Oscar D Bello

List of Publications by Year
in descending order

Source: https://exaly.com/author-pdf/335806/publications.pdf

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22	1,029	471509 17	713466 21
papers	citations	h-index	g-index

24	24	24	1681
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Synaptotagmin-1 membrane binding is driven by the C2B domain and assisted cooperatively by the C2A domain. Scientific Reports, 2020, 10, 18011.	3.3	22
2	Synaptotagmin 1 oligomers clamp and regulate different modes of neurotransmitter release. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 3819-3827.	7.1	47
3	AMPA receptor GluA2 subunit defects are a cause of neurodevelopmental disorders. Nature Communications, 2019, 10, 3094.	12.8	150
4	PDXK mutations cause polyneuropathy responsive to pyridoxal 5â€²â€phosphate supplementation. Annals of Neurology, 2019, 86, 225-240.	5.3	54
5	Mutations in the Neuronal Vesicular SNARE VAMP2 Affect Synaptic Membrane Fusion and Impair Human Neurodevelopment. American Journal of Human Genetics, 2019, 104, 721-730.	6.2	88
6	Rearrangements under confinement lead to increased binding energy of Synaptotagminâ€1 with anionic membranes in Mg 2+ and Ca 2+. FEBS Letters, 2018, 592, 1497-1506.	2.8	13
7	A loss-of-function homozygous mutation in DDX59 implicates a conserved DEAD-box RNA helicase in nervous system development and function. Human Mutation, 2018, 39, 187-192.	2.5	44
8	Synaptotagmin oligomerization is essential for calcium control of regulated exocytosis. Proceedings of the National Academy of Sciences of the United States of America, 2018, 115, E7624-E7631.	7.1	51
9	Homozygous mutations in <scp>VAMP</scp>1 cause a presynaptic congenital myasthenic syndrome. Annals of Neurology, 2017, 81, 597-603.	5.3	48
10	Kv1.1 channelopathy abolishes presynaptic spike width modulation by subthreshold somatic depolarization. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, 2395-2400.	7.1	31
11	Mutations in NKX6-2 Cause Progressive Spastic Ataxia and Hypomyelination. American Journal of Human Genetics, 2017, 100, 969-977.	6.2	38
12	Clinical, pathological and functional characterization of riboflavin-responsive neuropathy. Brain, 2017, 140, 2820-2837.	7.6	64
13	Circular oligomerization is an intrinsic property of synaptotagmin. ELife, 2017, 6, .	6.0	47
14	Dilation of fusion pores by crowding of SNARE proteins. ELife, 2017, 6, .	6.0	57
15	Nanodisc-cell fusion: control of fusion pore nucleation and lifetimes by SNARE protein transmembrane domains. Scientific Reports, 2016, 6, 27287.	3.3	39
16	Rab3A, a possible marker of cortical granules, participates in cortical granule exocytosis in mouse eggs. Experimental Cell Research, 2016, 347, 42-51.	2.6	16
17	Using ApoE Nanolipoprotein Particles To Analyze SNARE-Induced Fusion Pores. Langmuir, 2016, 32, 3015-3023.	3.5	22
18	Ring-like oligomers of Synaptotagmins and related C2 domain proteins. ELife, 2016, 5, .	6.0	57

#	Article	IF	Citations
19	Control of Fusion Pore Nucleation and Dynamics by SNARE Protein Transmembrane Domains. Biophysical Journal, 2015, 108, 408a.	0.5	0
20	Cortical Granule Exocytosis Is Mediated by Alpha-SNAP and N-Ethilmaleimide Sensitive Factor in Mouse Oocytes. PLoS ONE, 2015, 10, e0135679.	2.5	19
21	Calcium sensitive ring-like oligomers formed by synaptotagmin. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, 13966-13971.	7.1	76
22	RIM, Munc13, and Rab3A interplay in acrosomal exocytosis. Experimental Cell Research, 2012, 318, 478-488.	2.6	43