

Lyndsay M Murray

List of Publications by Year in Descending Order

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The third column is the impact factor (IF) of the journal, and the fourth column is the number of citations of the article.

23
papers

1,178
citations

17
h-index

25
g-index

25
ext. papers

1,391
ext. citations

5.1
avg. IF

3.92
L-index

#	Paper	IF	Citations
23	Synaptic withdrawal following nerve injury is influenced by postnatal maturity, muscle-specific properties, and the presence of underlying pathology in mice. <i>Journal of Anatomy</i> , 2020 , 237, 263-274	2.9	1
22	Altered mitochondrial bioenergetics are responsible for the delay in Wallerian degeneration observed in neonatal mice. <i>Neurobiology of Disease</i> , 2019 , 130, 104496	7.5	9
21	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	9.8	11
20	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	2.6	17
19	Interventions Targeting Glucocorticoid-Krüppel-like Factor 15-Branched-Chain Amino Acid Signaling Improve Disease Phenotypes in Spinal Muscular Atrophy Mice. <i>EBioMedicine</i> , 2018 , 31, 226-242	8.8	24
18	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	5.6	35
17	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	11	22
16	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	6	23
15	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	7.3	42
14	Myogenic program dysregulation is contributory to disease pathogenesis in spinal muscular atrophy. <i>Human Molecular Genetics</i> , 2014 , 23, 4249-59	5.6	45
13	Dissection of the transversus abdominis muscle for whole-mount neuromuscular junction analysis. <i>Journal of Visualized Experiments</i> , 2014 , e51162	1.6	11
12	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	3.6	3
11	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, 3432-44	5.6	44
10	The Smn-independent beneficial effects of trichostatin A on an intermediate mouse model of spinal muscular atrophy. <i>PLoS ONE</i> , 2014 , 9, e101225	3.7	17
9	Defects in neuromuscular junction remodelling in the Smn(2B/-) mouse model of spinal muscular atrophy. <i>Neurobiology of Disease</i> , 2013 , 49, 57-67	7.5	49
8	A novel function for the survival motoneuron protein as a translational regulator. <i>Human Molecular Genetics</i> , 2013 , 22, 668-84	5.6	77
7	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, 263-76	2.9	89

6	Reversible molecular pathology of skeletal muscle in spinal muscular atrophy. <i>Human Molecular Genetics</i> , 2011 , 20, 4334-44	5.6	75
5	The response of neuromuscular junctions to injury is developmentally regulated. <i>FASEB Journal</i> , 2011 , 25, 1306-13	0.9	6
4	Pre-symptomatic development of lower motor neuron connectivity in a mouse model of severe spinal muscular atrophy. <i>Human Molecular Genetics</i> , 2010 , 19, 420-33	5.6	87
3	Alternative splicing events are a late feature of pathology in a mouse model of spinal muscular atrophy. <i>PLoS Genetics</i> , 2009 , 5, e1000773	6	185
2	Loss of translation elongation factor (eEF1A2) expression in vivo differentiates between Wallerian degeneration and dying-back neuronal pathology. <i>Journal of Anatomy</i> , 2008 , 213, 633-45	2.9	25
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, 949-62	5.6	279