Don W Cleveland

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/3321894/publications.pdf

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195 papers 51,641 citations

114 h-index

997

196 g-index

204 all docs

204 docs citations

times ranked

204

35305 citing authors

#	Article	IF	CITATIONS
1	Antisense Drugs Make Sense for Neurological Diseases. Annual Review of Pharmacology and Toxicology, 2021, 61, 831-852.	9.4	54
2	HSP70 chaperones RNA-free TDP-43 into anisotropic intranuclear liquid spherical shells. Science, 2021, 371, .	12.6	200
3	Chromothripsis drives the evolution of gene amplification in cancer. Nature, 2021, 591, 137-141.	27.8	228
4	The SARS-CoV-2 nucleocapsid phosphoprotein forms mutually exclusive condensates with RNA and the membrane-associated M protein. Nature Communications, 2021, 12, 502.	12.8	307
5	Novel STMN2 Variant Linked to Amyotrophic Lateral Sclerosis Risk and Clinical Phenotype. Frontiers in Aging Neuroscience, 2021, 13, 658226.	3.4	38
6	Therapeutically viable generation of neurons with antisense oligonucleotide suppression of PTB. Nature Neuroscience, 2021, 24, 1089-1099.	14.8	40
7	Causes and consequences of micronuclei. Current Opinion in Cell Biology, 2021, 70, 91-99.	5.4	102
8	Transient genomic instability drives tumorigenesis through accelerated clonal evolution. Genes and Development, 2021, 35, 1093-1108.	5.9	48
9	Wild-type FUS corrects ALS-like disease induced by cytoplasmic mutant FUS through autoregulation. Molecular Neurodegeneration, 2021, 16, 61.	10.8	9
10	Gene expression regulated by RNA stability. Science, 2020, 367, 29-29.	12.6	7
11	Spinal subpial delivery of AAV9 enables widespread gene silencing and blocks motoneuron degeneration in ALS. Nature Medicine, 2020, 26, 118-130.	30.7	80
12	Reversing a model of Parkinson's disease with in situ converted nigral neurons. Nature, 2020, 582, 550-556.	27.8	316
13	Reduced C9ORF72 function exacerbates gain of toxicity from ALS/FTD-causing repeat expansion in C9orf72. Nature Neuroscience, 2020, 23, 615-624.	14.8	157
14	Antisense Oligonucleotide Therapies for Neurodegenerative Diseases. Annual Review of Neuroscience, 2019, 42, 385-406.	10.7	214
15	CRISPR-Cas9 Screens Identify the RNA Helicase DDX3X as a Repressor of C9ORF72 (GGGGCC)n Repeat-Associated Non-AUG Translation. Neuron, 2019, 104, 885-898.e8.	8.1	107
16	DNA replication acts as an error correction mechanism to maintain centromere identity by restricting CENP-A to centromeres. Nature Cell Biology, 2019, 21, 743-754.	10.3	65
17	BubR1 phosphorylates CENP-E as a switch enabling the transition from lateral association to end-on capture of spindle microtubules. Cell Research, 2019, 29, 562-578.	12.0	46
18	Cytoplasmic TDP-43 De-mixing Independent of Stress Granules Drives Inhibition of Nuclear Import, Loss of Nuclear TDP-43, and Cell Death. Neuron, 2019, 102, 339-357.e7.	8.1	331

