Aymeric Ravel-Chapuis

List of Publications by Year
in descending order

26	715	623734	552781
papers	citations	14 h-index	26 g-index

29	29	29	928
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	MUSK, a new target for mutations causing congenital myasthenic syndrome. Human Molecular Genetics, 2004, 13, 3229-3240.	2.9	175
2	The RNA-binding protein Staufen1 is increased in DM1 skeletal muscle and promotes alternative pre-mRNA splicing. Journal of Cell Biology, 2012, 196, 699-712.	5.2	104
3	Postsynaptic chromatin is under neural control at the neuromuscular junction. EMBO Journal, 2007, 26, 1117-1128.	7.8	40
4	Expression of mutant Ets protein at the neuromuscular synapse causes alterations in morphology and gene expression. EMBO Reports, 2002, 3, 1075-1081.	4.5	37
5	Staufen1 Regulates Multiple Alternative Splicing Events either Positively or Negatively in DM1 Indicating Its Role as a Disease Modifier. PLoS Genetics, 2016, 12, e1005827.	3.5	37
6	Synapse-Specific Gene Expression at the Neuromuscular Junction. Annals of the New York Academy of Sciences, 2003, 998, 53-65.	3.8	32
7	HDAC6 regulates microtubule stability and clustering of AChRs at neuromuscular junctions. Journal of Cell Biology, 2020, 219, .	5.2	32
8	The RNA-binding protein Staufen1 impairs myogenic differentiation via a c-mycâ€“dependent mechanism. Molecular Biology of the Cell, 2014, 25, 3765-3778.	2.1	30
9	Staufen1 impairs stress granule formation in skeletal muscle cells from myotonic dystrophy type 1 patients. Molecular Biology of the Cell, 2016, 27, 1728-1739.	2.1	30
10	Misregulation of calcium-handling proteins promotes hyperactivation of calcineurinâ€“NFAT signaling in skeletal muscle of DM1 mice. Human Molecular Genetics, 2017, 26, 2192-2206.	2.9	27
11	Pharmacological and physiological activation of AMPK improves the spliceopathy in DM1 mouse muscles. Human Molecular Genetics, 2018, 27, 3361-3376.	2.9	24
12	Maintenance of CCL5 mRNA stores by post-effector and memory CD8 T cells is dependent on transcription and is coupled to increased mRNA stability. European Journal of Immunology, 2006, 36, 2745-2754.	2.9	21
13	PAK1 and CtBP1 Regulate the Coupling of Neuronal Activity to Muscle Chromatin and Gene Expression. Molecular and Cellular Biology, 2015, 35, 4110-4120.	2.3	21
14	Muscle-specific expression of the RNA-binding protein Staufen1 induces progressive skeletal muscle atrophy via regulation of phosphatase tensin homolog. Human Molecular Genetics, 2017, 26, 1821-1838.	2.9	21
15	Novel Roles for Staufen1 in Embryonal and Alveolar Rhabdomyosarcoma via c-myc-dependent and -independent events. Scientific Reports, 2017, 7, 42342.	3.3	14
16	Thrombin downregulates muscle acetylcholine receptors via an IP3 signaling pathway by activating its G-protein-coupled protease-activated receptor-1. Journal of Cellular Physiology, 2003, 196, 105-112.	4.1	11
17	Expression of Pannexin 1 and Pannexin 3 during skeletal muscle development, regeneration, and Duchenne muscular dystrophy. Journal of Cellular Physiology, 2018, 233, 7057-7070.	4.1	11
18	Overexpression of Staufen1 in DM1 mouse skeletal muscle exacerbates dystrophic and atrophic features. Human Molecular Genetics, 2020, 29, 2185-2199.	2.9	8

#	Article	IF	Citations
19	Thrombin reduces MuSK and acetylcholine receptor expression along with neuromuscular contact size in vitro. European Journal of Neuroscience, 2004, 19, 2099-2108.	2.6	7
20	Staufen1s role as a splicing factor and a disease modifier in Myotonic Dystrophy Type I. Rare Diseases (Austin, Tex), 2016, 4, e1225644.	1.8	7
21	Differential regulation of autophagy by STAU1 in alveolar rhabdomyosarcoma and nonâ€transformed skeletal muscle cells. Cellular Oncology (Dordrecht), 2021, 44, 851-870.	4.4	7
22	Pharmacological and exerciseâ€induced activation of AMPK as emerging therapies for myotonic dystrophy type 1 patients. Journal of Physiology, 2022, 600, 3249-3264.	2.9	5
23	Combinatorial therapies for rescuing myotonic dystrophy type 1 skeletal muscle defects. Trends in Molecular Medicine, 2022, , .	6.7	4
24	A novel CARM1â€“HuR axis involved in muscle differentiation and plasticity misregulated in spinal muscular atrophy. Human Molecular Genetics, 2022, 31, 1453-1470.	2.9	2
25	AChR Î²-Subunit mRNAs Are Stabilized by HuR in a Mouse Model of Congenital Myasthenic Syndrome With Acetylcholinesterase Deficiency. Frontiers in Molecular Neuroscience, 2020, 13, 568171.	2.9	1
26	Mutations in MUSK cause congenital myasthenic syndrome. Journal of Physiology (Paris), 2006, 99, 2-3.	2.1	0