Harry T Orr

List of Publications by Year
in descending order

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169 papers	22,192 citations	13068 68 h-index	8835 145 g-index
179 all docs	179 docs citations	179 times ranked	12393 citing authors

#	Article	IF	Citations
1	Expansion of an unstable trinucleotide CAG repeat in spinocerebellar ataxia type 1. Nature Genetics, 1993, 4, 221-226.	9.4	1,673
2	Trinucleotide Repeat Disorders. Annual Review of Neuroscience, 2007, 30, 575-621.	5.0	1,289
3	Glutamine Repeats and Neurodegeneration. Annual Review of Neuroscience, 2000, 23, 217-247.	5.0	1,243
4	Ataxin-1 Nuclear Localization and Aggregation. Cell, 1998, 95, 41-53.	13.5	965
5	Chaperone suppression of aggregation and altered subcellular proteasome localization imply protein misfolding in SCA1. Nature Genetics, 1998, 19, 148-154.	9.4	802
6	Identification of genes that modify ataxin-1-induced neurodegeneration. Nature, 2000, 408, 101-106.	13.7	648
7	RNAi suppresses polyglutamine-induced neurodegeneration in a model of spinocerebellar ataxia. Nature Medicine, 2004, 10, 816-820.	15.2	643
8	SCA1 transgenic mice: A model for neurodegeneration caused by an expanded CAG trinucleotide repeat. Cell, 1995, 82, 937-948.	13.5	567
9	Ataxin-1 with an expanded glutamine tract alters nuclear matrix-associated structures. Nature, 1997, 389, 971-974.	13.7	531
10	Major histocompatibility antigens: The human (HLA-A,-B,-C) and murine (H-2K, H-2D) class I molecules. Cell, 1981, 24, 287-299.	13.5	517
11	Evidence for a mechanism predisposing to intergenerational CAG repeat instability in spinocerebellar ataxia type I. Nature Genetics, 1993, 5, 254-258.	9.4	489
12	Mutation of the E6-AP Ubiquitin Ligase Reduces Nuclear Inclusion Frequency While Accelerating Polyglutamine-Induced Pathology in SCA1 Mice. Neuron, 1999, 24, 879-892.	3.8	482
13	Interaction of Akt-Phosphorylated Ataxin-1 with 14-3-3 Mediates Neurodegeneration in Spinocerebellar Ataxia Type 1. Cell, 2003, 113, 457-468.	13.5	402
14	Identification and characterization of the gene causing type 1 spinocerebellar ataxia. Nature Genetics, 1994, 7, 513-520.	9.4	362
15	Polyglutamine expansion down-regulates specific neuronal genes before pathologic changes in SCA1. Nature Neuroscience, 2000, 3, 157-163.	7.1	341
16	Spinocerebellar ataxia type 5 in a family descended from the grandparents of President Lincoln maps to chromosome 11. Nature Genetics, 1994, 8, 280-284.	9.4	334
17	A Long CAG Repeat in the Mouse Sca1 Locus Replicates SCA1 Features and Reveals the Impact of Protein Solubility on Selective Neurodegeneration. Neuron, 2002, 34, 905-919.	3.8	320
18	Serine 776 of Ataxin-1 Is Critical for Polyglutamine-Induced Disease in SCA1 Transgenic Mice. Neuron, 2003, 38, 375-387.	3.8	303

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19	Expression analysis of the ataxinâ€“1 protein in tissues from normal and spinocerebellar ataxia type 1 individuals. Nature Genetics, 1995, 10, 94-98.	9.4	291
20	Opposing effects of polyglutamine expansion on native protein complexes contribute to SCA1. Nature, 2008, 452, 713-718.	13.7	287
21	Assembly and maturation of HLA-A and HLA-B antigens in vivo. Cell, 1979, 18, 979-991.	13.5	286
22	ATAXIN-1 Interacts with the Repressor Capicua in Its Native Complex to Cause SCA1 Neuropathology. Cell, 2006, 127, 1335-1347.	13.5	284
23	Polyglutamine spinocerebellar ataxias â€” from genes to potential treatments. Nature Reviews Neuroscience, 2017, 18, 613-626.	4.9	270
