Thorsten Schmidt

List of Publications by Year
in descending order

33	2,841	394286 19	454834 30
papers	citations	h-index	g-index

37	37	37	3357
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	ULK overexpression mitigates motor deficits and neuropathology in mouse models of Machado-Joseph disease. Molecular Therapy, 2022, 30, 370-387.	3.7	10
2	A Novel SCA3 Knock-in Mouse Model Mimics the Human SCA3 Disease Phenotype Including Neuropathological, Behavioral, and Transcriptional Abnormalities Especially in Oligodendrocytes. Molecular Neurobiology, 2022, 59, 495-522.	1.9	22
3	Neurodegenerative phosphoprotein signaling landscape inÂmodels of SCA3. Molecular Brain, 2021, 14, 57.	1.3	2
4	Pathophysiological interplay between O -GlcNAc transferase and the Machadoâ€“Joseph disease protein ataxin-3. Proceedings of the National Academy of Sciences of the United States of America, 2021, 118, .	3.3	5
5	The impact of an audience response system on a summative assessment, a controlled field study. BMC Medical Education, 2020, 20, 218.	1.0	6
6	Integration moderner Lehrmethoden in den Humangenetik-Unterricht in TÃ¼bingen. Medizinische Genetik, 2019, 31, 313-319.	0.1	1
7	Divalproex sodium regulates ataxin-3 translocation likely by an importin Î±1-dependent pathway. NeuroReport, 2019, 30, 760-764.	0.6	3
8	Vulnerability of frontal brain neurons for the toxicity of expanded ataxin-3. Human Molecular Genetics, 2019, 28, 1463-1473.	1.4	9
9	Physiological and pathophysiological characteristics of ataxin-3 isoforms. Journal of Biological Chemistry, 2019, 294, 644-661.	1.6	36
10	Karyopherin Î±-3 is a key protein in the pathogenesis of spinocerebellar ataxia type 3 controlling the nuclear localization of ataxin-3. Proceedings of the National Academy of Sciences of the United States of America, 2018, 115, E2624-E2633.	3.3	38
11	Animal Models of Machado-Joseph Disease. Advances in Experimental Medicine and Biology, 2018, 1049, 289-308.	0.8	0
12	Divalproex sodium modulates nuclear localization of ataxinâ€3 and prevents cellular toxicity caused by expanded ataxinâ€3. CNS Neuroscience and Therapeutics, 2018, 24, 404-411.	1.9	14
13	Mass spectrometry analyses of normal and polyglutamine expanded ataxin-3 reveal novel interaction partners involved in mitochondrial function. Neurochemistry International, 2018, 112, 5-17.	1.9	22
14	In vivo assessment of riluzole as a potential therapeutic drug for spinocerebellar ataxia type 3. Journal of Neurochemistry, 2016, 138, 150-162.	2.1	27
15	Consensus Paper: Pathological Mechanisms Underlying Neurodegeneration in Spinocerebellar Ataxias. Cerebellum, 2014, 13, 269-302.	1.4	114
16	Acetazolamide-responsive exercise-induced episodic ataxia associated with a novel homozygous DARS2 mutation. Journal of Medical Genetics, 2011, 48, 713-715.	1.5	45
17	Atlas of transgenic Tet-Off Ca2+/calmodulin-dependent protein kinase II and prion protein promoter activity in the mouse brain. NeuroImage, 2011, 54, 2603-2611.	2.1	21
18	N-terminal ataxin-3 causes neurological symptoms with inclusions, endoplasmic reticulum stress and ribosomal dislocation. Brain, 2011, 134, 1925-1942.	3.7	52

#	Article	IF	Citations
19	Erythropoietin receptor expression in normal and neoplastic choroid plexus. , 2011, 30, 33-40.		2
20	A transgenic mouse model of spinocerebellar ataxia type 3 resembling late disease onset and gender-specific instability of CAG repeats. Neurobiology of Disease, 2010, 37, 284-293.	2.1	51
21	Knockdown of transactive response DNA-binding protein (TDP-43) downregulates histone deacetylase 6. EMBO Journal, 2010, 29, 209-221.	3.5	200
22	Reversibility of symptoms in a conditional mouse model of spinocerebellar ataxia type 3. Human Molecular Genetics, 2009, 18, 4282-4295.	1.4	97
23	Identification and functional dissection of localization signals within ataxin-3. Neurobiology of Disease, 2009, 36, 280-292.	2.1	42
24	Neurodegeneration and Motor Dysfunction in a Conditional Model of Parkinson's Disease. Journal of Neuroscience, 2008, 28, 2471-2484.	1.7	164
25	Nuclear Localization of Ataxin-3 Is Required for the Manifestation of Symptoms in SCA3:In VivoEvidence. Journal of Neuroscience, 2007, 27, 7418-7428.	1.7	176
26	Expression mapping of tetracycline-responsive prion protein promoter: Digital atlasing for generating cell-specific disease models. NeuroImage, 2006, 33, 449-462.	2.1	26
27	Autosomal dominant cerebellar ataxias: clinical features, genetics, and pathogenesis. Lancet Neurology, The, 2004, 3, 291-304.	4.9	963
28	Transgenic rat model of Huntington's disease. Human Molecular Genetics, 2003, 12, 617-624.	1.4	329
29	Protein surveillance machinery in brains with spinocerebellar ataxia type 3: Redistribution and differential recruitment of 26S proteasome subunits and chaperones to neuronal intranuclear inclusions. Annals of Neurology, 2002, 51, 302-310.	2.8	133
30	Functional characterization of the human Huntingtonâ€™s disease gene promoter. Molecular Brain Research, 2001, 92, 85-97.	2.5	17
31	An Isoform of Ataxinâ€3 Accumulates in the Nucleus of Neuronal Cells in Affected Brain Regions of SCA3 Patients. Brain Pathology, 1998, 8, 669-679.	2.1	189
32	Isolation and characterization of the rat huntingtin promoter. Biochemical Journal, 1998, 336, 227-234.	1.7	23
33	Model Systems for Spinocerebellar Ataxias: Lessons Learned About the Pathogenesis. , 0, , .		1