.	1.7	24
90	An examination of Î±Bâ€crystallin as a modifier of SOD1 aggregate pathology and toxicity in models of familial amyotrophic lateral sclerosis. Journal of Neurochemistry, 2010, 113, 1092-1100.	3.9	19

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91	Analysis of Chaperone mRNA Expression in the Adult Mouse Brain by Meta Analysis of the Allen Brain Atlas. PLoS ONE, 2010, 5, e13675.	2.5	32
92	An examination of wild-type SOD1 in modulating the toxicity and aggregation of ALS-associated mutant SOD1. Human Molecular Genetics, 2010, 19, 4774-4789.	2.9	63
93	Synphilin-1 attenuates neuronal degeneration in the A53T Â-synuclein transgenic mouse model. Human Molecular Genetics, 2010, 19, 2087-2098.	2.9	65
94	Aggregation modulating elements in mutant human superoxide dismutase 1. Archives of Biochemistry and Biophysics, 2010, 503, 175-182.	3.0	19
95	Role of mutant SOD1 disulfide oxidation and aggregation in the pathogenesis of familial ALS. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 7774-7779.	7.1	159
96	Immature Copper-Zinc Superoxide Dismutase and Familial Amyotrophic Lateral Sclerosis. Experimental Biology and Medicine, 2009, 234, 1140-1154.	2.4	78
97	Variation in aggregation propensities among ALS-associated variants of SOD1: Correlation to human disease. Human Molecular Genetics, 2009, 18, 3217-3226.	2.9	214
98	Modulation of mutant superoxide dismutase 1 aggregation by coâ€expression of wildâ€type enzyme. Journal of Neurochemistry, 2009, 108, 1009-1018.	3.9	42
99	Immunoreactivity of the phosphorylated axonal neurofilament H subunit (pNFâ€H) in blood of ALS model rodents and ALS patients: evaluation of blood pNFâ€H as a potential ALS biomarker. Journal of Neurochemistry, 2009, 111, 1182-1191.	3.9	118
100	Amyloid precursor protein increases cortical neuron size in transgenic mice. Neurobiology of Aging, 2009, 30, 1238-1244.	3.1	49
101	Differential regulation of small heat shock proteins in transgenic mouse models of neurodegenerative diseases. Neurobiology of Aging, 2008, 29, 586-597.	3.1	44
102	Detergent-insoluble Aggregates Associated with Amyotrophic Lateral Sclerosis in Transgenic Mice Contain Primarily Full-length, Unmodified Superoxide Dismutase-1. Journal of Biological Chemistry, 2008, 283, 8340-8350.	3.4	79
103	A Limited Role for Disulfide Cross-linking in the Aggregation of Mutant SOD1 Linked to Familial Amyotrophic Lateral Sclerosis. Journal of Biological Chemistry, 2008, 283, 13528-13537.	3.4	97
104	Amyloid Pathology Is Associated with Progressive Monoaminergic Neurodegeneration in a Transgenic Mouse Model of Alzheimer's Disease. Journal of Neuroscience, 2008, 28, 13805-13814.	3.6	180
105	Limited Clearance of Pre-Existing Amyloid Plaques After Intracerebral Injection of AÎ² Antibodies in Two Mouse Models of Alzheimer Disease. Journal of Neuropathology and Experimental Neurology, 2008, 67, 30-40.	1.7	20
106	Receptor-Associated Protein (RAP) Plays a Central Role in Modulating AÎ² Deposition in APP/PS1 Transgenic Mice. PLoS ONE, 2008, 3, e3159.	2.5	12
107	Messenger RNA Oxidation Occurs Early in Disease Pathogenesis and Promotes Motor Neuron Degeneration in ALS. PLoS ONE, 2008, 3, e2849.	2.5	178
108	Rodent AÎ² Modulates the Solubility and Distribution of Amyloid Deposits in Transgenic Mice. Journal of Biological Chemistry, 2007, 282, 22707-22720.	3.4	98

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109	Alzheimer's-Type Amyloidosis in Transgenic Mice Impairs Survival of Newborn Neurons Derived from Adult Hippocampal Neurogenesis. Journal of Neuroscience, 2007, 27, 6771-6780.	3.6	203
110	N-Terminal Proteolysis of Full-Length Mutant Huntingtin in an Inducible PC12 Cell Model of Huntingtonâ€™s Disease. Cell Cycle, 2007, 6, 2970-2981.	2.6	59
111	Disease-associated Mutations at Copper Ligand Histidine Residues of Superoxide Dismutase 1 Diminish the Binding of Copper and Compromise Dimer Stability. Journal of Biological Chemistry, 2007, 282, 345-352.	3.4	46
