Christopher D Link

List of Publications by Year
in descending order

62 papers	6,676 citations	76326 40 h-index	123424 61 g-index
67 all docs	67 docs citations	67 times ranked	7812 citing authors

#	Article	IF	Citations
1	The gut microbiomeâ€“derived metabolite trimethylamine N-oxide modulates neuroinflammation and cognitive function with aging. GeroScience, 2021, 43, 377-394.	4.6	85
2	Is There a Brain Microbiome?. Neuroscience Insights, 2021, 16, 263310552110187.	1.6	31
3	Application of a bioinformatic pipeline to RNA-seq data identifies novel virus-like sequence in human blood. G3: Genes, Genomes, Genetics, 2021, 11, .	1.8	4
4	Amyloid beta acts synergistically as a pro-inflammatory cytokine. Neurobiology of Disease, 2021, 159, 105493.	4.4	29
5	TDP-43 knockdown causes innate immune activation via protein kinase R in astrocytes. Neurobiology of Disease, 2019, 132, 104514.	4.4	37
6	Î±-Sheet secondary structure in amyloid Î²-peptide drives aggregation and toxicity in Alzheimerâ€™s disease. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 8895-8900.	7.1	118
7	Heat shock in C. elegans induces downstream of gene transcription and accumulation of double-stranded RNA. PLoS ONE, 2019, 14, e0206715.	2.5	14
8	Neurodegeneration, Heterochromatin, and Double-Stranded RNA. Journal of Experimental Neuroscience, 2019, 13, 117906951983069.	2.3	17
9	Loss of glutathione redox homeostasis impairs proteostasis by inhibiting autophagy-dependent protein degradation. Cell Death and Differentiation, 2019, 26, 1545-1565.	11.2	30
10	Heterochromatin anomalies and double-stranded RNA accumulation underlie <i>C9orf72</i> poly(PR) toxicity. Science, 2019, 363, .	12.6	181
11	RNA self-assembly contributes to stress granule formation and defining the stress granule transcriptome. Proceedings of the National Academy of Sciences of the United States of America, 2018, 115, 2734-2739.	7.1	402
12	In vivo induction of membrane damage by Î²-amyloid peptide oligomers. Acta Neuropathologica Communications, 2018, 6, 131.	5.2	31
13	The <i>Caenorhabditis elegans</i> Ortholog of TDP-43 Regulates the Chromatin Localization of the Heterochromatin Protein 1 Homolog HPL-2. Molecular and Cellular Biology, 2018, 38, .	2.3	14
14	Transcriptome analysis of genetically matched human induced pluripotent stem cells disomic or trisomic for chromosome 21. PLoS ONE, 2018, 13, e0194581.	2.5	31
15	Sedimentation Velocity Analysis with Fluorescence Detection of Mutant Huntingtin Exon 1 Aggregation in <i>Drosophila melanogaster</i> and <i>Caenorhabditis elegans</i>. Biochemistry, 2017, 56, 4676-4688.	2.5	4
16	Repetitive element transcripts are elevated in the brain of C9orf72 ALS/FTLD patients. Human Molecular Genetics, 2017, 26, 3421-3431.	2.9	101
17	DLK-1, SEK-3 and PMK-3 Are Required for the Life Extension Induced by Mitochondrial Bioenergetic Disruption in C. elegans. PLoS Genetics, 2016, 12, e1006133.	3.5	52
18	Studying polyglutamine aggregation in <i>Caenorhabditis elegans</i> using an analytical ultracentrifuge equipped with fluorescence detection. Protein Science, 2016, 25, 605-617.	7.6	10

