Justin J Yerbury

List of Publications by Year
in descending order

89	4,910	87723 38	98622 67
papers	citations	h-index	g-index

97	97	97	6438
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Ubiquitin homeostasis disruption, a common cause of proteostasis collapse in amyotrophic lateral sclerosis?. Neural Regeneration Research, 2022, 17, 2218.	1.6	8
2	Vulnerability of the spinal motor neuron presynaptic terminal sub-proteome in ALS. Neuroscience Letters, 2022, 778, 136614.	1.0	3
3	TDP-43 is a ubiquitylation substrate of the SCFcyclin F complex. Neurobiology of Disease, 2022, 167, 105673.	2.1	11
4	P2X7 receptor activation mediates superoxide dismutase 1 (SOD1) release from murine NSC-34 motor neurons. Purinergic Signalling, 2022, 18, 451-467.	1.1	7
5	Proteostasis impairment and ALS. Progress in Biophysics and Molecular Biology, 2022, 174, 3-27.	1.4	7
6	Unbiased Label-Free Quantitative Proteomics of Cells Expressing Amyotrophic Lateral Sclerosis (ALS) Mutations in CCNF Reveals Activation of the Apoptosis Pathway: A Workflow to Screen Pathogenic Gene Mutations. Frontiers in Molecular Neuroscience, 2021, 14, 627740.	1.4	12
7	Non-Viral Vector-Mediated Gene Therapy for ALS: Challenges and Future Perspectives. Molecular Pharmaceutics, 2021, 18, 2142-2160.	2.3	31
8	Behavioural effects of cage systems on theG93ASuperoxide Dismutase 1transgenic mouse model for amyotrophic lateral sclerosis. Genes, Brain and Behavior, 2021, 20, e12735.	1.1	0
9	Disabled in academia: to be or not to be, that is the question. Trends in Neurosciences, 2021, 44, 507-509.	4.2	22
10	CuATSM improves motor function and extends survival but is not tolerated at a high dose in SOD1G93A mice with a C57BL/6 background. Scientific Reports, 2021, 11, 19392.	1.6	9
11	Mutant Cu/Zn Superoxide Dismutase (A4V) Turnover Is Altered in Cells Containing Inclusions. Frontiers in Molecular Neuroscience, 2021, 14, 771911.	1.4	6
12	Novel behavioural characteristics of the superoxide dismutase 1 G93A (SOD1^{G93A}) mouse model of amyotrophic lateral sclerosis include sexâ€dependent phenotypes. Genes, Brain and Behavior, 2020, 19, e12604.	1.1	14
13	The microglial NLRP3 inflammasome is activated by amyotrophic lateral sclerosis proteins. Glia, 2020, 68, 407-421.	2.5	133
14	Trajectory Taken by Dimeric Cu/Zn Superoxide Dismutase through the Protein Unfolding and Dissociation Landscape Is Modulated by Salt Bridge Formation. Analytical Chemistry, 2020, 92, 1702-1711.	3.2	9
15	Neurodegenerative disease-associated protein aggregates are poor inducers of the heat shock response in neuronal cells. Journal of Cell Science, 2020, 133, .	1.2	6
16	Selenium-based compounds: Emerging players in the ever-unfolding story of SOD1 in amyotrophic lateral sclerosis.. EBioMedicine, 2020, 59, 102997.	2.7	1
17	Ubiquitin Homeostasis Is Disrupted in TDP-43 and FUS Cell Models of ALS. IScience, 2020, 23, 101700.	1.9	28
18	The P2X7 receptor antagonist JNJ-47965567 administered thrice weekly from disease onset does not alter progression of amyotrophic lateral sclerosis in SOD1G93A mice. Purinergic Signalling, 2020, 16, 109-122.	1.1	23