112	Characterization of Huntingtin Pathologic Fragments in Human Huntington Disease, Transgenic Mice, and Cell Models. Journal of Neuropathology and Experimental Neurology, 2007, 66, 313-320.	1.7	72
113	Biotinylated antiâ€AÎ² antibody as a tool to diagnose preâ€clinical stages of Alzheimer's Disease (AD). FASEB Journal, 2007, 21, A20.	0.5	0
114	Investigation of RNA interference to suppress expression of full-length and fragment human huntingtin. NeuroMolecular Medicine, 2007, 9, 145-155.	3.4	0
115	Amyotrophic Lateral Sclerosis â€" Are Microglia Killing Motor Neurons?. New England Journal of Medicine, 2006, 355, 1611-1613.	27.0	24
116	Mapping superoxide dismutase 1 domains of non-native interaction: roles of intra- and intermolecular disulfide bonding in aggregation. Journal of Neurochemistry, 2006, 96, 1277-1288.	3.9	76
117	Progressive phenotype and nuclear accumulation of an amino-terminal cleavage fragment in a transgenic mouse model with inducible expression of full-length mutant huntingtin. Neurobiology of Disease, 2006, 21, 381-391.	4.4	59
118	Papillomavirus-Like Particles Are an Effective Platform for Amyloid-Î² Immunization in Rabbits and Transgenic Mice. Journal of Immunology, 2006, 177, 2662-2670.	0.8	52
119	Effects of CAG repeat length, HTT protein length and protein context on cerebral metabolism measured using magnetic resonance spectroscopy in transgenic mouse models of Huntington's disease. Journal of Neurochemistry, 2005, 95, 553-562.	3.9	74
120	Selected genetically engineered models relevant to human neurodegenerative disease. , 2005, , 176-195.		1
121	Persistent Amyloidosis following Suppression of AÎ² Production in a Transgenic Model of Alzheimer Disease. PLoS Medicine, 2005, 2, e355.	8.4	202
122	Somatodendritic accumulation of misfolded SOD1-L126Z in motor neurons mediates degeneration: Î±B-crystallin modulates aggregation. Human Molecular Genetics, 2005, 14, 2335-2347.	2.9	120
123	Environmental Enrichment Mitigates Cognitive Deficits in a Mouse Model of Alzheimer's Disease. Journal of Neuroscience, 2005, 25, 5217-5224.	3.6	455
124	BACE1, a Major Determinant of Selective Vulnerability of the Brain to Amyloid-Î² Amyloidogenesis, is Essential for Cognitive, Emotional, and Synaptic Functions. Journal of Neuroscience, 2005, 25, 11693-11709.	3.6	490
125	Episodic-like memory deficits in the APPswe/PS1dE9 mouse model of Alzheimer's disease: Relationships to Î²-amyloid deposition and neurotransmitter abnormalities. Neurobiology of Disease, 2005, 18, 602-617.	4.4	362
126	Coincident thresholds of mutant protein for paralytic disease and protein aggregation caused by restrictively expressed superoxide dismutase cDNA. Neurobiology of Disease, 2005, 20, 943-952.	4.4	95

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127	Transgenic mouse models of neurodegenerative disease. , 2004, , 533-557.		0
128	Nuclear-targeting of mutant huntingtin fragments produces Huntington's disease-like phenotypes in transgenic mice. Human Molecular Genetics, 2004, 13, 1599-1610.	2.9	87
129	APP processing and amyloid deposition in mice haplo-insufficient for presenilin 1. Neurobiology of Aging, 2004, 25, 885-892.	3.1	143
130	Mutant presenilins specifically elevate the levels of the 42 residue Î²-amyloid peptide in vivo: evidence for augmentation of a 42-specific Î³ secretase. Human Molecular Genetics, 2004, 13, 159-170.	2.9	1,350
131	Identifying new therapeutics for Huntington's disease. Clinical Neuroscience Research, 2003, 3, 179-186.	0.8	1
132	APP Processing and Synaptic Function. Neuron, 2003, 37, 925-937.	8.1	1,423
133	Normal cognitive behavior in two distinct congenic lines of transgenic mice hyperexpressing mutant APPSWE. Neurobiology of Disease, 2003, 12, 194-211.	4.4	74
134	Lipopolysaccharide-induced-neuroinflammation increases intracellular accumulation of amyloid precursor protein and amyloid Î² peptide in APPswe transgenic mice. Neurobiology of Disease, 2003, 14, 133-145.	4.4	374