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19	Chromosome segregation errors generate a diverse spectrum of simple and complex genomic rearrangements. Nature Genetics, 2019, 51, 705-715.	21.4	145
20	Phosphorylation of CENP-A on serine 7 does not control centromere function. Nature Communications, 2019, 10, 175.	12.8	17
21	Premature polyadenylation-mediated loss of stathmin-2 is a hallmark of TDP-43-dependent neurodegeneration. Nature Neuroscience, 2019, 22, 180-190.	14.8	345
22	Membralin deficiency dysregulates astrocytic glutamate homeostasis, leading to ALS-like impairment. Journal of Clinical Investigation, 2019, 129, 3103-3120.	8.2	27
23	Overriding FUS autoregulation in mice triggers gain-of-toxic dysfunctions in RNA metabolism and autophagy-lysosome axis. ELife, 2019, 8, .	6.0	65
24	Chromosomal instability drives metastasis through a cytosolic DNA response. Nature, 2018, 553, 467-472.	27.8	1,002
25	C9ORF72 GGGGCC repeat-associated non-AUG translation is upregulated by stress through eIF2α phosphorylation. Nature Communications, 2018, 9, 51.	12.8	166
26	Chemically Modified Cpf1-CRISPR RNAs Mediate Efficient Genome Editing in Mammalian Cells. Molecular Therapy, 2018, 26, 1228-1240.	8.2	60
27	ALS/FTD-Linked Mutation in FUS Suppresses Intra-axonal Protein Synthesis and Drives Disease Without Nuclear Loss-of-Function of FUS. Neuron, 2018, 100, 816-830.e7.	8.1	185
28	TRIP13 and APC15 drive mitotic exit by turnover of interphase- and unattached kinetochore-produced MCC. Nature Communications, 2018, 9, 4354.	12.8	39
29	Tuning Apoptosis and Neuroinflammation: TBK1 Restrains RIPK1. Cell, 2018, 174, 1339-1341.	28.9	11
30	Biological Spectrum of Amyotrophic Lateral Sclerosis Prions. Cold Spring Harbor Perspectives in Medicine, 2017, 7, a024133.	6.2	24
31	CENP-A Modifications on Ser68 and Lys124 Are Dispensable for Establishment, Maintenance, and Long-Term Function of Human Centromeres. Developmental Cell, 2017, 40, 104-113.	7.0	45
32	Centrosome Amplification Is Sufficient to Promote Spontaneous Tumorigenesis in Mammals. Developmental Cell, 2017, 40, 313-322.e5.	7.0	291
33	Human centromeric CENP-A chromatin is a homotypic, octameric nucleosome at all cell cycle points. Journal of Cell Biology, 2017, 216, 607-621.	5.2	53
34	Misfolded SOD1 is not a primary component of sporadic ALS. Acta Neuropathologica, 2017, 134, 97-111.	7.7	74
35	Centromeres are maintained by fastening CENP-A to DNA and directing an arginine anchor-dependent nucleosome transition. Nature Communications, 2017, 8, 15775.	12.8	75
36	Polyglutamine-Expanded Huntingtin Exacerbates Age-Related Disruption of Nuclear Integrity and Nucleocytoplasmic Transport. Neuron, 2017, 94, 48-57.e4.	8.1	190

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37	Mutant TDP-43 within motor neurons drives disease onset but not progression in amyotrophic lateral sclerosis. Acta Neuropathologica, 2017, 133, 907-922.	7.7	61
38	Selective Y centromere inactivation triggers chromosome shattering in micronuclei and repair by non-homologous end joining. Nature Cell Biology, 2017, 19, 68-75.	10.3	207
39	Gene-editing therapy for neurological disease. Nature Reviews Neurology, 2017, 13, 7-9.	10.1	26
40	Rebuilding Chromosomes After Catastrophe: Emerging Mechanisms of Chromothripsis. Trends in Cell Biology, 2017, 27, 917-930.	7.9	162
41	Rethinking Unconventional Translation in Neurodegeneration. Cell, 2017, 171, 994-1000.	28.9	56
42	Interrogating cell division errors using random and chromosome-specific missegregation approaches. Cell Cycle, 2017, 16, 1252-1258.	2.6	11
43	The <scp>AAA</scp> + <scp>ATP</scp> ase <scp>TRIP</scp> 13 remodels <scp>HORMA</scp> domains through Nâ€terminal engagement and unfolding. EMBO Journal, 2017, 36, 2419-2434.	7.8	69
44	Bidirectional Transcriptional Inhibition as Therapy for ALS/FTD Caused by Repeat Expansion in C9orf72. Neuron, 2016, 92, 1160-1163.	8.1	18
45	Gain of Toxicity from ALS/FTD-Linked Repeat Expansions in C9ORF72 Is Alleviated by Antisense Oligonucleotides Targeting GGGGCC-Containing RNAs. Neuron, 2016, 90, 535-550.	8.1	437
46	CENP-A Is Dispensable for Mitotic Centromere Function after Initial Centromere/Kinetochore Assembly. Cell Reports, 2016, 17, 2394-2404.	6.4	89
47	Decoding ALS: from genes to mechanism. Nature, 2016, 539, 197-206.	27.8	1,533
48	C9ORF72 poly(GA) aggregates sequester and impair HR23 and nucleocytoplasmic transport proteins. Nature Neuroscience, 2016, 19, 668-677.	14.8	268
49	Deletion or Inhibition of the Oxygen Sensor PHD1 Protects against Ischemic Stroke via Reprogramming of Neuronal Metabolism. Cell Metabolism, 2016, 23, 280-291.	16.2	77
50	Disrupted nuclear import-export in neurodegeneration. Science, 2016, 351, 125-126.	12.6	16
51	Epidermal development, growth control, and homeostasis in the face of centrosome amplification. Proceedings of the National Academy of Sciences of the United States of America, 2015, 112, E6311-20.	7.1	46
52	ALS-causative mutations in FUS/TLS confer gain and loss of function by altered association with SMN and U1-snRNP. Nature Communications, 2015, 6, 6171.	12.8	205
53	Macrophage Migration Inhibitory Factor as a Chaperone Inhibiting Accumulation of Misfolded SOD1. Neuron, 2015, 86, 218-232.	8.1	98
54	DNA Sequence-Specific Binding of CENP-B Enhances the Fidelity of Human Centromere Function. Developmental Cell, 2015, 33, 314-327.	7.0	207