24	Purkinje Cell Expression of a Mutant Allele ofSCA1in Transgenic Mice Leads to Disparate Effects on Motor Behaviors, Followed by a Progressive Cerebellar Dysfunction and Histological Alterations. Journal of Neuroscience, 1997, 17, 7385-7395.	1.7	261
25	Recovery from Polyglutamine-Induced Neurodegeneration in Conditional SCA1 Transgenic Mice. Journal of Neuroscience, 2004, 24, 8853-8861.	1.7	257
26	The cerebellar leucine-rich acidic nuclear protein interacts with ataxin-1. Nature, 1997, 389, 974-978.	13.7	246
27	Differential expression of HLA-E, HLA-F, and HLA-G transcripts in human tissue. Human Immunology, 1990, 29, 131-142.	1.2	219
28	RORÎ±-Mediated Purkinje Cell Development Determines Disease Severity in Adult SCA1 Mice. Cell, 2006, 127, 697-708.	13.5	210
29	Pathogenic Mechanisms of a Polyglutamine-mediated Neurodegenerative Disease, Spinocerebellar Ataxia Type 1. Journal of Biological Chemistry, 2009, 284, 7425-7429.	1.6	206
30	Disrupted cerebellar cortical development and progressive degeneration of Purkinje cells in SV40 T antigen transgenic mice. Neuron, 1992, 9, 955-966.	3.8	201
31	Mice Lacking Ataxin-1 Display Learning Deficits and Decreased Hippocampal Paired-Pulse Facilitation. Journal of Neuroscience, 1998, 18, 5508-5516.	1.7	197
32	miR-19, miR-101 and miR-130 co-regulate ATXN1 levels to potentially modulate SCA1 pathogenesis. Nature Neuroscience, 2008, 11, 1137-1139.	7.1	194
33	The Unstable Repeatsâ€”Three Evolving Faces of Neurological Disease. Neuron, 2013, 77, 825-843.	3.8	192
34	The AXH Domain of Ataxin-1 Mediates Neurodegeneration through Its Interaction with Gfi-1/Senseless Proteins. Cell, 2005, 122, 633-644.	13.5	189
35	The GSK3Î² signaling cascade and neurodegenerative disease. Current Opinion in Neurobiology, 2002, 12, 275-278.	2.0	188
36	Gametic and somatic tissueâ€“specific heterogeneity of the expanded SCA1 CAG repeat in spinocerebellar ataxia type 1. Nature Genetics, 1995, 10, 344-350.	9.4	179

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37	Tolerance is established in polyclonal CD4+ T cells by distinct mechanisms, according to self-peptide expression patterns. Nature Immunology, 2016, 17, 187-195.	7.0	178
38	Gene profiling links SCA1 pathophysiology to glutamate signaling in Purkinje cells of transgenic mice. Human Molecular Genetics, 2004, 13, 2535-2543.	1.4	168
39	HLA and maternalâ€fetal recognition. FASEB Journal, 1992, 6, 2344-2348.	0.2	151
40	Lithium Therapy Improves Neurological Function and Hippocampal Dendritic Arborization in a Spinocerebellar Ataxia Type 1 Mouse Model. PLoS Medicine, 2007, 4, e182.	3.9	147
41	Exercise and Genetic Rescue of SCA1 via the Transcriptional Repressor Capicua. Science, 2011, 334, 690-693.	6.0	144
42	Cell biology of spinocerebellar ataxia. Journal of Cell Biology, 2012, 197, 167-177.	2.3	144
43	Pumilio1 Haploinsufficiency Leads to SCA1-like Neurodegeneration by Increasing Wild-Type Ataxin1 Levels. Cell, 2015, 160, 1087-1098.	13.5	139
44	SCA1-like Disease in Mice Expressing Wild-Type Ataxin-1 with a Serine to Aspartic Acid Replacement at Residue 776. Neuron, 2010, 67, 929-935.	3.8	137
45	Aminopyridines Correct Early Dysfunction and Delay Neurodegeneration in a Mouse Model of Spinocerebellar Ataxia Type 1. Journal of Neuroscience, 2011, 31, 11795-11807.	1.7	137
46	The heavy chain of human histocompatibility antigen HLA-B7 contains an immunoglobulin-like region. Nature, 1979, 282, 266-270.	13.7	134
47	Polyglutamine neurodegenerative diseases and regulation of transcription: assembling the puzzle. Genes and Development, 2006, 20, 2183-2192.	2.7	129
48	Complete amino acid sequence of a papain-solubilized human histocompatibility antigen, HLA-B7. 2. Sequence determination and search for homologies. Biochemistry, 1979, 18, 5711-5720.	1.2	127
