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	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
2	APP Processing and Synaptic Function. Neuron, 2003, 37, 925-937.	8.1	1,423
3	An adverse property of a familial ALS-linked SOD1 mutation causes motor neuron disease characterized by vacuolar degeneration of mitochondria. Neuron, 1995, 14, 1105-1116.	8.1	1,394
4	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
5	Endoproteolysis of Presenilin 1 and Accumulation of Processed Derivatives In Vivo. Neuron, 1996, 17, 181-190.	8.1	1,054
6	BACE1 is the major Î²-secretase for generation of AÎ² peptides by neurons. Nature Neuroscience, 2001, 4, 233-234.	14.8	1,023
7	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
8	Co-expression of multiple transgenes in mouse CNS: a comparison of strategies. New Biotechnology, 2001, 17, 157-165.	2.7	712
9	Decreased expression of striatal signaling genes in a mouse model of Huntington's disease. Human Molecular Genetics, 2000, 9, 1259-1271.	2.9	645
10	Age-related CNS disorder and early death in transgenic FVB/N mice overexpressing Alzheimer amyloid precursor proteins. Neuron, 1995, 15, 1203-1218.	8.1	520
11	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
12	Environmental Enrichment Mitigates Cognitive Deficits in a Mouse Model of Alzheimer's Disease. Journal of Neuroscience, 2005, 25, 5217-5224.	3.6	455
13	ALZHEIMER'S DISEASE: Genetic Studies and Transgenic Models. Annual Review of Genetics, 1998, 32, 461-493.	7.6	384
14	Protein Topology of Presenilin 1. Neuron, 1996, 17, 1023-1030.	8.1	381
15	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
16	SOD1 rescues cerebral endothelial dysfunction in mice overexpressing amyloid precursor protein. Nature Neuroscience, 1999, 2, 157-161.	14.8	371
17	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
18	Effects of PS1 Deficiency on Membrane Protein Trafficking in Neurons. Neuron, 1998, 21, 1213-1221.	8.1	359

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19	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
20	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
21	C9orf72 BAC Mouse Model with Motor Deficits and Neurodegenerative Features of ALS/FTD. Neuron, 2016, 90, 521-534.	8.1	294
22	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
23	Intramuscular injection of Î±-synuclein induces CNS Î±-synuclein pathology and a rapid-onset motor phenotype in transgenic mice. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, 10732-10737.	7.1	277
24	RAN Translation in Huntington Disease. Neuron, 2015, 88, 667-677.	8.1	275
25	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
26	Asparagine-linked glycosylation of the scrapie and cellular prion proteins. Archives of Biochemistry and Biophysics, 1989, 274, 1-13.	3.0	246
27	Genetic Neurodegenerative Diseases: The Human Illness and Transgenic Models. Science, 1998, 282, 1079-1083.	12.6	223
28	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
29	Genetically engineered mouse models of neurodegenerative diseases. Nature Neuroscience, 2002, 5, 633-639.	14.8	219
30	Variation in aggregation propensities among ALS-associated variants of SOD1: Correlation to human disease. Human Molecular Genetics, 2009, 18, 3217-3226.	2.9	214
31	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
32	Persistent Amyloidosis following Suppression of AÎ² Production in a Transgenic Model of Alzheimer Disease. PLoS Medicine, 2005, 2, e355.	8.4	202
33	Endoproteolytic Processing and Stabilization of Wild-type and Mutant Presenilin. Journal of Biological Chemistry, 1997, 272, 24536-24541.	3.4	190
34	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
35	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
36	Stable Association of Presenilin Derivatives and Absence of Presenilin Interactions with APP. Neurobiology of Disease, 1998, 4, 438-453.	4.4	187

