Simone Engelender

List of Publications by Year
in descending order

67 papers	12,789 citations	94381 37 h-index	110317 64 g-index
68 all docs	68 docs citations	68 times ranked	24322 citing authors

#	Article	IF	Citations
1	Can We Treat Neurodegenerative Proteinopathies by Enhancing Protein Degradation?. Movement Disorders, 2022, 37, 1346-1359.	2.2	11
2	BMP5/7 protect dopaminergic neurons in an Î±-synuclein mouse model of Parkinsonâ€™s disease. Brain, 2021, 144, e15-e15.	3.7	11
3	ATAD3B is a mitophagy receptor mediating clearance of oxidative stressâ€induced damaged mitochondrial DNA. EMBO Journal, 2021, 40, e106283.	3.5	44
4	SUMOylation in Î±-Synuclein Homeostasis and Pathology. Frontiers in Aging Neuroscience, 2020, 12, 167.	1.7	16
5	Lipid and immune abnormalities causing age-dependent neurodegeneration and Parkinsonâ€™s disease. Journal of Neuroinflammation, 2019, 16, 153.	3.1	76
6	Physiological and pathological roles of LRRK2 in the nuclear envelope integrity. Human Molecular Genetics, 2019, 28, 3982-3996.	1.4	19
7	The NMDA receptor activation by <scp>d</scp> -serine and glycine is controlled by an astrocytic Phgdh-dependent serine shuttle. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 20736-20742.	3.3	89
8	Glycation potentiates Î±-synuclein-associated neurodegeneration in synucleinopathies. Brain, 2017, 140, 1399-1419.	3.7	153
9	SUMOylation and ubiquitination reciprocally regulate Î±-synuclein degradation and pathological aggregation. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, 13176-13181.	3.3	131
10	The Threshold Theory for Parkinson's Disease. Trends in Neurosciences, 2017, 40, 4-14.	4.2	153
11	AF-6 Protects Against Dopaminergic Dysfunction and Mitochondrial Abnormalities in Drosophila Models of Parkinsonâ€™s Disease. Frontiers in Cellular Neuroscience, 2017, 11, 241.	1.8	15
12	Ubiqutination via K27 and K29 chains signals aggregation and neuronal protection of LRRK2 by WSB1. Nature Communications, 2016, 7, 11792.	5.8	56
13	The PINK1, synphilin-1 and SIAH-1 complex constitutes a novel mitophagy pathway. Human Molecular Genetics, 2016, 25, 3476-3490.	1.4	108
14	Guidelines for the use and interpretation of assays for monitoring autophagy (3rd edition). Autophagy, 2016, 12, 1-222.	4.3	4,701
15	Synphilin-1 attenuates mutant LRRK2-induced neurodegeneration in Parkinson's disease models. Human Molecular Genetics, 2016, 25, 672-680.	1.4	19
16	Convergence of Parkin, PINK1, and Î±-Synuclein on Stress-induced Mitochondrial Morphological Remodeling. Journal of Biological Chemistry, 2015, 290, 13862-13874.	1.6	76
17	The alanineâ€serineâ€cysteineâ€1 (Ascâ€1) transporter controls glycine levels in the brain and is required for glycinergic inhibitory transmission. EMBO Reports, 2015, 16, 590-598.	2.0	48
18	α-Synuclein Ubiquitination and Novel Therapeutic Targets for Parkinsonâ€™s Disease. CNS and Neurological Disorders - Drug Targets, 2014, 13, 630-637.	0.8	24