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55	Chronic centrosome amplification without tumorigenesis. Proceedings of the National Academy of Sciences of the United States of America, 2015, 112, E6321-30.	7.1	70
56	Wild type human TDP-43 potentiates ALS-linked mutant TDP-43 driven progressive motor and cortical neuron degeneration with pathological features of ALS. Acta Neuropathologica Communications, 2015, 3, 36.	5.2	73
57	Novel clinical associations with specific C9ORF72 transcripts in patients with repeat expansions in C9ORF72. Acta Neuropathologica, 2015, 130, 863-876.	7.7	104
58	Cerebellar c9RAN proteins associate with clinical and neuropathological characteristics of C9ORF72 repeat expansion carriers. Acta Neuropathologica, 2015, 130, 559-573.	7.7	89
59	Translational profiling identifies a cascade of damage initiated in motor neurons and spreading to glia in mutant SOD1-mediated ALS. Proceedings of the National Academy of Sciences of the United States of America, 2015, 112, E6993-7002.	7.1	165
60	Synthetic CRISPR RNA-Cas9–guided genome editing in human cells. Proceedings of the National Academy of Sciences of the United States of America, 2015, 112, E7110-7.	7.1	151
61	The quantitative architecture of centromeric chromatin. ELife, 2014, 3, e02137.	6.0	179
62	Direct conversion of patient fibroblasts demonstrates non-cell autonomous toxicity of astrocytes to motor neurons in familial and sporadic ALS. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, 829-832.	7.1	296
63	Kinetochore–microtubule attachment throughout mitosis potentiated by the elongated stalk of the kinetochore kinesin CENP-E. Molecular Biology of the Cell, 2014, 25, 2272-2281.	2.1	40
64	Polo-like Kinase 4 Inhibition: A Strategy for Cancer Therapy?. Cancer Cell, 2014, 26, 151-153.	16.8	40
65	Mutations in CENPE define a novel kinetochore-centromeric mechanism for microcephalic primordial dwarfism. Human Genetics, 2014, 133, 1023-1039.	3.8	82
66	Catalytic Assembly of the Mitotic Checkpoint Inhibitor BubR1-Cdc20 by a Mad2-Induced Functional Switch in Cdc20. Molecular Cell, 2013, 51, 92-104.	9.7	88
67	Converging Mechanisms in ALS and FTD: Disrupted RNA and Protein Homeostasis. Neuron, 2013, 79, 416-438.	8.1	1,401
68	Kinetochore kinesin CENP-E is a processive bi-directional tracker of dynamic microtubule tips. Nature Cell Biology, 2013, 15, 1079-1088.	10.3	122
69	A two-step mechanism for epigenetic specification of centromere identity and function. Nature Cell Biology, 2013, 15, 1056-1066.	10.3	226
70	Therapeutic AAV9-mediated Suppression of Mutant SOD1 Slows Disease Progression and Extends Survival in Models of Inherited ALS. Molecular Therapy, 2013, 21, 2148-2159.	8.2	178
71	Enhancing Mitochondrial Calcium Buffering Capacity Reduces Aggregation of Misfolded SOD1 and Motor Neuron Cell Death without Extending Survival in Mouse Models of Inherited Amyotrophic Lateral Sclerosis. Journal of Neuroscience, 2013, 33, 4657-4671.	3.6	161
72	ALS-linked TDP-43 mutations produce aberrant RNA splicing and adult-onset motor neuron disease without aggregation or loss of nuclear TDP-43. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, E736-45.	7.1	370