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37	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
38	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
39	Messenger RNA Oxidation Occurs Early in Disease Pathogenesis and Promotes Motor Neuron Degeneration in ALS. PLoS ONE, 2008, 3, e2849.	2.5	178
40	Nuclear Accumulation of Truncated Atrophin-1 Fragments in a Transgenic Mouse Model of DRPLA. Neuron, 1999, 24, 275-286.	8.1	173
41	Amyloid Precursor Proteins Inhibit Heme Oxygenase Activity and Augment Neurotoxicity in Alzheimer's Disease. Neuron, 2000, 28, 461-473.	8.1	168
42	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
43	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
44	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
45	Capsid Serotype and Timing of Injection Determines AAV Transduction in the Neonatal Mice Brain. PLoS ONE, 2013, 8, e67680.	2.5	149
46	APP processing and amyloid deposition in mice haplo-insufficient for presenilin 1. Neurobiology of Aging, 2004, 25, 885-892.	3.1	143
47	Superoxide Dismutase 1 Subunits with Mutations Linked to Familial Amyotrophic Lateral Sclerosis Do Not Affect Wild-type Subunit Function. Journal of Biological Chemistry, 1995, 270, 3234-3238.	3.4	142
48	Hyperaccumulation of FAD-linked presenilin 1 variants in vivo. Nature Medicine, 1997, 3, 756-760.	30.7	140
49	An Alzheimer's Disease-Linked PS1 Variant Rescues the Developmental Abnormalities of PS1-Deficient Embryos. Neuron, 1998, 20, 603-609.	8.1	134
50	Release of the cellular prion protein from cultured cells after loss of its glycoinositol phospholipid anchor. Glycobiology, 1993, 3, 319-329.	2.5	129
51	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
52	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
53	Prion-like propagation of mutant SOD1 misfolding and motor neuron disease spread along neuroanatomical pathways. Acta Neuropathologica, 2016, 131, 103-114.	7.7	117
54	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

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55	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
56	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
57	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
58	Experimental transmissibility of mutant SOD1 motor neuron disease. Acta Neuropathologica, 2014, 128, 791-803.	7.7	100
59	Rodent AÎ² Modulates the Solubility and Distribution of Amyloid Deposits in Transgenic Mice. Journal of Biological Chemistry, 2007, 282, 22707-22720.	3.4	98
60	Thinking laterally about neurodegenerative proteinopathies. Journal of Clinical Investigation, 2013, 123, 1847-1855.	8.2	98
61	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
62	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
63	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
64	Therapeutic approaches targeting Apolipoprotein E function in Alzheimerâ€™s disease. Molecular Neurodegeneration, 2020, 15, 8.	10.8	89
65	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
66	Dichloroacetate exerts therapeutic effects in transgenic mouse models of Huntington's disease. Annals of Neurology, 2001, 50, 112-116.	5.3	79
67	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
68	Immature Copper-Zinc Superoxide Dismutase and Familial Amyotrophic Lateral Sclerosis. Experimental Biology and Medicine, 2009, 234, 1140-1154.	2.4	78
69	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
70	Normal cognitive behavior in two distinct congenic lines of transgenic mice hyperexpressing mutant APPSWE. Neurobiology of Disease, 2003, 12, 194-211.	4.4	74
71	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
72	Normal cognition in transgenic BRI2-AÎ² mice. Molecular Neurodegeneration, 2013, 8, 15.	10.8	74

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73	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
74	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
75	Prion-like Spreading in Tauopathies. Biological Psychiatry, 2018, 83, 337-346.	1.3	70
76	Synphilin-1 attenuates neuronal degeneration in the A53T Â-synuclein transgenic mouse model. Human Molecular Genetics, 2010, 19, 2087-2098.	2.9	65
77	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
78	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
79	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
80	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
81	Short AÎ² peptides attenuate AÎ²42 toxicity in vivo. Journal of Experimental Medicine, 2018, 215, 283-301.	8.5	56
82	Transgenic mouse models of neurodegenerative disease: Opportunities for therapeutic development. Current Neurology and Neuroscience Reports, 2002, 2, 457-464.	4.2	54
83	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
84	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
85	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
86	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
87	Amyloid precursor protein increases cortical neuron size in transgenic mice. Neurobiology of Aging, 2009, 30, 1238-1244.	3.1	49
88	Superoxide dismutase 1 encoding mutations linked to ALS adopts a spectrum of misfolded states. Molecular Neurodegeneration, 2011, 6, 77.	10.8	49
89	Distinct Behavioral and Neuropathological Abnormalities in Transgenic Mouse Models of HD and DRPLA. Neurobiology of Disease, 2001, 8, 405-418.	4.4	47
90	Structural similarity of wild-type and ALS-mutant superoxide dismutase-1 fibrils using limited proteolysis and atomic force microscopy. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 10934-10939.	7.1	47