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19	Site-Specific Differences in Proteasome-Dependent Degradation of Monoubiquitinated Î±-Synuclein. Chemistry and Biology, 2013, 20, 1207-1213.	6.2	40
20	AF-6 is a positive modulator of the PINK1/parkin pathway and is deficient in Parkinson's disease. Human Molecular Genetics, 2013, 22, 2083-2096.	1.4	25
21	Î±-synuclein fate. Autophagy, 2012, 8, 418-420.	4.3	26
22	Guidelines for the use and interpretation of assays for monitoring autophagy. Autophagy, 2012, 8, 445-544.	4.3	3,122
23	Î±-Synuclein fate is determined by USP9X-regulated monoubiquitination. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 18666-18671.	3.3	154
24	Periphilin is a novel interactor of synphilin-1, a protein implicated in Parkinson's disease. Neurogenetics, 2010, 11, 203-215.	0.7	2
25	Synphilin-1A Inhibits Seven in Absentia Homolog (SIAH) and Modulates Î±-Synuclein Monoubiquitylation and Inclusion Formation. Journal of Biological Chemistry, 2009, 284, 11706-11716.	1.6	31
26	Synphilin-1 isoforms in Parkinsonâ€™s disease: regulation by phosphorylation and ubiquitylation. Cellular and Molecular Life Sciences, 2008, 65, 80-88.	2.4	27
27	Monoubiquitylation of Î±-Synuclein by Seven in Absentia Homolog (SIAH) Promotes Its Aggregation in Dopaminergic Cells. Journal of Biological Chemistry, 2008, 283, 3316-3328.	1.6	153
28	Ubiquitination of Î±-synuclein and autophagy in Parkinson's disease. Autophagy, 2008, 4, 372-374.	4.3	90
29	The proteasomal subunit S6 ATPase is a novel synphilinâ€1 interacting proteinâ€”implications for Parkinson's disease. FASEB Journal, 2007, 21, 1759-1767.	0.2	48
30	Phosphorylation of Parkin by the Cyclin-dependent Kinase 5 at the Linker Region Modulates Its Ubiquitin-Ligase Activity and Aggregation. Journal of Biological Chemistry, 2007, 282, 12842-12850.	1.6	105
31	Mutation analysis of the seven in absentia homolog 1 (SIAH1) gene in Parkinsonâ€™s disease. Journal of Neural Transmission, 2006, 113, 1903-1908.	1.4	13
32	Synphilin Isoforms and the Search for a Cellular Model of Lewy Body Formation in Parkinson's Disease. Cell Cycle, 2006, 5, 2082-2086.	1.3	19
33	Synphilin-1A: An aggregation-prone isoform of synphilin-1 that causes neuronal death and is present in aggregates from Â synucleinopathy patients. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 5917-5922.	3.3	49
34	Glycogen Synthase Kinase 3Î² Modulates Synphilin-1 Ubiquitylation and Cellular Inclusion Formation by SIAH. Journal of Biological Chemistry, 2005, 280, 42877-42886.	1.6	41
35	Parkin Mediates Nonclassical, Proteasomal-Independent Ubiquitination of Synphilin-1: Implications for Lewy Body Formation. Journal of Neuroscience, 2005, 25, 2002-2009.	1.7	489
36	Synphilin-1 and parkin show overlapping expression patterns in human brain and form aggresomes in response to proteasomal inhibition. Neurobiology of Disease, 2005, 20, 401-411.	2.1	40