49	Spinocerebellar ataxia type 1. Seminars in Cell Biology, 1995, 6, 29-35.	3.5	125
50	RASâ€“MAPKâ€“MSK1 pathway modulates ataxin 1 protein levels and toxicity in SCA1. Nature, 2013, 498, 325-331.	13.7	119
51	Partial Loss of Ataxin-1 Function Contributes to Transcriptional Dysregulation in Spinocerebellar Ataxia Type 1 Pathogenesis. PLoS Genetics, 2010, 6, e1001021.	1.5	113
52	Disruption of the ATXN1â€“CIC complex causes a spectrum of neurobehavioral phenotypes in mice and humans. Nature Genetics, 2017, 49, 527-536.	9.4	113
53	RNA association and nucleocytoplasmic shuttling by ataxin-1. Journal of Cell Science, 2005, 118, 233-242.	1.2	109
54	Antisense oligonucleotideâ€“mediated ataxin-1 reduction prolongs survival in SCA1 mice and reveals disease-associated transcriptome profiles. JCI Insight, 2018, 3, .	2.3	106

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55	Polyglutamine diseases: protein cleavage and aggregation. Current Opinion in Neurobiology, 1999, 9, 566-570.	2.0	102
56	Increased Trinucleotide Repeat Instability with Advanced Maternal Age. Human Molecular Genetics, 1997, 6, 2135-2139.	1.4	97
57	Neuronal Atrophy Early in Degenerative Ataxia Is a Compensatory Mechanism to Regulate Membrane Excitability. Journal of Neuroscience, 2015, 35, 11292-11307.	1.7	93
58	ATXN1 Protein Family and CIC Regulate Extracellular Matrix Remodeling and Lung Alveolarization. Developmental Cell, 2011, 21, 746-757.	3.1	89
59	SUMOylation of the Polyglutamine Repeat Protein, Ataxin-1, Is Dependent on a Functional Nuclear Localization Signal. Journal of Biological Chemistry, 2005, 280, 21942-21948.	1.6	87
60	Cerebellar Transcriptome Profiles of ATXN1 Transgenic Mice Reveal SCA1 Disease Progression and Protection Pathways. Neuron, 2016, 89, 1194-1207.	3.8	86
61	The insulin-like growth factor pathway is altered in spinocerebellar ataxia type 1 and type 7. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 1291-1296.	3.3	85
62	Noninvasive Detection of Presymptomatic and Progressive Neurodegeneration in a Mouse Model of Spinocerebellar Ataxia Type 1. Journal of Neuroscience, 2010, 30, 3831-3838.	1.7	85
63	Use of HLA loss mutants to analyse the structure of the human major histocompatibility complex. Nature, 1982, 296, 454-456.	13.7	84
64	Altered Trafficking of Membrane Proteins in Purkinje Cells of SCA1 Transgenic Mice. American Journal of Pathology, 2001, 159, 905-913.	1.9	83
65	Reversing Neurodegeneration:A Promise Unfolds. Cell, 2000, 101, 1-4.	13.5	81
66	ATXN1-CIC Complex Is the Primary Driver of Cerebellar Pathology in Spinocerebellar Ataxia Type 1 through a Gain-of-Function Mechanism. Neuron, 2018, 97, 1235-1243.e5.	3.8	79
67	Emerging pathogenic pathways in the spinocerebellar ataxias. Current Opinion in Genetics and Development, 2009, 19, 247-253.	1.5	77
68	Duplication of Atxn1l suppresses SCA1 neuropathology by decreasing incorporation of polyglutamine-expanded ataxin-1 into native complexes. Nature Genetics, 2007, 39, 373-379.	9.4	75
69	Abnormalities in the Climbing Fiber-Purkinje Cell Circuitry Contribute to Neuronal Dysfunction inATXN1[82Q] Mice. Journal of Neuroscience, 2011, 31, 12778-12789.	1.7	75
70	In Vivo Viability of Postmitotic Purkinje Neurons Requires pRb Family Member Function. Molecular and Cellular Neurosciences, 1995, 6, 153-167.	1.0	70
71	Targeted Deletion of a Single Sca8 Ataxia Locus Allele in Mice Causes Abnormal Gait, Progressive Loss of Motor Coordination, and Purkinje Cell Dendritic Deficits. Journal of Neuroscience, 2006, 26, 9975-9982.	1.7	70
