Alexander J Whitworth

List of Publications by Year
in descending order

64 papers	15,772 citations	101543 36 h-index	118850 62 g-index
75 all docs	75 docs citations	75 times ranked	26102 citing authors

#	Article	IF	Citations
1	Guidelines for the use and interpretation of assays for monitoring autophagy (3rd edition). Autophagy, 2016, 12, 1-222.	9.1	4,701
2	Guidelines for the use and interpretation of assays for monitoring autophagy. Autophagy, 2012, 8, 445-544.	9.1	3,122
3	Mitochondrial pathology and apoptotic muscle degeneration in Drosophila parkin mutants. Proceedings of the National Academy of Sciences of the United States of America, 2003, 100, 4078-4083.	7.1	1,117
4	The PINK1/Parkin pathway regulates mitochondrial morphology. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 1638-1643.	7.1	783
5	Drosophila Parkin requires PINK1 for mitochondrial translocation and ubiquitinates Mitofusin. Proceedings of the National Academy of Sciences of the United States of America, 2010, 107, 5018-5023.	7.1	682
6	PINK1 cleavage at position A103 by the mitochondrial protease PARL. Human Molecular Genetics, 2011, 20, 867-879.	2.9	385
7	Increased glutathione S -transferase activity rescues dopaminergic neuron loss in a Drosophila model of Parkinson's disease. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 8024-8029.	7.1	374
8	Drosophila DJ-1 Mutants Are Selectively Sensitive to Environmental Toxins Associated with Parkinsonâ€™s Disease. Current Biology, 2005, 15, 1572-1577.	3.9	332
9	The C9orf72 protein interacts with Rab1a and the <scp>ULK</scp>1 complex to regulate initiation of autophagy. EMBO Journal, 2016, 35, 1656-1676.	7.8	327
10	The Parkinson's diseaseâ€“linked proteins Fbxo7 and Parkin interact to mediate mitophagy. Nature Neuroscience, 2013, 16, 1257-1265.	14.8	292
11	Rapamycin activation of 4E-BP prevents parkinsonian dopaminergic neuron loss. Nature Neuroscience, 2009, 12, 1129-1135.	14.8	288
12	Basal mitophagy is widespread in Drosophila but minimally affected by loss of Pink1 or parkin. Journal of Cell Biology, 2018, 217, 1613-1622.	5.2	253
13	The NAD+ Precursor Nicotinamide Riboside Rescues Mitochondrial Defects and Neuronal Loss in iPSC and Fly Models of Parkinsonâ€™s Disease. Cell Reports, 2018, 23, 2976-2988.	6.4	239
14	Increasing microtubule acetylation rescues axonal transport and locomotor deficits caused by LRRK2 Roc-COR domain mutations. Nature Communications, 2014, 5, 5245.	12.8	229
15	Genetic and genomic studies of Drosophila parkin mutants implicate oxidative stress and innate immune responses in pathogenesis. Human Molecular Genetics, 2005, 14, 799-811.	2.9	178
16	Rhomboid-7 and HtrA2/Omi act in a common pathway with the Parkinsonâ€™s disease factors Pink1 and Parkin. DMM Disease Models and Mechanisms, 2008, 1, 168-174.	2.4	174
17	PINK1/Parkin mitophagy and neurodegenerationâ€”what do we really know in vivo ?. Current Opinion in Genetics and Development, 2017, 44, 47-53.	3.3	143
18	Parkinson disease-linked GBA mutation effects reversed by molecular chaperones in human cell and fly models. Scientific Reports, 2016, 6, 31380.	3.3	133

