Lachlan A Jolly

List of Publications by Year
in descending order

35 papers	1,679 citations	304743 22 h-index	395702 33 g-index
35 all docs	35 docs citations	35 times ranked	2960 citing authors

#	Article	IF	Citations
1	Vav Proteins in Development of the Brain: A Potential Relationship to the Pathogenesis of Congenital Zika Syndrome?. Viruses, 2022, 14, 386.	3.3	2
2	The DUB Club: Deubiquitinating Enzymes and Neurodevelopmental Disorders. Biological Psychiatry, 2022, 92, 614-625.	1.3	8
3	Abnormal Behavior and Cortical Connectivity Deficits in Mice Lacking Usp9x. Cerebral Cortex, 2021, 31, 1763-1775.	2.9	5
4	Integrated in silico and experimental assessment of disease relevance of PCDH19 Âmissense variants. Human Mutation, 2021, 42, 1030-1041.	2.5	1
5	Impaired neural differentiation of MPS IIIA patient induced pluripotent stem cell-derived neural progenitor cells. Molecular Genetics and Metabolism Reports, 2021, 29, 100811.	1.1	1
6	Partial Loss of USP9X Function Leads to a Male Neurodevelopmental and Behavioral Disorder Converging on Transforming Growth Factor Î² Signaling. Biological Psychiatry, 2020, 87, 100-112.	1.3	42
7	Usp9X Controls Ankyrin-Repeat Domain Protein Homeostasis during Dendritic Spine Development. Neuron, 2020, 105, 506-521.e7.	8.1	34
8	A synonymous UPF3B variant causing a speech disorder implicates NMD as a regulator of neurodevelopmental disorder gene networks. Human Molecular Genetics, 2020, 29, 2568-2578.	2.9	9
9	Missense variant contribution to USP9X-female syndrome. Npj Genomic Medicine, 2020, 5, 53.	3.8	17
10	Inhibition of Upf2-Dependent Nonsense-Mediated Decay Leads to Behavioral and Neurophysiological Abnormalities by Activating the Immune Response. Neuron, 2019, 104, 665-679.e8.	8.1	43
11	A Upf3b-mutant mouse model with behavioral and neurogenesis defects. Molecular Psychiatry, 2018, 23, 1773-1786.	7.9	54
12	PCDH19 regulation of neural progenitor cell differentiation suggests asynchrony of neurogenesis as a mechanism contributing to PCDH19 Girls Clustering Epilepsy. Neurobiology of Disease, 2018, 116, 106-119.	4.4	39
13	Robust imaging and gene delivery to study human lymphoblastoid cell lines. Journal of Human Genetics, 2018, 63, 945-955.	2.3	2
14	USP9X deubiquitylating enzyme maintains RAPTOR protein levels, mTORC1 signalling and proliferation in neural progenitors. Scientific Reports, 2017, 7, 391.	3.3	27
15	Loss of Usp9x disrupts cell adhesion, and components of the Wnt and Notch signaling pathways in neural progenitors. Scientific Reports, 2017, 7, 8109.	3.3	24
16	Viperin is an important host restriction factor in control of Zika virus infection. Scientific Reports, 2017, 7, 4475.	3.3	98
17	Protocadherin Mutations inÂNeurodevelopmental Disorders. , 2016, , 221-231.		1
18	Pcdh19 Loss-of-Function Increases Neuronal Migration In Vitro but is Dispensable for Brain Development in Mice. Scientific Reports, 2016, 6, 26765.	3.3	52

#	Article	IF	Citations
19	De Novo Loss-of-Function Mutations in USP9X Cause a Female-Specific Recognizable Syndrome with Developmental Delay and Congenital Malformations. American Journal of Human Genetics, 2016, 98, 373-381.	6.2	95
20	IncreasedSTAG2dosage defines a novel cohesinopathy with intellectual disability and behavioral problems. Human Molecular Genetics, 2015, 24, 7171-7181.	2.9	28
21	La FAM fatale: USP9X in development and disease. Cellular and Molecular Life Sciences, 2015, 72, 2075-2089.	5.4	145
22	Homozygous mutation of STXBP5L explains an autosomal recessive infantile-onset neurodegenerative disorder. Human Molecular Genetics, 2015, 24, 2000-2010.	2.9	25
23	THOC2 Mutations Implicate mRNA-Export Pathway in X-Linked Intellectual Disability. American Journal of Human Genetics, 2015, 97, 302-310.	6.2	82
24	Seizures Are Regulated by Ubiquitin-specific Peptidase 9 X-linked (USP9X), a De-Ubiquitinase. PLoS Genetics, 2015, 11, e1005022.	3.5	66
25	HCFC1 loss-of-function mutations disrupt neuronal and neural progenitor cells of the developing brain. Human Molecular Genetics, 2015, 24, 3335-3347.	2.9	47
26	Mutations in USP9X Are Associated with X-Linked Intellectual Disability and Disrupt Neuronal Cell Migration and Growth. American Journal of Human Genetics, 2014, 94, 470-478.	6.2	117
27	UPF3B Gene and Nonsense-Mediated mRNA Decay in Autism Spectrum Disorders. , 2014, , 1663-1678.		3
28	Loss of FMR2 further emphasizes the link between deregulation of immediate early response genes FOS and JUN and intellectual disability. Human Molecular Genetics, 2013, 22, 2984-2991.	2.9	10
29	The UPF3B gene, implicated in intellectual disability, autism, ADHD and childhood onset schizophrenia regulates neural progenitor cell behaviour and neuronal outgrowth. Human Molecular Genetics, 2013, 22, 4673-4687.	2.9	101
30	Loss of Usp9x Disrupts Cortical Architecture, Hippocampal Development and TGFÎ²-Mediated Axonogenesis. PLoS ONE, 2013, 8, e68287.	2.5	77
31	A Noncoding, Regulatory Mutation Implicates HCFC1 in Nonsyndromic Intellectual Disability. American Journal of Human Genetics, 2012, 91, 694-702.	6.2	89
32	Transcriptome profiling of UPF3B/NMD-deficient lymphoblastoid cells from patients with various forms of intellectual disability. Molecular Psychiatry, 2012, 17, 1103-1115.	7.9	97
33	A Focal Epilepsy and Intellectual Disability Syndrome Is Due to a Mutation in TBC1D24. American Journal of Human Genetics, 2010, 87, 371-375.	6.2	111
34	USP9X Enhances the Polarity and Self-Renewal of Embryonic Stem Cell-derived Neural Progenitors. Molecular Biology of the Cell, 2009, 20, 2015-2029.	2.1	52
35	The FAM Deubiquitylating Enzyme Localizes to Multiple Points of Protein Trafficking in Epithelia, where It Associates with E-cadherin and Î²-catenin. Molecular Biology of the Cell, 2004, 15, 1591-1599.	2.1	75