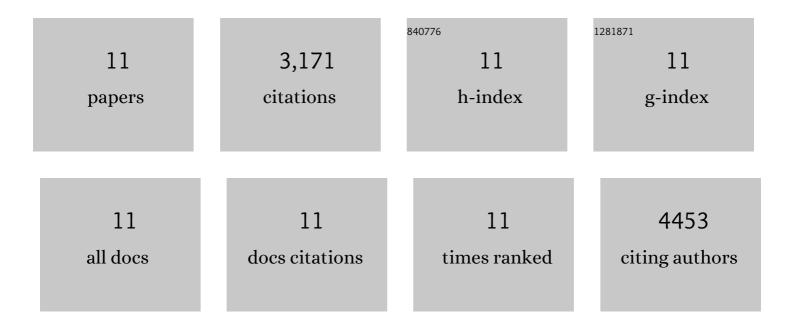
## Michael W Baughn

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/1689388/publications.pdf Version: 2024-02-01



#	Article	IF	CITATIONS
1	Premature polyadenylation-mediated loss of stathmin-2 is a hallmark of TDP-43-dependent neurodegeneration. Nature Neuroscience, 2019, 22, 180-190.	14.8	345
2	Sense-encoded poly-CR dipeptide repeat proteins correlate to neurodegeneration and uniquely co-localize with TDP-43 in dendrites of repeat-expanded C9orf72 amyotrophic lateral sclerosis. Acta Neuropathologica, 2018, 135, 459-474.	7.7	152
3	ALS/FTD-Linked Mutation in FUS Suppresses Intra-axonal Protein Synthesis and Drives Disease Without Nuclear Loss-of-Function of FUS. Neuron, 2018, 100, 816-830.e7.	8.1	185
4	Transcriptome–pathology correlation identifies interplay between TDP-43 and the expression of its kinase CK1E in sporadic ALS. Acta Neuropathologica, 2018, 136, 405-423.	7.7	69
5	Gain of Toxicity from ALS/FTD-Linked Repeat Expansions in C9ORF72 Is Alleviated by Antisense Oligonucleotides Targeting GGGGCC-Containing RNAs. Neuron, 2016, 90, 535-550.	8.1	437
6	Frequency and Complexity of De Novo Structural Mutation in Autism. American Journal of Human Genetics, 2016, 98, 667-679.	6.2	88
7	Targeting RNA Foci in iPSC-Derived Motor Neurons from ALS Patients with a <i>C9ORF72</i> Repeat Expansion. Science Translational Medicine, 2013, 5, 208ra149.	12.4	586
8	Lack of C9ORF72 coding mutations supports a gain of function for repeat expansions in amyotrophic lateral sclerosis. Neurobiology of Aging, 2013, 34, 2234.e13-2234.e19.	3.1	59
9	Targeted degradation of sense and antisense <i>C9orf72</i> RNA foci as therapy for ALS and frontotemporal degeneration. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, E4530-9.	7.1	508
10	Divergent roles of ALS-linked proteins FUS/TLS and TDP-43 intersect in processing long pre-mRNAs. Nature Neuroscience, 2012, 15, 1488-1497.	14.8	628
11	Sporadic ALS has compartment-specific aberrant exon splicing and altered cell–matrix adhesion biology. Human Molecular Genetics, 2010, 19, 313-328.	2.9	114