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19	Spt4 selectively regulates the expression of <i>C9orf72</i> sense and antisense mutant transcripts. Science, 2016, 353, 708-712.	12.6	116
20	Caenorhabditis elegans as a model system to study post-translational modifications of human transthyretin. Scientific Reports, 2016, 6, 37346.	3.3	12
21	C9ORF72 poly(GA) aggregates sequester and impair HR23 and nucleocytoplasmic transport proteins. Nature Neuroscience, 2016, 19, 668-677.	14.8	268
22	Identifying Aβ-specific pathogenic mechanisms using a nematode model of Alzheimer's disease. Neurobiology of Aging, 2015, 36, 857-866.	3.1	22
23	Distinct brain transcriptome profiles in C9orf72-associated and sporadic ALS. Nature Neuroscience, 2015, 18, 1175-1182.	14.8	330
24	A semi-automated motion-tracking analysis of locomotion speed in the C. elegans transgenics overexpressing beta-amyloid in neurons. Frontiers in Genetics, 2014, 5, 202.	2.3	19
25	<scp>TDP</scp>â€1, the <i><scp>C</scp>aenorhabditis elegans</i> ortholog of <scp>TDP</scp>â€43, limits the accumulation of doubleâ€stranded <scp>RNA</scp>. EMBO Journal, 2014, 33, 2947-2966.	7.8	62
26	Alzheimer's disease drug discovery: in vivo screening using Caenorhabditis elegans as a model for Î²-amyloid peptide-induced toxicity. Drug Discovery Today: Technologies, 2013, 10, e115-e119.	4.0	95
27	Cell Death by Glutamine Repeats?. Science, 2012, 335, 926-927.	12.6	1
28	Utility of an improved model of amyloid-beta (Aβ1-42) toxicity in Caenorhabditis elegans for drug screening for Alzheimerâ€™s disease. Molecular Neurodegeneration, 2012, 7, 57.	10.8	188
29	A glycine zipper motif mediates the formation of toxic Î²-amyloid oligomers in vitro and in vivo. Molecular Neurodegeneration, 2011, 6, 61.	10.8	37
30	Assaying β-amyloid Toxicity using a Transgenic C. elegans Model. Journal of Visualized Experiments, 2010, , .	0.3	57
31	Lifeâ€span extension by dietary restriction is mediated by NLPâ€7 signaling and coelomocyte endocytosis in<i>C. elegans</i>. FASEB Journal, 2010, 24, 383-392.	0.5	52
32	Genetic Mechanisms of Coffee Extract Protection in a<i>Caenorhabditis elegans</i>Model of Î²-Amyloid Peptide Toxicity. Genetics, 2010, 186, 857-866.	2.9	106
33	Neurotoxic effects of TDP-43 overexpression in C. elegans. Human Molecular Genetics, 2010, 19, 3206-3218.	2.9	205
34	Behavioral Phenotyping of a Transgenic Caenorhabditis Elegans Expressing Neuronal Amyloid-Î². Journal of Alzheimer's Disease, 2010, 19, 681-690.	2.6	92
35	Insulin-like Signaling Determines Survival during Stress via Posttranscriptional Mechanisms in C. elegans. Cell Metabolism, 2010, 12, 260-272.	16.2	113
36	F1-01-01: Coffee protects against in vivo Abeta toxicity via activation of the skn-1 (Nrf2) detoxification pathway. , 2010, 6, S60-S61.		0