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19	The PINK1/Parkin pathway: a mitochondrial quality control system?. Journal of Bioenergetics and Biomembranes, 2009, 41, 499-503.	2.3	118
20	Genome-wide RNAi screen identifies the Parkinson disease GWAS risk locus SREBF1 as a regulator of mitophagy. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, 8494-8499.	7.1	109
21	SRSF1-dependent nuclear export inhibition of C9ORF72 repeat transcripts prevents neurodegeneration and associated motor deficits. Nature Communications, 2017, 8, 16063.	12.8	106
22	Drosophila models pioneer a new approach to drug discovery for Parkinson's disease. Drug Discovery Today, 2006, 11, 119-126.	6.4	95
23	Discovery of catalytically active orthologues of the Parkinson's disease kinase PINK1: analysis of substrate specificity and impact of mutations. Open Biology, 2011, 1, 110012.	3.6	88
24	Axonal transport defects are a common phenotype inDrosophilamodels of ALS. Human Molecular Genetics, 2016, 25, ddw105.	2.9	88
25	Mitochondrial impairment activates the Wallerian pathway through depletion of NMNAT2 leading to SARM1-dependent axon degeneration. Neurobiology of Disease, 2020, 134, 104678.	4.4	87
26	TRAP1 rescues PINK1 loss-of-function phenotypes. Human Molecular Genetics, 2013, 22, 2829-2841.	2.9	81
27	Drosophila Models of Parkinson's Disease. Advances in Genetics, 2011, 73, 1-50.	1.8	80
28	Mechanisms of Parkinson's Disease. Current Topics in Developmental Biology, 2017, 121, 173-200.	2.2	79
29	Drosophila HtrA2 is dispensable for apoptosis but acts downstream of PINK1 independently from Parkin. Cell Death and Differentiation, 2009, 16, 1118-1125.	11.2	77
30	The Complex I Subunit NDUFA10 Selectively Rescues Drosophila pink1 Mutants through a Mechanism Independent of Mitophagy. PLoS Genetics, 2014, 10, e1004815.	3.5	68
31	VPS35pathogenic mutations confer no dominant toxicity but partial loss of function inDrosophilaand genetically interact withparkin. Human Molecular Genetics, 2015, 24, 6106-6117.	2.9	67
32	Metallobiology and therapeutic chelation of biometals (copper, zinc and iron) in Alzheimerâ€™s disease: Limitations, and current and future perspectives. Journal of Trace Elements in Medicine and Biology, 2021, 67, 126779.	3.0	60
33	Modulation of mitochondrial function and morphology by interaction of Omi/HtrA2 with the mitochondrial fusion factor OPA1. Experimental Cell Research, 2010, 316, 1213-1224.	2.6	57
34	Superoxide Dismutase (SOD)-mimetic M40403 Is Protective in Cell and Fly Models of Paraquat Toxicity. Journal of Biological Chemistry, 2016, 291, 9257-9267.	3.4	56
35	How could Parkin-mediated ubiquitination of mitofusin promote mitophagy?. Autophagy, 2010, 6, 660-662.	9.1	55
36	Comprehensive Genetic Characterization of Mitochondrial Ca2+ Uniporter Components Reveals Their Different Physiological Requirements InÂVivo. Cell Reports, 2019, 27, 1541-1550.e5.	6.4	46

