Quasar S Padiath

List of Publications by Year
in descending order

14 papers	695 citations	1040056 9 h-index	1058476 14 g-index
15 all docs	15 docs citations	15 times ranked	1094 citing authors

#	Article	IF	Citations
1	Lamin B1 duplications cause autosomal dominant leukodystrophy. Nature Genetics, 2006, 38, 1114-1123.	21.4	365
2	Concentric organization of A- and B-type lamins predicts their distinct roles in the spatial organization and stability of the nuclear lamina. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 4307-4315.	7.1	98
3	TUBB4A mutations result in specific neuronal and oligodendrocytic defects that closely match clinically distinct phenotypes. Human Molecular Genetics, 2017, 26, 4506-4518.	2.9	59
4	Defects of Lipid Synthesis Are Linked to the Age-Dependent Demyelination Caused by Lamin B1 Overexpression. Journal of Neuroscience, 2015, 35, 12002-12017.	3.6	51
5	CAPOS syndrome and hemiplegic migraine in a novel pedigree with the specific ATP1A3 mutation. Journal of the Neurological Sciences, 2015, 358, 453-456.	0.6	23
6	Autosomal Dominant Leukodystrophy: A Disease of the Nuclear Lamina. Frontiers in Cell and Developmental Biology, 2019, 7, 41.	3.7	21
7	Genomic deletions upstream of lamin B1 lead to atypical autosomal dominant leukodystrophy. Neurology: Genetics, 2019, 5, e305.	1.9	16
8	TUBB4A mutations result in both glial and neuronal degeneration in an H-ABC leukodystrophy mouse model. ELife, 2020, 9, .	6.0	15
9	Lamin B1 mediated demyelination: Linking Lamins, Lipids and Leukodystrophies. Nucleus, 2016, 7, 547-553.	2.2	14
10	An atypical form of AOA2 with myoclonus associated with mutations in SETX and AFG3L2. BMC Medical Genetics, 2015, 16, 16.	2.1	12
11	Mice overexpressing lamin B1 in oligodendrocytes recapitulate the age-dependent motor signs, but not the early autonomic cardiovascular dysfunction of autosomal-dominant leukodystrophy (ADLD). Experimental Neurology, 2018, 301, 1-12.	4.1	11
12	scMAPA: Identification of cell-typeâ€“specific alternative polyadenylation in complex tissues. GigaScience, 2022, 11, .	6.4	4
13	Development and Optimization of a High-Content Analysis Platform to Identify Suppressors of Lamin B1 Overexpression as a Therapeutic Strategy for Autosomal Dominant Leukodystrophy. SLAS Discovery, 2020, 25, 939-949.	2.7	3
14	Deletion of conserved nonâ€coding sequences downstream from NKX2â€1: A novel diseaseâ€causing mechanism for benign hereditary chorea. Molecular Genetics & Genomic Medicine, 2021, 9, e1647.	1.2	3