Leslie M Thompson

List of Publications by Year in Descending Order

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The third column is the impact factor (IF) of the journal, and the fourth column is the number of citations of the article.

24	3,050	16	44
papers	citations	h-index	g-index
44 ext. papers	3,639 ext. citations	12.9 avg, IF	3.94 L-index

#	Paper	IF	Citations
24	Answer ALS, a large-scale resource for sporadic and familial ALS combining clinical and multi-omics data from induced pluripotent cell lines <i>Nature Neuroscience</i> , 2022 , 25, 226-237	25.5	6
23	An integrated multi-omic analysis of iPSC-derived motor neurons from C9ORF72 ALS patients. <i>IScience</i> , 2021 , 24, 103221	6.1	5
22	Huntingtonly disease mice and human brain tissue exhibit increased G3BP1 granules and TDP43 mislocalization. <i>Journal of Clinical Investigation</i> , 2021 , 131,	15.9	7
21	Cell Therapy for Huntington'd Disease: Learning from Failure. Movement Disorders, 2021, 36, 787-788	7	1
20	Calcium Dynamics in Astrocytes During Cell Injury. <i>Frontiers in Bioengineering and Biotechnology</i> , 2020 , 8, 912	5.8	6
19	Mutant huntingtin impairs PNKP and ATXN3, disrupting DNA repair and transcription. <i>ELife</i> , 2019 , 8,	8.9	45
18	Human Neural Stem Cell Transplantation Rescues Functional Deficits in R6/2 and Q140 Huntington's Disease Mice. <i>Stem Cell Reports</i> , 2018 , 10, 58-72	8	57
17	Fractionation for Resolution of Soluble and Insoluble Huntingtin Species. <i>Journal of Visualized Experiments</i> , 2018 ,	1.6	6
16	Genome-wide Analyses Identify KIF5A as a Novel ALS Gene. <i>Neuron</i> , 2018 , 97, 1268-1283.e6	13.9	296
15	The ubiquitin conjugating enzyme Ube2W regulates solubility of the Huntingtonও disease protein, huntingtin. <i>Neurobiology of Disease</i> , 2018 , 109, 127-136	7.5	15
14	Modeling Psychomotor Retardation using iPSCs from MCT8-Deficient Patients Indicates a Prominent Role for the Blood-Brain Barrier. <i>Cell Stem Cell</i> , 2017 , 20, 831-843.e5	18	130
13	Huntington d Disease iPSC-Derived Brain Microvascular Endothelial Cells Reveal WNT-Mediated Angiogenic and Blood-Brain Barrier Deficits. <i>Cell Reports</i> , 2017 , 19, 1365-1377	10.6	143
12	KEAP1-modifying small molecule reveals muted NRF2 signaling responses in neural stem cells from Huntingtonly disease patients. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2017 , 114, E4676-E4685	11.5	65
11	A Comparison of mRNA Sequencing with Random Primed and 3UDirected Libraries. <i>Scientific Reports</i> , 2017 , 7, 14626	4.9	27
10	SIRT2- and NRF2-Targeting Thiazole-Containing Compound with Therapeutic Activity in Huntington's Disease Models. <i>Cell Chemical Biology</i> , 2016 , 23, 849-861	8.2	54
9	PIAS1 Regulates Mutant Huntingtin Accumulation and Huntington Disease-Associated Phenotypes In Vivo. <i>Neuron</i> , 2016 , 90, 507-20	13.9	45
8	TRiC subunits enhance BDNF axonal transport and rescue striatal atrophy in Huntington's disease. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, E5655-64	11.5	53

LIST OF PUBLICATIONS

7	Reinstating aberrant mTORC1 activity in Huntington disease mice improves disease phenotypes. <i>Neuron</i> , 2015 , 85, 303-15	13.9	115
6	Potential function for the Huntingtin protein as a scaffold for selective autophagy. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2014 , 111, 16889-94	11.5	183
5	Comparison of phosphodiesterase 10A, dopamine receptors D1 and D2 and dopamine transporter ligand binding in the striatum of the R6/2 and BACHD mouse models of Huntingtonly disease. <i>Journal of Huntingtonly Disease</i> , 2014 , 3, 333-41	1.9	13
4	A transgenic minipig model of Huntington's Disease. Journal of Huntington's Disease, 2013, 2, 47-68	1.9	70
3	Identifying polyglutamine protein species in situ that best predict neurodegeneration. <i>Nature Chemical Biology</i> , 2011 , 7, 925-34	11.7	152
2	Histone deacetylase inhibitors arrest polyglutamine-dependent neurodegeneration in Drosophila. <i>Nature</i> , 2001 , 413, 739-43	50.4	1028
1	Thanatophoric dysplasia (types I and II) caused by distinct mutations in fibroblast growth factor receptor 3. <i>Nature Genetics</i> , 1995 , 9, 321-8	36.3	524