135	Copper-binding-site-null SOD1 causes ALS in transgenic mice: aggregates of non-native SOD1 delineate a common feature. Human Molecular Genetics, 2003, 12, 2753-2764.	2.9	279
136	Environmental Enrichment Exacerbates Amyloid Plaque Formation in a Transgenic Mouse Model of Alzheimer Disease. Journal of Neuropathology and Experimental Neurology, 2003, 62, 1220-1227.	1.7	190
137	Early phenotypes that presage late-onset neurodegenerative disease allow testing of modifiers in Hdh CAG knock-in mice. Human Molecular Genetics, 2002, 11, 633-640.	2.9	162
138	Polyglutamine and transcription: gene expression changes shared by DRPLA and Huntington's disease mouse models reveal context-independent effects. Human Molecular Genetics, 2002, 11, 1927-1937.	2.9	185
139	Genetically engineered models of neurodegenerative diseases. , 2002, , 1841-1862.		1
140	Rapid Detection of Protein Aggregates in the Brains of Alzheimer Patients and Transgenic Mouse Models of Amyloidosis. Alzheimer Disease and Associated Disorders, 2002, 16, 191-195.	1.3	34
141	AÎ² Deposition Does Not Cause the Aggregation of Endogenous Tau in Transgenic Mice. Alzheimer Disease and Associated Disorders, 2002, 16, 196-201.	1.3	18
142	High Molecular Weight Complexes of Mutant Superoxide Dismutase 1: Age-Dependent and Tissue-Specific Accumulation. Neurobiology of Disease, 2002, 9, 139-148.	4.4	189
143	Fibrillar Inclusions and Motor Neuron Degeneration in Transgenic Mice Expressing Superoxide Dismutase 1 with a Disrupted Copper-Binding Site. Neurobiology of Disease, 2002, 10, 128-138.	4.4	223
144	Accumulation of proteolytic fragments of mutant presenilin 1 and accelerated amyloid deposition are co-regulated in transgenic mice. Neurobiology of Aging, 2002, 23, 171-177.	3.1	18

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145	Cyclooxygenase (COX)-2 and cell cycle activity in a transgenic mouse model of Alzheimer's Disease neuropathology. Neurobiology of Aging, 2002, 23, 327-334.	3.1	107
146	AÎ² deposition is essential to AD neuropathology. Journal of Alzheimer's Disease, 2002, 4, 133-138.	2.6	6
147	Transgenic mouse models of neurodegenerative disease: Opportunities for therapeutic development. Current Neurology and Neuroscience Reports, 2002, 2, 457-464.	4.2	54
148	Genetically engineered mouse models of neurodegenerative diseases. Nature Neuroscience, 2002, 5, 633-639.	14.8	219
149	Distinct Behavioral and Neuropathological Abnormalities in Transgenic Mouse Models of HD and DRPLA. Neurobiology of Disease, 2001, 8, 405-418.	4.4	47
150	Creatine Increases Survival and Delays Motor Symptoms in a Transgenic Animal Model of Huntington's Disease. Neurobiology of Disease, 2001, 8, 479-491.	4.4	270
151	Coenzyme Q10 and remacemide hydrochloride ameliorate motor deficits in a Huntington's disease transgenic mouse model. Neuroscience Letters, 2001, 315, 149-153.	2.1	154
152	Co-expression of multiple transgenes in mouse CNS: a comparison of strategies. New Biotechnology, 2001, 17, 157-165.	2.7	712
153	Dichloroacetate exerts therapeutic effects in transgenic mouse models of Huntington's disease. Annals of Neurology, 2001, 50, 112-116.	5.3	79
154	Genetically Engineered Models Relevant to Neurodegenerative Disorders: Their Value for Understanding Disease Mechanisms and Designing/Testing Experimental Therapeutics. Journal of Molecular Neuroscience, 2001, 17, 233-257.	2.3	14
155	BACE1 is the major Î²-secretase for generation of AÎ² peptides by neurons. Nature Neuroscience, 2001, 4, 233-234.	14.8	1,023
156	Î²-Amyloid Peptide Vaccination Results in Marked Changes in Serum and Brain AÎ² Levels in APPswe/PS1Î”E9 Mice, as Detected by SELDI-TOF-Based ProteinChip^{Â®} Technology. DNA and Cell Biology, 2001, 20, 713-721.	1.9	46
157	Decreased expression of striatal signaling genes in a mouse model of Huntington's disease. Human Molecular Genetics, 2000, 9, 1259-1271.	2.9	645
