Lyndsay M Murray

List of Publications by Citations

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	Selective vulnerability of motor neurons and dissociation of pre- and post-synaptic pathology at the neuromuscular junction in mouse models of spinal muscular atrophy. *Human Molecular Genetics*, **2008**, 17, 949-62	5.6	279
22	Alternative splicing events are a late feature of pathology in a mouse model of spinal muscular atrophy. *PLoS Genetics*, **2009**, 5, e1000773	6	185
21	A critical smn threshold in mice dictates onset of an intermediate spinal muscular atrophy phenotype associated with a distinct neuromuscular junction pathology. *Neuromuscular Disorders*, **2012**, 22, 263-76	2.9	89
20	Pre-symptomatic development of lower motor neuron connectivity in a mouse model of severe spinal muscular atrophy. *Human Molecular Genetics*, **2010**, 19, 420-33	5.6	87
19	A novel function for the survival motoneuron protein as a translational regulator. *Human Molecular Genetics*, **2013**, 22, 668-84	5.6	77
18	Reversible molecular pathology of skeletal muscle in spinal muscular atrophy. *Human Molecular Genetics*, **2011**, 20, 4334-44	5.6	75
17	Defects in neuromuscular junction remodelling in the Smn(2B/-) mouse model of spinal muscular atrophy. *Neurobiology of Disease*, **2013**, 49, 57-67	7.5	49
16	Myogenic program dysregulation is contributory to disease pathogenesis in spinal muscular atrophy. *Human Molecular Genetics*, **2014**, 23, 4249-59	5.6	45
15	Defects in pancreatic development and glucose metabolism in SMN-depleted mice independent of canonical spinal muscular atrophy neuromuscular pathology. *Human Molecular Genetics*, **2014**, 23, 3432-44	5.6	44
14	Transcriptional profiling of differentially vulnerable motor neurons at pre-symptomatic stage in the Smn (2b/-) mouse model of spinal muscular atrophy. *Acta Neuropathologica Communications*, **2015**, 3, 55	7.3	42
13	Temporal and tissue-specific variability of SMN protein levels in mouse models of spinal muscular atrophy. *Human Molecular Genetics*, **2018**, 27, 2851-2862	5.6	35
12	Loss of translation elongation factor (eEF1A2) expression in vivo differentiates between Wallerian degeneration and dying-back neuronal pathology. *Journal of Anatomy*, **2008**, 213, 633-45	2.9	25
11	Interventions Targeting Glucocorticoid-Krüppel-like Factor 15-Branched-Chain Amino Acid Signaling Improve Disease Phenotypes in Spinal Muscular Atrophy Mice. *EBioMedicine*, **2018**, 31, 226-242	8.8	24
10	Comparison of independent screens on differentially vulnerable motor neurons reveals alpha-synuclein as a common modifier in motor neuron diseases. *PLoS Genetics*, **2017**, 13, e1006680	6	23
9	PLAA Mutations Cause a Lethal Infantile Epileptic Encephalopathy by Disrupting Ubiquitin-Mediated Endolysosomal Degradation of Synaptic Proteins. *American Journal of Human Genetics*, **2017**, 100, 706-724	11	22
8	Pathogenic commonalities between spinal muscular atrophy and amyotrophic lateral sclerosis: Converging roads to therapeutic development. *European Journal of Medical Genetics*, **2018**, 61, 685-698	2.6	17
7	The Smn-independent beneficial effects of trichostatin A on an intermediate mouse model of spinal muscular atrophy. *PLoS ONE*, **2014**, 9, e101225	3.7	17

6	Reduced P53 levels ameliorate neuromuscular junction loss without affecting motor neuron pathology in a mouse model of spinal muscular atrophy. *Cell Death and Disease*, **2019**, 10, 515	9.8	11
5	Dissection of the transversus abdominis muscle for whole-mount neuromuscular junction analysis. *Journal of Visualized Experiments*, **2014**, e51162	1.6	11
4	Altered mitochondrial bioenergetics are responsible for the delay in Wallerian degeneration observed in neonatal mice. *Neurobiology of Disease*, **2019**, 130, 104496	7.5	9
3	The response of neuromuscular junctions to injury is developmentally regulated. *FASEB Journal*, **2011**, 25, 1306-13	0.9	6
2	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. *Virology*, **2014**, 468-470, 444-453	3.6	3
1	Synaptic withdrawal following nerve injury is influenced by postnatal maturity, muscle-specific properties, and the presence of underlying pathology in mice. *Journal of Anatomy*, **2020**, 237, 263-274	2.9	1