72	Polyglutamine neurodegeneration: expanded glutamines enhance native functions. Current Opinion in Genetics and Development, 2012, 22, 251-255.	1.5	69

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73	Class I-like HLA genes map telomeric to the HLA-A2 locus in human cells. Nature, 1983, 302, 534-536.	13.7	68
74	Extensive cryptic splicing upon loss of RBM17 and TDP43 in neurodegeneration models. Human Molecular Genetics, 2016, 25, ddw337.	1.4	68
75	Progress in pathogenesis studies of spinocerebellar ataxia type 1. Philosophical Transactions of the Royal Society B: Biological Sciences, 1999, 354, 1079-1081.	1.8	66
76	Regional rescue of spinocerebellar ataxia type 1 phenotypes by 14-3-3 Îµ haploinsufficiency in mice underscores complex pathogenicity in neurodegeneration. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 2142-2147.	3.3	65
77	HLA non-A,B,C class I genes: Their structure and expression. Immunologic Research, 1990, 9, 265-274.	1.3	63
78	Mapmodulin/Leucine-rich Acidic Nuclear Protein Binds the Light Chain of Microtubule-associated Protein 1B and Modulates Neuritogenesis. Journal of Biological Chemistry, 2003, 278, 34691-34699.	1.6	62
79	Cloning and Developmental Expression Analysis of the Murine Homolog of the Spinocerebellar Ataxia Type 1 Gene (Sca1). Human Molecular Genetics, 1996, 5, 33-40.	1.4	59
80	Organization of the human class I major histocompatibility complex genes. Immunologic Research, 1987, 6, 1-10.	1.3	58
81	Partial loss of Tip60 slows mid-stage neurodegeneration in a spinocerebellar ataxia type 1 (SCA1) mouse model. Human Molecular Genetics, 2011, 20, 2204-2212.	1.4	58
82	Calcium Dynamics and Electrophysiological Properties of Cerebellar Purkinje Cells in SCA1 Transgenic Mice. Journal of Neurophysiology, 2001, 85, 1750-1760.	0.9	57
83	Phosphorylation of ATXN1 at Ser776 in the cerebellum. Journal of Neurochemistry, 2009, 110, 675-686.	2.1	55
84	Regional differences of somatic CAG repeat instability do not account for selective neuronal vulnerability in a knock-in mouse model of SCA1. Human Molecular Genetics, 2003, 12, 2789-2795.	1.4	54
85	Molecular analysis of the variant alloantigen HLA-B27d (HLA-Bâ^—2703) identifies a unique single amino acid substitution. Human Immunology, 1988, 21, 209-219.	1.2	53
86	Purkinje Cell Ataxin-1 Modulates Climbing Fiber Synaptic Input in Developing and Adult Mouse Cerebellum. Journal of Neuroscience, 2013, 33, 5806-5820.	1.7	50
87	Mapping of class I DNA sequences within the human major histocompatibility complex. Immunogenetics, 1983, 18, 489-502.	1.2	49
88	Spinocerebellar Ataxia Type 1â€"Modeling the Pathogenesis of a Polyglutamine Neurodegenerative Disorder in Transgenic Mice. Journal of Neuropathology and Experimental Neurology, 2000, 59, 265-270.	0.9	49
89	14-3-3 Binding to Ataxin-1(ATXN1) Regulates Its Dephosphorylation at Ser-776 and Transport to the Nucleus. Journal of Biological Chemistry, 2011, 286, 34606-34616.	1.6	49
90	The Effects of the Polyglutamine Repeat Protein Ataxin-1 on the UbL-UBA Protein A1Up. Journal of Biological Chemistry, 2004, 279, 42290-42301.	1.6	44

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91	Reduction of Purkinje Cell Pathology in SCA1 Transgenic Mice by p53 Deletion. Neurobiology of Disease, 2001, 8, 974-981.	2.1	39
92	Generation and Characterization of LANP/pp32 Null Mice. Molecular and Cellular Biology, 2004, 24, 3140-3149.	1.1	38
93	Complete amino acid sequence of a papain-solubilized human histocompatibility antigen, HLA-B7. 1. Isolation and amino acid composition of fragments and of tryptic and chymotryptic peptides. Biochemistry, 1979, 18, 5704-5711.	1.2	36