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73	Degeneration and impaired regeneration of gray matter oligodendrocytes in amyotrophic lateral sclerosis. Nature Neuroscience, 2013, 16, 571-579.	14.8	485
74	Targeted degradation of sense and antisense <i>C9orf72</i> RNA foci as therapy for ALS and frontotemporal degeneration. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, E4530-9.	7.1	508
75	C1q induction and global complement pathway activation do not contribute to ALS toxicity in mutant SOD1 mice. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, E4385-92.	7.1	60
76	Chromosome missegregation rate predicts whether aneuploidy will promote or suppress tumors. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, E4134-41.	7.1	207
77	Polo-like kinase 4 controls centriole duplication but does not directly regulate cytokinesis. Molecular Biology of the Cell, 2012, 23, 1838-1845.	2.1	35
78	The autoregulated instability of Polo-like kinase 4 limits centrosome duplication to once per cell cycle. Genes and Development, 2012, 26, 2684-2689.	5.9	132
79	Divergent roles of ALS-linked proteins FUS/TLS and TDP-43 intersect in processing long pre-mRNAs. Nature Neuroscience, 2012, 15, 1488-1497.	14.8	628
80	Centrosomes, chromosome instability (CIN) and aneuploidy. Current Opinion in Cell Biology, 2012, 24, 809-815.	5.4	103
81	Inducible, reversible system for the rapid and complete degradation of proteins in mammalian cells. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, E3350-7.	7.1	277
82	Misregulated RNA processing in amyotrophic lateral sclerosis. Brain Research, 2012, 1462, 3-15.	2.2	150
83	Single-Stranded RNAs Use RNAi to Potently and Allele-Selectively Inhibit Mutant Huntingtin Expression. Cell, 2012, 150, 895-908.	28.9	250
84	Sustained Therapeutic Reversal of Huntington's Disease by Transient Repression of Huntingtin Synthesis. Neuron, 2012, 74, 1031-1044.	8.1	635
85	Elevated PGC-1α Activity Sustains Mitochondrial Biogenesis and Muscle Function without Extending Survival in a Mouse Model of Inherited ALS. Cell Metabolism, 2012, 15, 778-786.	16.2	158
86	Chromoanagenesis and cancer: mechanisms and consequences of localized, complex chromosomal rearrangements. Nature Medicine, 2012, 18, 1630-1638.	30.7	231
87	Human Neural Stem Cell Replacement Therapy for Amyotrophic Lateral Sclerosis by Spinal Transplantation. PLoS ONE, 2012, 7, e42614.	2.5	95
88	Replicating centromeric chromatin: Spatial and temporal control of CENP-A assembly. Experimental Cell Research, 2012, 318, 1353-1360.	2.6	28
89	Epigenetic Centromere Propagation and the Nature of CENP-A Nucleosomes. Cell, 2011, 144, 471-479.	28.9	311
90	Long pre-mRNA depletion and RNA missplicing contribute to neuronal vulnerability from loss of TDP-43. Nature Neuroscience, 2011, 14, 459-468.	14.8	1,050