158	Enhanced Synaptic Potentiation in Transgenic Mice Expressing presenilin 1 Familial Alzheimer's Disease Mutation Is Normalized with a Benzodiazepine. Neurobiology of Disease, 2000, 7, 54-63.	4.4	62
159	Amyloid Precursor Proteins Inhibit Heme Oxygenase Activity and Augment Neurotoxicity in Alzheimer's Disease. Neuron, 2000, 28, 461-473.	8.1	168
160	The Value of Transgenic Models for the Study of Neurodegenerative Diseases. Annals of the New York Academy of Sciences, 2000, 920, 179-191.	3.8	51
161	SOD1 rescues cerebral endothelial dysfunction in mice overexpressing amyloid precursor protein. Nature Neuroscience, 1999, 2, 157-161.	14.8	371
162	Nuclear Accumulation of Truncated Atrophin-1 Fragments in a Transgenic Mouse Model of DRPLA. Neuron, 1999, 24, 275-286.	8.1	173

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163	Synaptic Transmission and Hippocampal Long-Term Potentiation in Transgenic Mice Expressing FAD-Linked Presenilin 1. Neurobiology of Disease, 1999, 6, 56-62.	4.4	109
164	Alzheimer disease â€" when and why?. Nature Genetics, 1998, 19, 314-316.	21.4	36
165	Transgenic Mouse Models of Alzheimer's Disease and Amyotrophic Lateral Sclerosis. Brain Pathology, 1998, 8, 735-757.	4.1	27
166	Effects of PS1 Deficiency on Membrane Protein Trafficking in Neurons. Neuron, 1998, 21, 1213-1221.	8.1	359
167	An Alzheimer's Disease-Linked PS1 Variant Rescues the Developmental Abnormalities of PS1-Deficient Embryos. Neuron, 1998, 20, 603-609.	8.1	134
168	ALZHEIMER'S DISEASE: Genetic Studies and Transgenic Models. Annual Review of Genetics, 1998, 32, 461-493.	7.6	384
169	Stable Association of Presenilin Derivatives and Absence of Presenilin Interactions with APP. Neurobiology of Disease, 1998, 4, 438-453.	4.4	187
170	Axonal Transport of Mutant Superoxide Dismutase 1 and Focal Axonal Abnormalities in the Proximal Axons of Transgenic Mice. Neurobiology of Disease, 1998, 5, 27-35.	4.4	96
171	Genetic Neurodegenerative Diseases: The Human Illness and Transgenic Models. Science, 1998, 282, 1079-1083.	12.6	223
172	Evidence That Levels of Presenilins (PS1 and PS2) Are Coordinately Regulated by Competition for Limiting Cellular Factors. Journal of Biological Chemistry, 1997, 272, 28415-28422.	3.4	302
173	Endoproteolytic Processing and Stabilization of Wild-type and Mutant Presenilin. Journal of Biological Chemistry, 1997, 272, 24536-24541.	3.4	190
174	Accelerated Amyloid Deposition in the Brains of Transgenic Mice Coexpressing Mutant Presenilin 1 and Amyloid Precursor Proteins. Neuron, 1997, 19, 939-945.	8.1	964
175	Hyperaccumulation of FAD-linked presenilin 1 variants in vivo. Nature Medicine, 1997, 3, 756-760.	30.7	140
176	Transgenic models of neurodegenerative diseases. Current Opinion in Neurobiology, 1996, 6, 651-660.	4.2	30
177	Familial Alzheimer's Diseaseâ€"Linked Presenilin 1 Variants Elevate Aî²1â€"42/1â€"40 Ratio In Vitro and In Vivo. Neuron, 1996, 17, 1005-1013.	8.1	1,471
178	Protein Topology of Presenilin 1. Neuron, 1996, 17, 1023-1030.	8.1	381
179	Endoproteolysis of Presenilin 1 and Accumulation of Processed Derivatives In Vivo. Neuron, 1996, 17, 181-190.	8.1	1,054
180	Transgenic and gene-targeting approaches to model disorders of motor neurons. Seminars in Neuroscience, 1996, 8, 163-169.	2.2	2

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181	Inherited Neurodegenerative Diseases and Transgenic Models. Brain Pathology, 1996, 6, 467-480.	4.1	9
182	A vector for expressing foreign genes in the brains and hearts of transgenic mice. Genetic Analysis, Techniques and Applications, 1996, 13, 159-163.	1.5	323
183	Loss of functional prion protein: a role in prion disorders?. Chemistry and Biology, 1996, 3, 619-621.	6.0	14
184	Motor Neuron Disease and Model Systems: Aetiologies, Mechanisms and Therapies. Novartis Foundation Symposium, 1996, 196, 3-17.	1.1	1
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