

Francesca Maltecca

List of Publications by Year in descending order

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Version: 2024-02-01

12
papers

498
citations

933264

10
h-index

1199470

12
g-index

13
all docs

13
docs citations

13
times ranked

864
citing authors

#	ARTICLE	IF	CITATIONS
1	Haploinsufficiency of <i>AFG3L2</i> , the Gene Responsible for Spinocerebellar Ataxia Type 28, Causes Mitochondria-Mediated Purkinje Cell Dark Degeneration. <i>Journal of Neuroscience</i> , 2009, 29, 9244-9254.	1.7	99
2	The Mitochondrial Protease AFG3L2 Is Essential for Axonal Development. <i>Journal of Neuroscience</i> , 2008, 28, 2827-2836.	1.7	92
3	Purkinje neuron Ca ²⁺ influx reduction rescues ataxia in SCA28 model. <i>Journal of Clinical Investigation</i> , 2015, 125, 263-274.	3.9	67
4	Respiratory dysfunction by AFG3L2 deficiency causes decreased mitochondrial calcium uptake via organellar network fragmentation. <i>Human Molecular Genetics</i> , 2012, 21, 3858-3870.	1.4	49
5	Altered organization of the intermediate filament cytoskeleton and relocalization of proteostasis modulators in cells lacking the ataxia protein saccin. <i>Human Molecular Genetics</i> , 2017, 26, 3130-3143.	1.4	46
6	<i>m</i> -AAA and <i>i</i> -AAA complexes work coordinately regulating OMA1, the stress-activated supervisor of mitochondrial dynamics. <i>Journal of Cell Science</i> , 2018, 131, .	1.2	40
7	Impaired turnover of hyperfused mitochondria in severe axonal neuropathy due to a novel DRP1 mutation. <i>Human Molecular Genetics</i> , 2020, 29, 177-188.	1.4	30
8	Mice harbouring a SCA28 patient mutation in AFG3L2 develop late-onset ataxia associated with enhanced mitochondrial proteotoxicity. <i>Neurobiology of Disease</i> , 2019, 124, 14-28.	2.1	23
9	Pathogenic variants in the AFG3L2 proteolytic domain cause SCA28 through haploinsufficiency and proteostatic stress-driven OMA1 activation. <i>Journal of Medical Genetics</i> , 2019, 56, 499-511.	1.5	20
10	The WRB Subunit of the Get3 Receptor is Required for the Correct Integration of its Partner CAML into the ER. <i>Scientific Reports</i> , 2019, 9, 11887.	1.6	16
11	A novel AFG3L2 mutation close to AAA domain leads to aberrant OMA1 and OPA1 processing in a family with optic atrophy. <i>Acta Neuropathologica Communications</i> , 2020, 8, 93.	2.4	10
12	Upregulation of Peroxiredoxin 3 Protects <i>Afg3l2</i> -KO Cortical Neurons <i>In Vitro</i> from Oxidative Stress: A Paradigm for Neuronal Cell Survival under Neurodegenerative Conditions. <i>Oxidative Medicine and Cellular Longevity</i> , 2019, 2019, 1-13.	1.9	6