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37	Superoxide Radical Dismutation as New Therapeutic Strategy in Parkinsonâ€™s Disease. , 2018, 9, 716.		42
38	Temporally dynamic response to Wingless directs the sequential elaboration of the proximodistal axis of the Drosophila wing. Developmental Biology, 2003, 254, 277-288.	2.0	39
39	Effects of Five Ayurvedic Herbs on Locomotor Behaviour in a <scp>Drosophila melanogaster</scp> Parkinson's Disease Model. Phytotherapy Research, 2014, 28, 1789-1795.	5.8	37
40	Mitochondrial defects and neuromuscular degeneration caused by altered expression of Drosophila Gdap1: implications for the Charcotâ€“Marieâ€“Tooth neuropathy. Human Molecular Genetics, 2015, 24, 21-36.	2.9	37
41	Enhancing Mitofusin/Marf ameliorates neuromuscular dysfunction in Drosophila models of TDP-43 proteinopathies. Neurobiology of Aging, 2017, 54, 71-83.	3.1	35
42	SREBF1links lipogenesis to mitophagy and sporadic Parkinson disease. Autophagy, 2014, 10, 1476-1477.	9.1	33
43	Superoxide dismutating molecules rescue the toxic effects of PINK1 and parkin loss. Human Molecular Genetics, 2018, 27, 1618-1629.	2.9	28
44	Mutation in the MICOS subunit gene APOO (MIC26) associated with an X-linked recessive mitochondrial myopathy, lactic acidosis, cognitive impairment and autistic features. Journal of Medical Genetics, 2021, 58, 155-167.	3.2	28
45	A SCA7 CAG/CTG repeat expansion is stable in Drosophila melanogaster despite modulation of genomic context and gene dosage. Gene, 2005, 347, 35-41.	2.2	23
46	Molecular Mechanisms of PINK1-Related Neurodegeneration. Current Neurology and Neuroscience Reports, 2011, 11, 283-290.	4.2	23
47	Mitochondrially-targeted APOBEC1 is a potent mtDNA mutator affecting mitochondrial function and organismal fitness in Drosophila. Nature Communications, 2019, 10, 3280.	12.8	23
48	Inhibition of the deubiquitinase USP8 corrects a Drosophila PINK1 model of mitochondria dysfunction. Life Science Alliance, 2019, 2, e201900392.	2.8	22
49	DGAT1 activity synchronises with mitophagy to protect cells from metabolic rewiring by ironâ€‰â€‰depletion. EMBO Journal, 2022, 41, e109390.	7.8	22
50	The STING pathway does not contribute to behavioural or mitochondrial phenotypes in Drosophila Pink1/parkin or mtDNA mutator models. Scientific Reports, 2020, 10, 2693.	3.3	20
51	Decreasing pdzd8-mediated mitoâ€“ER contacts improves organismal fitness and mitigates AÎ²₄₂toxicity. Life Science Alliance, 2022, 5, e202201531.	2.8	20
52	Antioxidant Therapy in Parkinsonâ€™s Disease: Insights from Drosophila melanogaster. Antioxidants, 2020, 9, 52.	5.1	19
53	DJ-1: A promising therapeutic candidate for ischemia-reperfusion injury. Redox Biology, 2021, 41, 101884.	9.0	18
54	Modeling Pathogenic Mutations of Human Twinkle in Drosophila Suggests an Apoptosis Role in Response to Mitochondrial Defects. PLoS ONE, 2012, 7, e43954.	2.5	18

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55	An assessment of the rescue action of resveratrol in parkin loss of function-induced oxidative stress in Drosophila melanogaster. Scientific Reports, 2022, 12, 3922.	3.3	15
56	Drosophila phosphatidylinositol-4 kinase fwd promotes mitochondrial fission and can suppress Pink1/parkin phenotypes. PLoS Genetics, 2020, 16, e1008844.	3.5	14
57	SRSF1-dependent inhibition of C9ORF72-repeat RNA nuclear export: genome-wide mechanisms for neuroprotection in amyotrophic lateral sclerosis. Molecular Neurodegeneration, 2021, 16, 53.	10.8	13
58	A late-stage assembly checkpoint of the human mitochondrial ribosome large subunit. Nature Communications, 2022, 13, 929.	12.8	13
59	Protective capacity of carotenoid trans-astaxanthin in rotenone-induced toxicity in Drosophila melanogaster. Scientific Reports, 2022, 12, 4594.	3.3	13
60	The many faces of mitophagy. EMBO Reports, 2014, 15, 5-6.	4.5	10
61	Characterization of Drosophila ATPsynC mutants as a new model of mitochondrial ATP synthase disorders. PLoS ONE, 2018, 13, e0201811.	2.5	7
62	Comment on â€œmt-Keima detects PINK1-PRKN mitophagy in vivo with greater sensitivity than mito-QCâ€• Autophagy, 2021, 17, 4477-4479.	9.1	4
63	Translating translation: Regulated protein translation as a biomedical intervention. Fly, 2009, 3, 278-280.	1.7	3
64	Drosophila Models of Parkinson Disease. , 2005, , 173-182.		1