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91	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
92	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
93	Reversible Pathologic and Cognitive Phenotypes in an Inducible Model of Alzheimer-Amyloidosis. Journal of Neuroscience, 2013, 33, 3765-3779.	3.6	46
94	Murine AÎ² over-production produces diffuse and compact Alzheimer-type amyloid deposits. Acta Neuropathologica Communications, 2015, 3, 72.	5.2	46
95	Differential regulation of small heat shock proteins in transgenic mouse models of neurodegenerative diseases. Neurobiology of Aging, 2008, 29, 586-597.	3.1	44
96	Cytosolic proteins lose solubility as amyloid deposits in a transgenic mouse model of Alzheimer-type amyloidosis. Human Molecular Genetics, 2013, 22, 2765-2774.	2.9	43
97	Subcellular Localization of Matrin 3 Containing Mutations Associated with ALS and Distal Myopathy. PLoS ONE, 2015, 10, e0142144.	2.5	43
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	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
100	Conformational specificity of the C4F6 SOD1 antibody; low frequency of reactivity in sporadic ALS cases. Acta Neuropathologica Communications, 2014, 2, 55.	5.2	41
101	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
102	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
103	Unbiased screen reveals ubiquilin-1 and -2 highly associated with huntingtin inclusions. Brain Research, 2013, 1524, 62-73.	2.2	38
104	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
105	Alzheimer disease â€” when and why?. Nature Genetics, 1998, 19, 314-316.	21.4	36
106	Characterization of Protein Structural Changes in Living Cells Using Time-Lapsed FTIR Imaging. Analytical Chemistry, 2015, 87, 6025-6031.	6.5	35
107	Blood-based biomarkers of inflammation in amyotrophic lateral sclerosis. Molecular Neurodegeneration, 2022, 17, 11.	10.8	35
108	Motor neuron disease caused by mutations in superoxide dismutase 1. Current Opinion in Neurology, 1995, 8, 294-302.	3.6	34

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109	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
110	Regenerative medicine in Alzheimer's disease. Translational Research, 2014, 163, 432-438.	5.0	34
111	Cellular fusion for gene delivery to SCA1 affected Purkinje neurons. Molecular and Cellular Neurosciences, 2011, 47, 61-70.	2.2	33
112	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
113	Transgenic models of neurodegenerative diseases. Current Opinion in Neurobiology, 1996, 6, 651-660.	4.2	30
114	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
115	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
116	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
117	PMP22 Regulates Cholesterol Trafficking and ABCA1-Mediated Cholesterol Efflux. Journal of Neuroscience, 2019, 39, 5404-5418.	3.6	29
118	ALS-Linked SOD1 Mutants Enhance Neurite Outgrowth and Branching in Adult Motor Neurons. IScience, 2019, 11, 294-304.	4.1	28
119	Transgenic Mouse Models of Alzheimer's Disease and Amyotrophic Lateral Sclerosis. Brain Pathology, 1998, 8, 735-757.	4.1	27
120	Changes in proteome solubility indicate widespread proteostatic disruption in mouse models of neurodegenerative disease. Acta Neuropathologica, 2018, 136, 919-938.	7.7	27
121	Identification of Proteins Sensitive to Thermal Stress in Human Neuroblastoma and Glioma Cell Lines. PLoS ONE, 2012, 7, e49021.	2.5	27
122	Aberrant accrual of BIN1 near Alzheimerâ€™s disease amyloid deposits in transgenic models. Brain Pathology, 2019, 29, 485-501.	4.1	25
123	Amyotrophic Lateral Sclerosis â€" Are Microglia Killing Motor Neurons?. New England Journal of Medicine, 2006, 355, 1611-1613.	27.0	24
124	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
125	A Preclinical Assessment of Neural Stem Cells as Delivery Vehicles for Anti-Amyloid Therapeutics. PLoS ONE, 2012, 7, e34097.	2.5	24
126	Robust cytoplasmic accumulation of phosphorylated TDP-43 in transgenic models of tauopathy. Acta Neuropathologica, 2013, 126, 39-50.	7.7	24