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37	AIP-1 ameliorates Î²-amyloid peptide toxicity in a Caenorhabditis elegans Alzheimer's disease model. Human Molecular Genetics, 2009, 18, 2739-2747.	2.9	56
38	What have worm models told us about the mechanisms of neuronal dysfunction in human neurodegenerative diseases?. Molecular Neurodegeneration, 2009, 4, 38.	10.8	62
39	Aberrant cleavage of TDP-43 enhances aggregation and cellular toxicity. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 7607-7612.	7.1	523
40	The Î² amyloid peptide can act as a modular aggregation domain. Neurobiology of Disease, 2008, 32, 420-425.	4.4	8
41	Suppression of in Vivo Î²-Amyloid Peptide Toxicity by Overexpression of the HSP-16.2 Small Chaperone Protein. Journal of Biological Chemistry, 2008, 283, 784-791.	3.4	133
42	Decreased Insulin-Receptor Signaling Promotes the Autophagic Degradation of Î²-Amyloid Peptide in<i>C. elegans</i>. Autophagy, 2007, 3, 569-580.	9.1	125
43	Corrigendum to â€œCompensatory regulation among ER chaperones in <i>C. elegans</i>â€•[FEBS Lett. 579 (2005) 3063â€“3068]. FEBS Letters, 2007, 581, 5952-5952.	2.8	2
44	Proteomic identification of proteins specifically oxidized in Caenorhabditis elegans expressing human AÎ²(1â€“42): Implications for Alzheimer's disease. Neurobiology of Aging, 2006, 27, 1239-1249.	3.1	89
45	C. elegans models of age-associated neurodegenerative diseases: Lessons from transgenic worm models of Alzheimerâ€™s disease. Experimental Gerontology, 2006, 41, 1007-1013.	2.8	181
46	Amyloid-Â-Induced Pathological Behaviors Are Suppressed by Ginkgo biloba Extract EGb 761 and Ginkgolides in Transgenic Caenorhabditis elegans. Journal of Neuroscience, 2006, 26, 13102-13113.	3.6	359
47	Conversion of Green Fluorescent Protein into a Toxic, Aggregation-prone Protein by C-terminal Addition of a Short Peptide. Journal of Biological Chemistry, 2006, 281, 1808-1816.	3.4	72
48	Invertebrate models of Alzheimer's disease. Genes, Brain and Behavior, 2005, 4, 147-156.	2.2	89
49	Soy isoflavone glycitein protects against beta amyloid-induced toxicity and oxidative stress in transgenic Caenorhabditis elegans. BMC Neuroscience, 2005, 6, 54.	1.9	123
50	A pilot proteomic study of amyloid precursor interactors in Alzheimer's disease. Annals of Neurology, 2005, 58, 277-289.	5.3	62
51	Compensatory regulation among ER chaperones in C. elegans. FEBS Letters, 2005, 579, 3063-3068.	2.8	71
52	A stress-responsive glutathione S-transferase confers resistance to oxidative stress in Caenorhabditis elegans. Free Radical Biology and Medicine, 2003, 34, 1405-1415.	2.9	162
53	Gene expression analysis in a transgenic Caenorhabditis elegans Alzheimer's disease model. Neurobiology of Aging, 2003, 24, 397-413.	3.1	261
54	Oxidative stress precedes fibrillar deposition of Alzheimer's disease amyloid Î²-peptide (1â€“42) in a transgenic Caenorhabditis elegans model. Neurobiology of Aging, 2003, 24, 415-420.	3.1	345

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55	Expression of the small heatâ€shock protein Hspâ€16â€2 in Caenorhabditis elegans is suppressed by Ginkgo biloba extract EGb 761. FASEB Journal, 2003, 17, 2305-2307.	0.5	120
56	Interaction of intracellular Â amyloid peptide with chaperone proteins. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 9439-9444.	7.1	192
57	Reporter Transgenes for Study of Oxidant Stress in Caenorhabditis elegans. Methods in Enzymology, 2002, 353, 497-505.	1.0	82
58	Visualization of fibrillar amyloid deposits in living, transgenic Caenorhabditis elegans animals using the sensitive amyloid dye, X-34. Neurobiology of Aging, 2001, 22, 217-226.	3.1	147
59	Transgenic invertebrate models of age-associated neurodegenerative diseases. Mechanisms of Ageing and Development, 2001, 122, 1639-1649.	4.6	97
60	In Vitro and in vivo Protein Oxidation Induced by Alzheimer's Disease Amyloid beta-Peptide (1-42). Annals of the New York Academy of Sciences, 1999, 893, 265-268.	3.8	24
61	Direct observation of stress response in Caenorhabditis elegans using a reporter transgene. Cell Stress and Chaperones, 1999, 4, 235.	2.9	178
62	In Vivo Aggregation of Î²â€Amyloid Peptide Variants. Journal of Neurochemistry, 1998, 71, 1616-1625.	3.9	146