94	SCA1â€"Phosphorylation, a regulator of Ataxin-1 function and pathogenesis. Progress in Neurobiology, 2012, 99, 179-185.	2.8	33
95	RNA Targets of the Fragile X Protein. Cell, 2001, 107, 555-557.	13.5	30
96	Neuron protection agency. Nature, 2004, 431, 747-748.	13.7	30
97	Polyglutamine Disease Toxicity Is Regulated by Nemo-like Kinase in Spinocerebellar Ataxia Type 1. Journal of Neuroscience, 2013, 33, 9328-9336.	1.7	30
98	HLA-G Transgenic Mice: A Model for Studying Expression and Function at the Maternal/Fetal Interface. Immunological Reviews, 1995, 147, 53-65.	2.8	29
99	In vivo monitoring of recovery from neurodegeneration in conditional transgenic SCA1 mice. Experimental Neurology, 2011, 232, 290-298.	2.0	29
100	A native interactor scaffolds and stabilizes toxic ATAXIN-1 oligomers in SCA1. ELife, 2015, 4, .	2.8	29
101	FTD and ALS: Genetic Ties that Bind. Neuron, 2011, 72, 189-190.	3.8	28
102	The Transcription Factor E2F-1 in SV40 T Antigen-Induced Cerebellar Purkinje Cell Degeneration. Molecular and Cellular Neurosciences, 1998, 12, 16-28.	1.0	27
103	A cell-based screen for modulators of ataxin-1 phosphorylation. Human Molecular Genetics, 2005, 14, 1095-1105.	1.4	27
104	Reduction of protein kinase A-mediated phosphorylation of ATXN1-S776 in Purkinje cells delays onset of Ataxia in a SCA1 mouse model. Neurobiology of Disease, 2018, 116, 93-105.	2.1	27
105	Targeting inhibitory cerebellar circuitry to alleviate behavioral deficits in a mouse model for studying idiopathic autism. Neuropsychopharmacology, 2020, 45, 1159-1170.	2.8	26
106	Assessing recovery from neurodegeneration in spinocerebellar ataxia 1: Comparison of in vivo magnetic resonance spectroscopy with motor testing, gene expression and histology. Neurobiology of Disease, 2015, 74, 158-166.	2.1	25
107	Susceptibility to Cell Death Induced by Mutant SV40 T-Antigen Correlates with Purkinje Neuron Functional Development. Molecular and Cellular Neurosciences, 1997, 9, 42-62.	1.0	24
108	Motor neuron degeneration correlates with respiratory dysfunction in SCA1. DMM Disease Models and Mechanisms, 2018, 11, .	1.2	23

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109	The Ins and Outs of a Polyglutamine Neurodegenerative Disease: Spinocerebellar Ataxia Type 1 (SCA1). Neurobiology of Disease, 2000, 7, 129-134.	2.1	22
110	Amino Acids in a Region of Ataxin-1 Outside of the Polyglutamine Tract Influence the Course of Disease in SCA1 Transgenic Mice. NeuroMolecular Medicine, 2002, 1, 33-42.	1.8	22
111	Diminishing return for mechanistic therapeutics with neurodegenerative disease duration?. BioEssays, 2016, 38, 977-980.	1.2	22
112	Linkage of an Alzheimer disease susceptibility locus to markers on human chromosome 21. American Journal of Medical Genetics Part A, 1991, 40, 449-453.	2.4	21
113	Protocol for genetic testing in Huntington disease: Three years of experience in Minnesota. American Journal of Medical Genetics Part A, 1991, 40, 518-522.	2.4	21
114	Genetically engineered mouse models of the trinucleotide-repeat spinocerebellar ataxias. Brain Research Bulletin, 2012, 88, 33-42.	1.4	21
115	Cytotoxic T Lymphocyte Recognition of HLA-G in Mice. Human Immunology, 1997, 55, 127-139.	1.2	20
116	Mouse Models of Human CAG Repeat Disorders. Brain Pathology, 1997, 7, 965-977.	2.1	20
117	Altered Capicua expression drives regional Purkinje neuron vulnerability through ion channel gene dysregulation in spinocerebellar ataxia type 1. Human Molecular Genetics, 2020, 29, 3249-3265.	1.4	20
