Matthew D Howell

List of Publications by Year
in descending order

Source: https://exaly.com/author-pdf/7825635/publications.pdf

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18	920	471371 17	839398 18
papers	citations	h-index	g-index

18	18	18	1369
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Diverse role of survival motor neuron protein. Biochimica Et Biophysica Acta - Gene Regulatory Mechanisms, 2017, 1860, 299-315.	0.9	210
2	Canonical Transient Receptor Channel 5 (TRPC5) and TRPC1/4 Contribute to Seizure and Excitotoxicity by Distinct Cellular Mechanisms. Molecular Pharmacology, 2013, 83, 429-438.	1.0	113
3	ADAMTS expression and function in central nervous system injury and disorders. Matrix Biology, 2015, 44-46, 70-76.	1.5	73
4	Spinal muscular atrophy: An update on therapeutic progress. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2013, 1832, 2180-2190.	1.8	62
5	Antisense oligonucleotide mediated therapy of spinal muscular atrophy. Translational Neuroscience, 2013, 4, 1-7.	0.7	53
6	Lectican proteoglycans, their cleaving metalloproteinases, and plasticity in the central nervous system extracellular microenvironment. Neuroscience, 2012, 217, 6-18.	1.1	52
7	Severe impairment of male reproductive organ development in a low SMN expressing mouse model of spinal muscular atrophy. Scientific Reports, 2016, 6, 20193.	1.6	50
8	A Short Antisense Oligonucleotide Ameliorates Symptoms of Severe Mouse Models of Spinal Muscular Atrophy. Molecular Therapy - Nucleic Acids, 2014, 3, e174.	2.3	47
9	Advances in therapeutic development for spinal muscular atrophy. Future Medicinal Chemistry, 2014, 6, 1081-1099.	1.1	43
10	Hippocampal administration of chondroitinase ABC increases plaque-adjacent synaptic marker and diminishes amyloid burden in aged APPswe/PS1dE9 mice. Acta Neuropathologica Communications, 2015, 3, 54.	2.4	38
11	Abnormal postâ€translational and extracellular processing of brevican in plaqueâ€bearing mice overâ€expressing APPsw. Journal of Neurochemistry, 2010, 113, 784-795.	2.1	33
12	Gender-Specific Amelioration of SMA Phenotype upon Disruption of a Deep Intronic Structure by an Oligonucleotide. Molecular Therapy, 2017, 25, 1328-1341.	3.7	25
13	Activation of a cryptic 5â€² splice site reverses the impact of pathogenic splice site mutations in the spinal muscular atrophy gene. Nucleic Acids Research, 2017, 45, 12214-12240.	6.5	25
14	TIA1 is a gender-specific disease modifier of a mild mouse model of spinal muscular atrophy. Scientific Reports, 2017, 7, 7183.	1.6	23
15	Panel of synaptic protein ELISAs for evaluating neurological phenotype. Experimental Brain Research, 2010, 201, 885-893.	0.7	22
16	Advances in therapeutic development for spinal muscular atrophy. Future Medicinal Chemistry, 2014, 6, 1081-1099.	1.1	21
17	Selective Decline of Synaptic Protein Levels in the Frontal Cortex of Female Mice Deficient in the Extracellular Metalloproteinase ADAMTS1. PLoS ONE, 2012, 7, e47226.	1.1	20
18	Altered Synaptic Marker Abundance in the Hippocampal Stratum Oriens of Ts65Dn Mice is Associated with Exuberant Expression of Versican. ASN Neuro, 2012, 4, AN20110037.	1.5	10