

Anne Fourest-Lieuvin

List of Publications by Year in descending order

Source: <https://exaly.com/author-pdf/7815522/publications.pdf>

Version: 2024-02-01

22
papers

1,115
citations

623734

14
h-index

839539

18
g-index

22
all docs

22
docs citations

22
times ranked

1714
citing authors

#	ARTICLE	IF	CITATIONS
1	Tau can switch microtubule network organizations: from random networks to dynamic and stable bundles. <i>Molecular Biology of the Cell</i> , 2018, 29, 154-165.	2.1	49
2	Deletion of the microtubule-associated protein 6 (MAP6) results in skeletal muscle dysfunction. <i>Skeletal Muscle</i> , 2018, 8, 30.	4.2	21
3	A TIRF microscopy assay to decode how tau regulates EB™s tracking at microtubule ends. <i>Methods in Cell Biology</i> , 2017, 141, 179-197.	1.1	14
4	The Microtubule-Associated Protein CLIMP-63 is a New Member of the Calcium Release Complex. <i>Biophysical Journal</i> , 2016, 110, 181a-182a.	0.5	0
5	Triadin and CLIMP-63 form a link between triads and microtubules in muscle cells. <i>Journal of Cell Science</i> , 2016, 129, 3744-3755.	2.0	37
6	Tau antagonizes end-binding protein tracking at microtubule ends through a phosphorylation-dependent mechanism. <i>Molecular Biology of the Cell</i> , 2016, 27, 2924-2934.	2.1	60
7	Tau co-organizes dynamic microtubule and actin networks. <i>Scientific Reports</i> , 2015, 5, 9964.	3.3	149
8	Exon Skipping as a Therapeutic Strategy Applied to a RyR1 Mutation Causing Severe Core Myopathy. <i>Biophysical Journal</i> , 2013, 104, 203a.	0.5	0
9	Exon Skipping as a Therapeutic Strategy Applied to an RYR1 Mutation with Pseudo-Exon Inclusion Causing a Severe Core Myopathy. <i>Human Gene Therapy</i> , 2013, 24, 702-713.	2.7	27
10	Role of Triadin in the Organization of Reticulum Membrane at the Muscle Triad. <i>Journal of Cell Science</i> , 2012, 125, 3443-53.	2.0	20
11	Role of Triadin in the Organization of Reticulum Membrane at the Muscle Triad. <i>Biophysical Journal</i> , 2012, 102, 363a.	0.5	0
12	Identification of the First Mutations in the Human Triadin Gene, Associated to Catecholaminergic Tachycardia, a Pathology of the Cardiac Calcium Release Complex. <i>Biophysical Journal</i> , 2012, 102, 408a-409a.	0.5	0
13	Absence of triadin, a protein of the calcium release complex, is responsible for cardiac arrhythmia with sudden death in human. <i>Human Molecular Genetics</i> , 2012, 21, 2759-2767.	2.9	227
14	Mutation of Ser172 in Yeast β Tubulin Induces Defects in Microtubule Dynamics and Cell Division. <i>PLoS ONE</i> , 2010, 5, e13553.	2.5	16
15	Triadin: what possible function 20 years later?. <i>Journal of Physiology</i> , 2009, 587, 3117-3121.	2.9	36
16	Triadin Function In Sarcoplasmic Reticulum Structure?. <i>Biophysical Journal</i> , 2009, 96, 237a.	0.5	1
17	Purification of tubulin from limited volumes of cultured cells. <i>Protein Expression and Purification</i> , 2006, 45, 183-190.	1.3	10
18	Microtubule Regulation in Mitosis: Tubulin Phosphorylation by the Cyclin-dependent Kinase Cdk1. <i>Molecular Biology of the Cell</i> , 2006, 17, 1041-1050.	2.1	160

#	ARTICLE	IF	CITATIONS
19	Phosphorylation of Microtubule-associated Protein STOP by Calmodulin Kinase II. Journal of Biological Chemistry, 2006, 281, 19561-19569.	3.4	47
20	Suppression of nuclear oscillations in <i>Saccharomyces cerevisiae</i> expressing Glu tubulin. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 5577-5582.	7.1	73
21	STOP Proteins are Responsible for the High Degree of Microtubule Stabilization Observed in Neuronal Cells. Journal of Cell Biology, 1998, 142, 167-179.	5.2	111
22	Nonneuronal isoforms of STOP protein are responsible for microtubule cold stability in mammalian fibroblasts. Proceedings of the National Academy of Sciences of the United States of America, 1998, 95, 6055-6060.	7.1	57