Aislinn Williams

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/6290324/publications.pdf Version: 2024-02-01



#	Article	IF	CITATIONS
1	Polyglutamine neurodegeneration: protein misfolding revisited. Trends in Neurosciences, 2008, 31, 521-528.	8.6	325
2	SOD1 mutations disrupt redox-sensitive Rac regulation of NADPH oxidase in a familial ALS model. Journal of Clinical Investigation, 2008, 118, 659-70.	8.2	282
3	The Deubiquitinating Enzyme Ataxin-3, a Polyglutamine Disease Protein, Edits Lys63 Linkages in Mixed Linkage Ubiquitin Chains. Journal of Biological Chemistry, 2008, 283, 26436-26443.	3.4	226
4	CHIP Suppresses Polyglutamine Aggregation and Toxicity <i>In Vitro</i> and <i>In Vivo</i> . Journal of Neuroscience, 2005, 25, 9152-9161.	3.6	217
5	Live-cell imaging reveals divergent intracellular dynamics of polyglutamine disease proteins and supports a sequestration model of pathogenesis. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 9310-9315.	7.1	185
6	Redox modifier genes in amyotrophic lateral sclerosis in mice. Journal of Clinical Investigation, 2007, 117, 2913-2919.	8.2	131
7	The Machado–Joseph disease-associated mutant form of ataxin-3 regulates parkin ubiquitination and stability. Human Molecular Genetics, 2011, 20, 141-154.	2.9	129
8	In vivo suppression of polyglutamine neurotoxicity by C-terminus of Hsp70-interacting protein (CHIP) supports an aggregation model of pathogenesis. Neurobiology of Disease, 2009, 33, 342-353.	4.4	97
9	JosD1, a Membrane-targeted Deubiquitinating Enzyme, Is Activated by Ubiquitination and Regulates Membrane Dynamics, Cell Motility, and Endocytosis. Journal of Biological Chemistry, 2013, 288, 17145-17155.	3.4	63
10	Cohort Profile: The Heinz C. Prechter Longitudinal Study of Bipolar Disorder. International Journal of Epidemiology, 2018, 47, 28-28n.	1.9	58
11	IP3Receptors and Associated Ca2+Signals Localize to Satellite Cells and to Components of the Neuromuscular Junction in Skeletal Muscle. Journal of Neuroscience, 2003, 23, 8185-8192.	3.6	40
12	The best-laid plans go oft awry: synaptogenic growth factor signaling in neuropsychiatric disease. Frontiers in Synaptic Neuroscience, 2014, 6, 4.	2.5	36
13	Deletion of fibroblast growth factor 22 (FGF22) causes a depression-like phenotype in adult mice. Behavioural Brain Research, 2016, 307, 11-17.	2.2	23
14	SCN2A channelopathies in the autism spectrum of neuropsychiatric disorders: a role for pluripotent stem cells?. Molecular Autism, 2020, 11, 23.	4.9	16
15	Risk Factors Associated With Antidepressant Exposure and History of Antidepressant-Induced Mania in Bipolar Disorder. Journal of Clinical Psychiatry, 2018, 79, .	2.2	15
16	Atypical psychotic symptoms and Dandy–Walker variant. Neurocase, 2016, 22, 472-475.	0.6	7
17	Sex differences in the incidence of antidepressant-induced mania (AIM) in bipolar disorders. Neuropsychopharmacology, 2019, 44, 224-225.	5.4	5
18	Deletion of the voltageâ€gated calcium channel, <scp>Ca_V1</scp> .3, causes deficits in motor performance and associative learning. Genes, Brain and Behavior, 2022, 21, e12791.	2.2	5

AISLINN WILLIAMS

#	Article	IF	CITATIONS
19	Putative biological predictors of treatment response in bipolar disorders. Personalized Medicine in Psychiatry, 2017, 1-2, 39-58.	0.1	1
20	Corrigendum to "In vivo suppression of polyglutamine neurotoxicity by C-terminus of Hsp70-interacting protein (CHIP) supports an aggregation model of pathogenesisâ€{Neurobiology of Disease 33/3 (2009) 342–353]. Neurobiology of Disease, 2012, 46, 503.	4.4	0
21	716. Abnormal Calcium Signaling Dynamics in iPSC-Derived Bipolar Disorder Neurons. Biological Psychiatry, 2017, 81, S290.	1.3	0
22	T101. Accelerated Maturation Phenotypes in Patient-Derived Cell Models of Bipolar Disorder. Biological Psychiatry, 2018, 83, S167-S168.	1.3	0