#	Article	IF	Citations
19	Tryptophan residue 32 in human Cu-Zn superoxide dismutase modulates prion-like propagation and strain selection. PLoS ONE, 2020, 15, e0227655.	1.1	22
20	Proteome Homeostasis Dysfunction: A Unifying Principle in ALS Pathogenesis. Trends in Neurosciences, 2020, 43, 274-284.	4.2	47
21	The pivotal role of ubiquitin-activating enzyme E1 (UBA1) in neuronal health and neurodegeneration. International Journal of Biochemistry and Cell Biology, 2020, 123, 105746.	1.2	16
22	Prion-Like Propagation of Protein Misfolding and Aggregation in Amyotrophic Lateral Sclerosis. Frontiers in Molecular Neuroscience, 2019, 12, 262.	1.4	101
23	Using Tetracysteine-Tagged TDP-43 with a Biarsenical Dye To Monitor Real-Time Trafficking in a Cell Model of Amyotrophic Lateral Sclerosis. Biochemistry, 2019, 58, 4086-4095.	1.2	9
24	The Ubiquitin Proteasome System Is a Key Regulator of Pluripotent Stem Cell Survival and Motor Neuron Differentiation. Cells, 2019, 8, 581.	1.8	31
25	The metastability of the proteome of spinal motor neurons underlies their selective vulnerability in ALS. Neuroscience Letters, 2019, 704, 89-94.	1.0	22
26	Dynamic interplay between H-current and M-current controls motoneuron hyperexcitability in amyotrophic lateral sclerosis. Cell Death and Disease, 2019, 10, 310.	2.7	38
27	CuATSM Protects Against the In Vitro Cytotoxicity of Wild-Type-Like Copperâ€“Zinc Superoxide Dismutase Mutants but not Mutants That Disrupt Metal Binding. ACS Chemical Neuroscience, 2019, 10, 1555-1564.	1.7	21
28	Strategies to promote the maturation of ALS-associated SOD1 mutants: small molecules return to the fold. Neural Regeneration Research, 2019, 14, 1511.	1.6	9
29	Association of Regulatory T-Cell Expansion With Progression of Amyotrophic Lateral Sclerosis. JAMA Neurology, 2018, 75, 681.	4.5	120
30	The cysteine-reactive small molecule ebselen facilitates effective SOD1 maturation. Nature Communications, 2018, 9, 1693.	5.8	71
31	Assessment of metal concentrations in the SOD1G93A mouse model of amyotrophic lateral sclerosis and its potential role in muscular denervation, with particular focus on muscle tissue. Molecular and Cellular Neurosciences, 2018, 88, 319-329.	1.0	2
32	Pathogenic mutation in the ALS/FTD gene, CCNF, causes elevated Lys48-linked ubiquitylation and defective autophagy. Cellular and Molecular Life Sciences, 2018, 75, 335-354.	2.4	44
33	Dysregulation of microRNA biogenesis machinery and microRNA/RNA ratio in skeletal muscle of amyotrophic lateral sclerosis mice. Muscle and Nerve, 2018, 57, 838-847.	1.0	9
34	TryptophanÂ32-mediated SOD1 aggregation is attenuated by pyrimidine-like compounds in living cells. Scientific Reports, 2018, 8, 15590.	1.6	32
35	Nucleo-cytoplasmic transport of TDP-43 studied in real time: impaired microglia function leads to axonal spreading of TDP-43 in degenerating motor neurons. Acta Neuropathologica, 2018, 136, 445-459.	3.9	66
36	SOD1A4V aggregation alters ubiquitin homeostasis in a cell model of ALS. Journal of Cell Science, 2018, 131, .	1.2	39

#	Article	IF	Citations
37	Spinal motor neuron protein supersaturation patterns are associated with inclusion body formation in ALS. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, E3935-E3943.	3.3	91
38	Flow cytometric measurement of the cellular propagation of TDP-43 aggregation. Prion, 2017, 11, 195-204.	0.9	32
39	Addition of exogenous SOD1 aggregates causes TDP-43 mislocalisation and aggregation. Cell Stress and Chaperones, 2017, 22, 893-902.	1.2	15
40	Longitudinal assessment of metal concentrations and copper isotope ratios in the G93A SOD1 mouse model of amyotrophic lateral sclerosis. Metallomics, 2017, 9, 161-174.	1.0	12
41	The relevance of contact-independent cell-to-cell transfer of TDP-43 and SOD1 in amyotrophic lateral sclerosis. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2017, 1863, 2762-2771.	1.8	29
42	Casein kinase II phosphorylation of cyclin F at serine 621 regulates the Lys48-ubiquitylation E3 ligase activity of the SCF (cyclin F) complex. Open Biology, 2017, 7, 170058.	1.5	29
43	Motor neuron disease proteins activate complement and generate C5a. Molecular Immunology, 2017, 89, 168.	1.0	0
44	Improving the Delivery of SOD1 Antisense Oligonucleotides to Motor Neurons Using Calcium Phosphate-Lipid Nanoparticles. Frontiers in Neuroscience, 2017, 11, 476.	1.4	53
45	The heat shock response in neurons and astroglia and its role in neurodegenerative diseases. Molecular Neurodegeneration, 2017, 12, 65.	4.4	60
46	Clusterin protects neurons against intracellular proteotoxicity. Acta Neuropathologica Communications, 2017, 5, 81.	2.4	47
47	P2X7 receptor antagonism in amyotrophic lateral sclerosis. Neural Regeneration Research, 2017, 12, 749.	1.6	15
48	P2X7 antagonism using Brilliant Blue G reduces body weight loss and prolongs survival in female SOD1^{G93A}amyotrophic lateral sclerosis mice. PeerJ, 2017, 5, e3064.	0.9	41
49	Susceptibility of Mutant SOD1 to Form a Destabilized Monomer Predicts Cellular Aggregation and Toxicity but Not In vitro Aggregation Propensity. Frontiers in Neuroscience, 2016, 10, 499.	1.4	75
50	Rapid flow cytometric measurement of protein inclusions and nuclear trafficking. Scientific Reports, 2016, 6, 31138.	1.6	31
51	Walking the tightrope: proteostasis and neurodegenerative disease. Journal of Neurochemistry, 2016, 137, 489-505.	2.1	176
52	CCNF mutations in amyotrophic lateral sclerosis and frontotemporal dementia. Nature Communications, 2016, 7, 11253.	5.8	174
53	Disease Mechanisms in ALS: Misfolded SOD1 Transferred Through Exosome-Dependent and Exosome-Independent Pathways. Cellular and Molecular Neurobiology, 2016, 36, 377-381.	1.7	80
54	Protein aggregates stimulate macropinocytosis facilitating their propagation. Prion, 2016, 10, 119-126.	0.9	20