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91	Understanding the role of TDP-43 and FUS/TLS in ALS and beyond. Current Opinion in Neurobiology, 2011, 21, 904-919.	4.2	308
92	Misfolded SOD1 Associated with Motor Neuron Mitochondria Alters Mitochondrial Shape and Distribution Prior to Clinical Onset. PLoS ONE, 2011, 6, e22031.	2.5	116
93	An expansion in ALS genetics. Nature, 2010, 466, 1052-1053.	27.8	26
94	Polo-like kinase 4 kinase activity limits centrosome overduplication by autoregulating its own stability. Journal of Cell Biology, 2010, 188, 191-198.	5.2	251
95	Removal of Spindly from microtubule-attached kinetochores controls spindle checkpoint silencing in human cells. Genes and Development, 2010, 24, 957-971.	5.9	173
96	ALS-linked mutant superoxide dismutase 1 (SOD1) alters mitochondrial protein composition and decreases protein import. Proceedings of the National Academy of Sciences of the United States of America, 2010, 107, 21146-21151.	7.1	155
97	Centriole duplication. Cell Cycle, 2010, 9, 2803-2808.	2.6	43
98	ALS-associated mutations in TDP-43 increase its stability and promote TDP-43 complexes with FUS/TLS. Proceedings of the National Academy of Sciences of the United States of America, 2010, 107, 13318-13323.	7.1	391
99	Aurora Kinases and Protein Phosphatase 1 Mediate Chromosome Congression through Regulation of CENP-E. Cell, 2010, 142, 444-455.	28.9	207
100	Misfolded Mutant SOD1 Directly Inhibits VDAC1 Conductance in a Mouse Model of Inherited ALS. Neuron, 2010, 67, 575-587.	8.1	256
101	TDP-43 and FUS/TLS: emerging roles in RNA processing and neurodegeneration. Human Molecular Genetics, 2010, 19, R46-R64.	2.9	840
102	Double-strand DNA breaks recruit the centromeric histone CENP-A. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 15762-15767.	7.1	134
103	Phosphorylation of Highly Conserved Neurofilament Medium KSP Repeats Is Not Required for Myelin-Dependent Radial Axonal Growth. Journal of Neuroscience, 2009, 29, 1277-1284.	3.6	51
104	Boveri revisited: chromosomal instability, aneuploidy and tumorigenesis. Nature Reviews Molecular Cell Biology, 2009, 10, 478-487.	37.0	745
105	Unattached Kinetochores Catalyze Production of an Anaphase Inhibitor that Requires a Mad2 Template to Prime Cdc20 for BubR1 Binding. Developmental Cell, 2009, 16, 105-117.	7.0	225
106	Centromere-Specific Assembly of CENP-A Nucleosomes Is Mediated by HJURP. Cell, 2009, 137, 472-484.	28.9	588
107	Non–cell autonomous toxicity in neurodegenerative disorders: ALS and beyond. Journal of Cell Biology, 2009, 187, 761-772.	5.2	913
108	Activated protein C therapy slows ALS-like disease in mice by transcriptionally inhibiting SOD1 in motor neurons and microglia cells. Journal of Clinical Investigation, 2009, 119, 3437-49.	8.2	158

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109	Human Condensin Function Is Essential for Centromeric Chromatin Assembly and Proper Sister Kinetochore Orientation. PLoS ONE, 2009, 4, e6831.	2.5	73
110	The curious ways of ALS. Nature, 2008, 454, 284-285.	27.8	23
111	Astrocytes as determinants of disease progression in inherited amyotrophic lateral sclerosis. Nature Neuroscience, 2008, 11, 251-253.	14.8	1,015
112	Beyond Genetics: Surprising Determinants of Cell Fate in Antitumor Drugs. Cancer Cell, 2008, 14, 103-105.	16.8	12
113	The Aneuploidy Paradox in Cell Growth and Tumorigenesis. Cancer Cell, 2008, 14, 431-433.	16.8	93
114	Mutant SOD1 in cell types other than motor neurons and oligodendrocytes accelerates onset of disease in ALS mice. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 7594-7599.	7.1	258
115	Selective association of misfolded ALS-linked mutant SOD1 with the cytoplasmic face of mitochondria. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 4022-4027.	7.1	237
116	Common molecular signature in SOD1 for both sporadic and familial amyotrophic lateral sclerosis. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 12524-12529.	7.1	171
117	Propagation of centromeric chromatin requires exit from mitosis. Journal of Cell Biology, 2007, 176, 795-805.	5 . 2	558
118	An epigenetic mark generated by the incorporation of CENP-A into centromeric nucleosomes. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 5008-5013.	7.1	132
119	Comment on "A Centrosome-Independent Role for Â-TuRC Proteins in the Spindle Assembly Checkpoint". Science, 2007, 316, 982c-982c.	12.6	21
120	Toxicity from different SOD1 mutants dysregulates the complement system and the neuronal regenerative response in ALS motor neurons. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 7319-7326.	7.1	124
121	CENP-A-containing Nucleosomes: Easier Disassembly versus Exclusive Centromeric Localization. Journal of Molecular Biology, 2007, 370, 555-573.	4.2	126
122	Centromere Identity Maintained by Nucleosomes Assembled with Histone H3 Containing the CENP-A Targeting Domain. Molecular Cell, 2007, 25, 309-322.	9.7	231
123	An immunological epitope selective for pathological monomer-misfolded SOD1 in ALS. Nature Medicine, 2007, 13, 754-759.	30.7	199
124	Glial cells as intrinsic components of non-cell-autonomous neurodegenerative disease. Nature Neuroscience, 2007, 10, 1355-1360.	14.8	406
125	Aneuploidy Acts Both Oncogenically and as a Tumor Suppressor. Cancer Cell, 2007, 11, 25-36.	16.8	652
126	Amyotrophic lateral sclerosis and gene therapy. Nature Clinical Practice Neurology, 2006, 2, 462-463.	2.5	3

