

Lyndsay M Murray

List of Publications by Year in descending order

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Version: 2024-02-01

25
papers

1,505
citations

471477

17
h-index

580810

25
g-index

25
all docs

25
docs citations

25
times ranked

1451
citing authors

#	ARTICLE	IF	CITATIONS
1	Selective vulnerability of motor neurons and dissociation of pre- and post-synaptic pathology at the neuromuscular junction in mouse models of spinal muscular atrophy. <i>Human Molecular Genetics</i> , 2008, 17, 949-962.	2.9	333
2	Alternative Splicing Events Are a Late Feature of Pathology in a Mouse Model of Spinal Muscular Atrophy. <i>PLoS Genetics</i> , 2009, 5, e1000773.	3.5	210
3	A critical smn threshold in mice dictates onset of an intermediate spinal muscular atrophy phenotype associated with a distinct neuromuscular junction pathology. <i>Neuromuscular Disorders</i> , 2012, 22, 263-276.	0.6	116
4	A novel function for the survival motoneuron protein as a translational regulator. <i>Human Molecular Genetics</i> , 2013, 22, 668-684.	2.9	106
5	Pre-symptomatic development of lower motor neuron connectivity in a mouse model of severe spinal muscular atrophy. <i>Human Molecular Genetics</i> , 2010, 19, 420-433.	2.9	98
6	Reversible molecular pathology of skeletal muscle in spinal muscular atrophy. <i>Human Molecular Genetics</i> , 2011, 20, 4334-4344.	2.9	89
7	Transcriptional profiling of differentially vulnerable motor neurons at pre-symptomatic stage in the Smn 2b ⁻ mouse model of spinal muscular atrophy. <i>Acta Neuropathologica Communications</i> , 2015, 3, 55.	5.2	61
8	Defects in neuromuscular junction remodelling in the Smn2B ⁺ mouse model of spinal muscular atrophy. <i>Neurobiology of Disease</i> , 2013, 49, 57-67.	4.4	59
9	Myogenic program dysregulation is contributory to disease pathogenesis in spinal muscular atrophy. <i>Human Molecular Genetics</i> , 2014, 23, 4249-4259.	2.9	59
10	Defects in pancreatic development and glucose metabolism in SMN-depleted mice independent of canonical spinal muscular atrophy neuromuscular pathology. <i>Human Molecular Genetics</i> , 2014, 23, 3432-3444.	2.9	55
11	Temporal and tissue-specific variability of SMN protein levels in mouse models of spinal muscular atrophy. <i>Human Molecular Genetics</i> , 2018, 27, 2851-2862.	2.9	55
12	PLAA Mutations Cause a Lethal Infantile Epileptic Encephalopathy by Disrupting Ubiquitin-Mediated Endolysosomal Degradation of Synaptic Proteins. <i>American Journal of Human Genetics</i> , 2017, 100, 706-724.	6.2	37
13	Interventions Targeting Glucocorticoid-KrÄ¼ppel-like Factor 15-Branched-Chain Amino Acid Signaling Improve Disease Phenotypes in Spinal Muscular Atrophy Mice. <i>EBioMedicine</i> , 2018, 31, 226-242.	6.1	37
14	Comparison of independent screens on differentially vulnerable motor neurons reveals alpha-synuclein as a common modifier in motor neuron diseases. <i>PLoS Genetics</i> , 2017, 13, e1006680.	3.5	36
15	Loss of translation elongation factor (<i>eEF1A2</i>) expression <i>in vivo</i> differentiates between Wallerian degeneration and dying-back neuronal pathology. <i>Journal of Anatomy</i> , 2008, 213, 633-645.	1.5	28
16	Pathogenic commonalities between spinal muscular atrophy and amyotrophic lateral sclerosis: Converging roads to therapeutic development. <i>European Journal of Medical Genetics</i> , 2018, 61, 685-698.	1.3	28
17	Reduced P53 levels ameliorate neuromuscular junction loss without affecting motor neuron pathology in a mouse model of spinal muscular atrophy. <i>Cell Death and Disease</i> , 2019, 10, 515.	6.3	21
18	The Smn-Independent Beneficial Effects of Trichostatin A on an Intermediate Mouse Model of Spinal Muscular Atrophy. <i>PLoS ONE</i> , 2014, 9, e101225.	2.5	21

#	ARTICLE	IF	CITATIONS
19	Dissection of the &Transversus Abdominis&/em' Muscle for Whole-mount Neuromuscular Junction Analysis. <i>Journal of Visualized Experiments</i> , 2014, , e51162.	0.3	17
20	Altered mitochondrial bioenergetics are responsible for the delay in Wallerian degeneration observed in neonatal mice. <i>Neurobiology of Disease</i> , 2019, 130, 104496.	4.4	15
21	The response of neuromuscular junctions to injury is developmentally regulated. <i>FASEB Journal</i> , 2011, 25, 1306-1313.	0.5	10
22	Synaptic withdrawal following nerve injury is influenced by postnatal maturity, muscleâ“s specific properties, and the presence of underlying pathology in mice. <i>Journal of Anatomy</i> , 2020, 237, 263-274.	1.5	4
23	Motor unit recovery following Smn restoration in mouse models of spinal muscular atrophy. <i>Human Molecular Genetics</i> , 2022, 31, 3107-3119.	2.9	4
24	A reduction in the human adenovirus virion size through use of a shortened fibre protein does not enhance muscle transduction following systemic or localised delivery in mice. <i>Virology</i> , 2014, 468-470, 444-453.	2.4	3
25	How far away is spinal muscular atrophy gene therapy?. <i>Expert Review of Neurotherapeutics</i> , 2015, 15, 965-968.	2.8	3