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37	Ubiquitylation of synphilin-1 and Â-synuclein by SIAH and its presence in cellular inclusions and Lewy bodies imply a role in Parkinson's disease. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 5500-5505.	3.3	177
38	Identification and functional characterization of a novel R621C mutation in the synphilin-1 gene in Parkinson's disease. Human Molecular Genetics, 2003, 12, 1223-1231.	1.4	124
39	Inducible PC12 cell model of Huntingtonâ€™s disease shows toxicity and decreased histone acetylation. NeuroReport, 2003, 14, 565-568.	0.6	68
40	Synphilin-1 Is Developmentally Localized to Synaptic Terminals, and Its Association with Synaptic Vesicles Is Modulated by Î±-Synuclein. Journal of Biological Chemistry, 2002, 277, 23927-23933.	1.6	94
41	Initial stages of radial glia astrocytic transformation in the early postnatal anterior subventricular zone. Journal of Neurobiology, 2002, 52, 251-265.	3.7	84
42	Immunocytochemical localization of synphilin-1, an Î±-synuclein-associated protein, in neurodegenerative disorders. Acta Neuropathologica, 2002, 103, 209-214.	3.9	76
43	Synphilin-1 is developmentally localized to synaptic terminals, and its association with synaptic vesicles is modulated by Î±-synuclein.. Journal of Biological Chemistry, 2002, 277, 34651-34654.	1.6	0
44	Genetic Analysis of Synphilin-1 in Familial Parkinson's Disease. Neurobiology of Disease, 2001, 8, 317-323.	2.1	18
45	No pathogenic mutations in the synphilin-1 gene in Parkinson's disease. Neuroscience Letters, 2001, 307, 125-127.	1.0	18
46	A new strategy to decrease N-methyl-D-aspartate (NMDA) receptor coactivation: Inhibition of D-serine synthesis by converting serine racemase into an eliminase. Proceedings of the National Academy of Sciences of the United States of America, 2001, 98, 5294-5299.	3.3	87
47	Inducible expression of mutant alpha-synuclein decreases proteasome activity and increases sensitivity to mitochondria-dependent apoptosis. Human Molecular Genetics, 2001, 10, 919-926.	1.4	442
48	Synphilin-1 is present in Lewy bodies in Parkinson's disease. Annals of Neurology, 2000, 47, 521-523.	2.8	246
49	Organization of the human synphilin-1 gene, a candidate for Parkinson's disease. Mammalian Genome, 2000, 11, 763-766.	1.0	21
50	Human huntingtin-associated protein (HAP-1) gene: genomic organisation and an intragenic polymorphism. Gene, 2000, 254, 181-187.	1.0	6
51	Human serine racemase: moleular cloning, genomic organization and functional analysis. Gene, 2000, 256, 183-188.	1.0	137
52	Synphilin-1 is present in Lewy bodies in Parkinson's disease. , 2000, 47, 521.		1
53	Synphilinâ€1 is present in Lewy bodies in Parkinson's disease. Annals of Neurology, 2000, 47, 521-523.	2.8	5
54	Synphilin-1: a possible molecular determinant for Parkinson's disease. Anais Da Academia Brasileira De Ciencias, 2000, 72, 439-439.	0.3	0

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55	Synphilin-1 associates with Î±-synuclein and promotes the formation of cytosolic inclusions. Nature Genetics, 1999, 22, 110-114.	9.4	473
56	Chromosomal localization of the Huntingtin Associated Protein (HAP-1) gene in mouse and humans with radiation hybrid and interspecific backcross mapping. Mammalian Genome, 1999, 10, 397-398.	1.0	5
57	Gene structure and map location of the murine homolog of the Huntington-associated protein, Hap1. Mammalian Genome, 1998, 9, 565-570.	1.0	26
58	Chapter 28 Pathogenesis of neurodegenerative diseases associated with expanded glutamine repeats: New answers, new questions. Progress in Brain Research, 1998, 117, 397-419.	0.9	35
59	Huntingtin-associated protein 1 (HAP1) binds to a Trio-like polypeptide, with a rac1 guanine nucleotide exchange factor domain. Human Molecular Genetics, 1997, 6, 1519-1525.	1.4	114
60	Sarco/endoplasmic reticulum Ca2+-ATPase isoforms: diverse responses to acidosis. Biochemical Journal, 1997, 321, 545-550.	1.7	51
61	Reaction Mechanism of the Sarcoplasmic Reticulum Ca2+-ATPase. Advances in Molecular and Cell Biology, 1997, , 1-31.	0.1	3
62	Huntington's Disease and Dentatorubralâ€Pallidoluysian Atrophy: Proteins, Pathogenesis and Pathology. Brain Pathology, 1997, 7, 1003-1016.	2.1	60
63	Inhibition of creatine kinase byS-nitrosoglutathione. FEBS Letters, 1996, 392, 274-276.	1.3	132
64	Regulation of the channel function of Ca2+-ATPase. Biochimica Et Biophysica Acta - Bioenergetics, 1996, 1275, 105-110.	0.5	15
65	The Ca2+-ATPase Isoforms of Platelets Are Located in Distinct Functional Ca2+ Pools and Are Uncoupled by a Mechanism Different from That of Skeletal Muscle Ca2+-ATPase. Journal of Biological Chemistry, 1995, 270, 21050-21055.	1.6	37
66	Protein interactions with a gender-specific gene ofSchistosoma mansoni: characterization by DNase I footprinting, band shift and UV cross-linking. Molecular and Cellular Biochemistry, 1993, 124, 159-168.	1.4	8
67	Protein-DNA associations in a gender-specific gene of Schistosoma mansoni: characterization by UV cross-linking, DNase I footprinting and band shift assays. Memorias Do Instituto Oswaldo Cruz, 1992, 87, 67-70.	0.8	0