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127	Onset and Progression in Inherited ALS Determined by Motor Neurons and Microglia. Science, 2006, 312, 1389-1392.	12.6	1,457
128	ALS: A Disease of Motor Neurons and Their Nonneuronal Neighbors. Neuron, 2006, 52, 39-59.	8.1	1,271
129	The human CENP-A centromeric nucleosome-associated complex. Nature Cell Biology, 2006, 8, 458-469.	10.3	615
130	Progressive spinal axonal degeneration and slowness in ALS2â€deficient mice. Annals of Neurology, 2006, 60, 95-104.	5.3	69
131	Gene transfer demonstrates that muscle is not a primary target for non-cell-autonomous toxicity in familial amyotrophic lateral sclerosis. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 19546-19551.	7.1	140
132	Antisense oligonucleotide therapy for neurodegenerative disease. Journal of Clinical Investigation, 2006, 116, 2290-2296.	8.2	425
133	Elevation of the Hsp70 chaperone does not effect toxicity in mouse models of familial amyotrophic lateral sclerosis. Journal of Neurochemistry, 2005, 93, 875-882.	3.9	92
134	On the road to cancer: aneuploidy and the mitotic checkpoint. Nature Reviews Cancer, 2005, 5, 773-785.	28.4	1,046
135	Unstable microtubule capture at kinetochores depleted of the centromere-associated protein CENP-F. EMBO Journal, 2005, 24, 3927-3939.	7.8	104
136	Virusâ€delivered small RNA silencing sustains strength in amyotrophic lateral sclerosis. Annals of Neurology, 2005, 57, 773-776.	5.3	108
137	MEDICINE: Treating Neurodegenerative Diseases with Antibiotics. Science, 2005, 307, 361-362.	12.6	51
138	ZW10 links mitotic checkpoint signaling to the structural kinetochore. Journal of Cell Biology, 2005, 169, 49-60.	5.2	221
139	Microtubule capture by CENP-E silences BubR1-dependent mitotic checkpoint signaling. Journal of Cell Biology, 2005, 170, 873-880.	5.2	134
140	Altered axonal architecture by removal of the heavily phosphorylated neurofilament tail domains strongly slows superoxide dismutase 1 mutant-mediated ALS. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 10351-10356.	7.1	70
141	Human Zwint-1 Specifies Localization of Zeste White 10 to Kinetochores and Is Essential for Mitotic Checkpoint Signaling. Journal of Biological Chemistry, 2004, 279, 54590-54598.	3.4	106
142	Structural determinants for generating centromeric chromatin. Nature, 2004, 430, 578-582.	27.8	364
143	The Neuroprotective Factor Wld ^s Does Not Attenuate Mutant SOD1-Mediated Motor Neuron Disease. NeuroMolecular Medicine, 2004, 5, 193-204.	3.4	71
144	Lethality to human cancer cells through massive chromosome loss by inhibition of the mitotic checkpoint. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 8699-8704.	7.1	389