118	Molecular and endocrine characterization of a mutation involving a recombination between the steroid 21-hydroxylase functional gene and pseudogene. Journal of Steroid Biochemistry and Molecular Biology, 1991, 38, 677-686.	1.2	19
119	Spinocerebellar Ataxia Type 1: Molecular Mechanisms of Neurodegeneration and Preclinical Studies. Advances in Experimental Medicine and Biology, 2018, 1049, 135-145.	0.8	19
120	Characterization of the Zebrafishatxn1/axhGene Family. Journal of Neurogenetics, 2009, 23, 313-323.	0.6	18
121	Use of DNA probes from the 5â€² flanking region of the HLA-B gene to examine polymorphism at the HLA-B locus. Human Immunology, 1986, 16, 137-147.	1.2	16
122	Diagnosis of classical steroid 21-hydroxylase deficiency using an HLA-B locus-specific DNA-probe. American Journal of Medical Genetics Part A, 1988, 29, 703-712.	2.4	16
123	PAK1 regulates ATXN1 levels providing an opportunity to modify its toxicity in spinocerebellar ataxia type 1. Human Molecular Genetics, 2018, 27, 2863-2873.	1.4	16
124	Antisense Oligonucleotide Therapeutic Approach for Suppression of Ataxin-1 Expression: A Safety Assessment. Molecular Therapy - Nucleic Acids, 2020, 21, 1006-1016.	2.3	16
125	Ataxin-1 oligomers induce local spread of pathology and decreasing them by passive immunization slows Spinocerebellar ataxia type 1 phenotypes. ELife, 2015, 4, .	2.8	16
126	Autistic-like behavior and cerebellar dysfunction in Bmal1 mutant mice ameliorated by mTORC1 inhibition. Molecular Psychiatry, 2023, 28, 3727-3738.	4.1	16

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127	Microarrays and polyglutamine disorders: reports from the Hereditary Disease Array Group. Human Molecular Genetics, 2002, 11, 1909-1910.	1.4	15
128	Identification of a novel phosphorylation site in ataxin-1. Biochimica Et Biophysica Acta - Molecular Cell Research, 2005, 1744, 11-18.	1.9	15
129	Visualizing and mapping the cerebellum with serial optical coherence scanner. Neurophotonics, 2016, 4, 1.	1.7	15
130	The ataxin-1 interactome reveals direct connection with multiple disrupted nuclear transport pathways. Nature Communications, 2020, 11, 3343.	5.8	15
131	Consensus Paper: Strengths and Weaknesses of Animal Models of Spinocerebellar Ataxias and Their Clinical Implications. Cerebellum, 2022, 21, 452-481.	1.4	15
132	Overexpression of CREB reduces CRE-mediated transcription: behavioral and cellular analyses in transgenic mice. Molecular and Cellular Neurosciences, 2004, 25, 602-611.	1.0	12
133	Hsp70/Hsc70 regulates the effect phosphorylation has on stabilizing ataxin-1. Journal of Neurochemistry, 2007, 102, 2040-2048.	2.1	12
134	Modulation of ATXN1 S776 phosphorylation reveals the importance of allele-specific targeting in SCA1. JCI Insight, 2021, 6, .	2.3	12
135	Unstable trinucleotide repeats and the diagnosis of neurodegenerative disease. Human Pathology, 1994, 25, 598-601.	1.1	11
136	Treadmill training increases the motor activity and neuron survival of the cerebellum in a mouse model of spinocerebellar ataxia type 1. Kaohsiung Journal of Medical Sciences, 2019, 35, 679-685.	0.8	11
137	Dual targeting of brain regionâ€specific kinases potentiates neurological rescue in Spinocerebellar ataxia type 1. EMBO Journal, 2021, 40, e106106.	3.5	11
138	Cholecystokinin 1 receptor activation restores normal mTORC1 signaling and is protective to Purkinje cells of SCA mice. Cell Reports, 2021, 37, 109831.	2.9	11
139	Sequence of a murine cDNA, pcp-4, that encodes the homolog of the rat brain-specific antigen PEP-19. Nucleic Acids Research, 1990, 18, 1304-1304.	6.5	10