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127	Vulnerability of newly synthesized proteins to proteostasis stress. Journal of Cell Science, 2016, 129, 1892-901.	2.0	24
128	Role of Disulfide Cross-Linking of Mutant SOD1 in the Formation of Inclusion-Body-Like Structures. PLoS ONE, 2012, 7, e47838.	2.5	23
129	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
130	Tryptophan residue 32 in human Cu-Zn superoxide dismutase modulates prion-like propagation and strain selection. PLoS ONE, 2020, 15, e0227655.	2.5	22
131	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
132	Distinctive features of the D101N and D101G variants of superoxide dismutase 1; two mutations that produce rapidly progressing motor neuron disease. Journal of Neurochemistry, 2014, 128, 305-314.	3.9	21
133	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
134	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
135	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
136	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
137	Aggregation modulating elements in mutant human superoxide dismutase 1. Archives of Biochemistry and Biophysics, 2010, 503, 175-182.	3.0	19
138	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
139	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
140	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
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	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
143	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
144	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

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145	Diversity in AÎ² deposit morphology and secondary proteome insolubility across models of Alzheimer-typeÂamyloidosis. Acta Neuropathologica Communications, 2020, 8, 43.	5.2	16
146	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
147	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
148	Loss of functional prion protein: a role in prion disorders?. Chemistry and Biology, 1996, 3, 619-621.	6.0	14
149	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
150	Heterogeneity of Matrin 3 in the developing and aging murine central nervous system. Journal of Comparative Neurology, 2016, 524, 2740-2752.	1.6	14
151	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
152	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
153	Features of wild-type human SOD1 limit interactions with misfolded aggregates of mouse G86R Sod1. Molecular Neurodegeneration, 2013, 8, 46.	10.8	12
154	Collusion of Î±-Synuclein and AÎ² aggravating co-morbidities in a novel prion-type mouse model. Molecular Neurodegeneration, 2021, 16, 63.	10.8	12
155	Subcellular diversion of cholesterol by gainâ€•and lossâ€•ofâ€function mutations in <scp>PMP22</scp>. Glia, 2020, 68, 2300-2315.	4.9	11
156	Differences in memory development among C57BL/6NCrl, 129S2/SvPasCrl, and FVB/NCrl mice after delay and trace fear conditioning. Comparative Medicine, 2014, 64, 4-12.	1.0	11
157	Inherited Neurodegenerative Diseases and Transgenic Models. Brain Pathology, 1996, 6, 467-480.	4.1	9
158	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
159	Phenotypic diversity in ALS and the role of poly-conformational protein misfolding. Acta Neuropathologica, 2020, 142, 41-55.	7.7	9
160	An Analysis of Interactions between Fluorescently-Tagged Mutant and Wild-Type SOD1 in Intracellular Inclusions. PLoS ONE, 2013, 8, e83981.	2.5	7
161	Analysis of Mutant SOD1 Electrophoretic Mobility by Blue Native Gel Electrophoresis; Evidence for Soluble Multimeric Assemblies. PLoS ONE, 2014, 9, e104583.	2.5	7
162	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

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163	Remodeling Alzheimer-amyloidosis models by seeding. Molecular Neurodegeneration, 2021, 16, 8.	10.8	7
164	Influence of base-pairing in the leader region on in vitro translation of Rous sarcoma virus RNA. Virus Research, 1985, 3, 141-151.	2.2	6
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#	Article	IF	Citations
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