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145	UNRAVELING THE MECHANISMS INVOLVED IN MOTOR NEURON DEGENERATION IN ALS. Annual Review of Neuroscience, 2004, 27, 723-749.	10.7	1,270
146	Toxicity of Familial ALS-Linked SOD1 Mutants from Selective Recruitment to Spinal Mitochondria. Neuron, 2004, 43, 5-17.	8.1	497
147	Dynamics of Centromere and Kinetochore Proteins. Current Biology, 2004, 14, 942-952.	3.9	170
148	Has gene therapy for ALS arrived?. Nature Medicine, 2003, 9, 1256-1257.	30.7	9
149	Centromeres and Kinetochores. Cell, 2003, 112, 407-421.	28.9	926
150	Activating and Silencing the Mitotic Checkpoint through CENP-E-Dependent Activation/Inactivation of BubR1. Cell, 2003, 114, 87-98.	28.9	221
151	Centromere-associated protein-E is essential for the mammalian mitotic checkpoint to prevent aneuploidy due to single chromosome loss. Journal of Cell Biology, 2003, 162, 551-563.	5.2	233
152	Gene replacement in mice reveals that the heavily phosphorylated tail of neurofilament heavy subunit does not affect axonal caliber or the transit of cargoes in slow axonal transport. Journal of Cell Biology, 2002, 158, 681-693.	5.2	124
153	Focal loss of the glutamate transporter EAAT2 in a transgenic rat model of SOD1 mutant-mediated amyotrophic lateral sclerosis (ALS). Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 1604-1609.	7.1	766
154	Motoneuron Death Triggered by a Specific Pathway Downstream of Fas. Neuron, 2002, 35, 1067-1083.	8.1	407
155	Unstable Kinetochore-Microtubule Capture and Chromosomal Instability Following Deletion of CENP-E. Developmental Cell, 2002, 3, 351-365.	7.0	295
156	Slow axonal transport: fast motors in the slow lane. Current Opinion in Cell Biology, 2002, 14, 58-62.	5.4	80
157	Mutant SOD1 causes motor neuron disease independent of copper chaperone–mediated copper loading. Nature Neuroscience, 2002, 5, 301-307.	14.8	253
158	Mps1 Is a Kinetochore-Associated Kinase Essential for the Vertebrate Mitotic Checkpoint. Cell, 2001, 106, 83-93.	28.9	303
159	Hypoxia and Lou Gehrig. Nature Genetics, 2001, 28, 107-108.	21.4	23
160	From charcot to lou gehrig: deciphering selective motor neuron death in als. Nature Reviews Neuroscience, 2001, 2, 806-819.	10.2	1,264
161	Formation of advanced glycation endâ€productâ€modified superoxide dismutaseâ€1 (SOD1) is one of the mechanisms responsible for inclusions common to familial amyotrophic lateral sclerosis patients with SOD1 gene mutation, and transgenic mice expressing human SOD1 gene mutation. Neuropathology, 2001, 21, 67-81.	1.2	24
162	Going new places using an old MAP: tau, microtubules and human neurodegenerative disease. Current Opinion in Cell Biology, 2001, 13, 41-48.	5.4	218

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163	CELL CYCLE: Some Importin News About Spindle Assembly. Science, 2001, 291, 1718-1719.	12.6	26
164	Oxidation versus aggregation â€" how do SOD1 mutants cause ALS?. Nature Medicine, 2000, 6, 1320-1321.	30.7	91
165	CENP-E forms a link between attachment of spindle microtubules to kinetochores and the mitotic checkpoint. Nature Cell Biology, 2000, 2, 484-491.	10.3	343
166	Toxicity of ALS-Linked SOD1 Mutants. Science, 2000, 288, 399a-399.	12.6	45
167	CENP-E as an Essential Component of the Mitotic Checkpoint In Vitro. Cell, 2000, 102, 817-826.	28.9	211
168	The Copper Chaperone CCS Is Abundant in Neurons and Astrocytes in Human and Rodent Brain. Journal of Neurochemistry, 1999, 72, 422-429.	3.9	107
169	Slowing of axonal transport is a very early event in the toxicity of ALS–linked SOD1 mutants to motor neurons. Nature Neuroscience, 1999, 2, 50-56.	14.8	559
170	From Charcot to SOD1. Neuron, 1999, 24, 515-520.	8.1	222
171	Aggregation and Motor Neuron Toxicity of an ALS-Linked SOD1 Mutant Independent from Wild-Type SOD1. Science, 1998, 281, 1851-1854.	12.6	1,038
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