140	Pathogenesis of Polyglutamine-Induced Disease: A Model for SCA1. Molecular Genetics and Metabolism, 1999, 66, 172-178.	0.5	10
141	Polarization-sensitive optical coherence tomography reveals gray matter and white matter atrophy in SCA1 mouse models. Neurobiology of Disease, 2018, 116, 69-77.	2.1	10
142	Regulation of Ataxin-1 Phosphorylation and Its Impact on Biology. Methods in Molecular Biology, 2013, 1010, 201-209.	0.4	10
143	HLA Class I Gene Family: Characterization of Genes Encoding Non-HLA-A,B,C Proteins. , 1989, , 33-40.		10
144	Qs in the Nucleus. Neuron, 2001, 31, 875-876.	3.8	9

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145	RNA gains a new function: a mediator of neurodegeneration. Trends in Neurosciences, 2004, 27, 233-234.	4.2	9
146	Lurcher, nPIST, and Autophagy. Neuron, 2002, 35, 813-814.	3.8	8
147	Nuclear Ataxias. Cold Spring Harbor Perspectives in Biology, 2010, 2, a000786-a000786.	2.3	8
148	Toxic RNA as a driver of disease in a common form of ALS and dementia. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 7533-7534.	3.3	8
149	Antisense RNA Sequences Modulating the Ataxin-1 Message: Molecular Model of Gene Therapy for Spinocerebellar Ataxia Type 1, a Dominant-Acting Unstable Trinucleotide Repeat Disease. Cell Transplantation, 2008, 17, 723-734.	1.2	7
150	Unstable Nucleotide Repeat Minireview Series: A Molecular Biography of Unstable Repeat Disorders. Journal of Biological Chemistry, 2009, 284, 7405.	1.6	7
151	Reduction of mutant ATXN1 rescues premature death in a conditional SCA1 mouse model. JCI Insight, 2022, 7, .	2.3	6
152	Cross-species genetic screens identify transglutaminase 5 as a regulator of polyglutamine-expanded ataxin-1. Journal of Clinical Investigation, 2022, 132, .	3.9	6
153	The Major Histocompatibility Complex: Analysis at the Protein and DNA Levels. , 1982, , 1-51.		5
154	Into the depths of ataxia. Journal of Clinical Investigation, 2004, 113, 505-507.	3.9	5
155	Chapter 4 Complete Primary Structure of Human Histocompatibility Antigen Hla-B7: Evolutionary and Functional Implications. Current Topics in Developmental Biology, 1980, 14, 97-113.	1.0	3
156	Are Polyglutamine Diseases Expanding?. Neuron, 2011, 70, 377-378.	3.8	3
157	Stephen T. Warren, Ph.D. (1953â€“2021): A remembrance. American Journal of Human Genetics, 2022, 109, 3-11.	2.6	2
158	Cholecystokinin Activation of Cholecystokinin 1 Receptors: a Purkinje Cell Neuroprotective Pathway. Cerebellum, 2023, 22, 756-760.	1.4	2
159	Complete Primary Structure of Human Histocompatibility Antigen HLA-B7. , 1981, , 479-493.		1
160	Transfer and Expression of Human Non-A,B,C Class I Genes in Human HLA A,B,C Null Lymphoblastoid Cells. , 1989, , 159-161.		1
161	Animal Models of Spinocerebellar Ataxia Type 1 (SCA1). , 2005, , 623-630.		0
162	Animal Models of Spinocerebellar Ataxia Type 1. , 2015, , 979-990.		0

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163	Cholecystokinin 1 Receptor (Cck1R) Activates mTORC1 Signaling and is Protective to Purkinje Cells in SCA Mice. SSRN Electronic Journal, 0, , .	0.4	0
164	Spinocerebellar ataxia type 1. , 2001, , 409-418.		0
165	Spinocerebellar Ataxia 1 (SCA1). , 2003, , 35-43.		0
166	Phosphorylation of Ataxin-1: A Link Between Basic Research and Clinical Application in Spinocerebellar Ataxia Type 1. , 2006, , 339-349.		0
167	Spinocerebellar Ataxia Type 1. , 2007, , 149-155.		0
168	Transgenic Mouse Models of CAG Trinucleotide Repeat Neurologic Diseases. , 1999, , 163-185.		0
169	Spinocerebellar Ataxia Type 1. Contemporary Clinical Neuroscience, 2006, , 87-99.	0.3	0