#	Article	IF	Citations
55	Network Approaches to the Understanding of Alzheimerâ€™s Disease: From Model Organisms to Humans. Methods in Molecular Biology, 2016, 1303, 447-458.	0.4	3
56	Distinct partitioning of ALS associated TDP-43, FUS and SOD1 mutants into cellular inclusions. Scientific Reports, 2015, 5, 13416.	1.6	109
57	SerpinB2 (PAI-2) Modulates Proteostasis via Binding Misfolded Proteins and Promotion of Cytoprotective Inclusion Formation. PLoS ONE, 2015, 10, e0130136.	1.1	30
58	The role of macropinocytosis in the propagation of protein aggregation associated with neurodegenerative diseases. Frontiers in Physiology, 2015, 6, 277.	1.3	45
59	Evaluation of Skin Fibroblasts from Amyotrophic Lateral Sclerosis Patients for the Rapid Study of Pathological Features. Neurotoxicity Research, 2015, 28, 138-146.	1.3	30
60	SOD1 protein aggregates stimulate macropinocytosis in neurons to facilitate their propagation. Molecular Neurodegeneration, 2015, 10, 57.	4.4	68
61	Intercellular propagated misfolding of wild-type Cu/Zn superoxide dismutase occurs via exosome-dependent and -independent mechanisms. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, 3620-3625.	3.3	373
62	Misfolded Polyglutamine, Polyalanine, and Superoxide Dismutase 1 Aggregate via Distinct Pathways in the Cell. Journal of Biological Chemistry, 2014, 289, 6669-6680.	1.6	39
63	Proteaseâ€activated alphaâ€2â€macroglobulin can inhibit amyloid formation via two distinct mechanisms. FEBS Letters, 2013, 587, 398-403.	1.3	43
64	Extracellular aggregated Cu/Zn superoxide dismutase activates microglia to give a cytotoxic phenotype. Glia, 2013, 61, 409-419.	2.5	81
65	Extracellular Chaperones and Proteostasis. Annual Review of Biochemistry, 2013, 82, 295-322.	5.0	156
66	Extracellular wildtype and mutant SOD1 induces ERâ€“Golgi pathology characteristic of amyotrophic lateral sclerosis in neuronal cells. Cellular and Molecular Life Sciences, 2013, 70, 4181-4195.	2.4	59
67	The small heat shock proteins Î±B-crystallin and Hsp27 suppress SOD1 aggregation in vitro. Cell Stress and Chaperones, 2013, 18, 251-257.	1.2	76
68	P2X7 Receptor Activation Induces Reactive Oxygen Species Formation and Cell Death in Murine EOC13 Microglia. Mediators of Inflammation, 2013, 2013, 1-18.	1.4	79
69	Glutathionylation potentiates benign superoxide dismutase 1 variants to the toxic forms associated with amyotrophic lateral sclerosis. Scientific Reports, 2013, 3, 3275.	1.6	41
70	Roles of Extracellular Chaperones in Amyloidosis. Journal of Molecular Biology, 2012, 421, 499-516.	2.0	55
71	Extracellular Chaperones. Topics in Current Chemistry, 2011, 328, 241-268.	4.0	24
72	Clusterin facilitates in vivo clearance of extracellular misfolded proteins. Cellular and Molecular Life Sciences, 2011, 68, 3919-3931.	2.4	111

#	Article	IF	Citations
73	Extracellular chaperones modulate the effects of Alzheimerâ€™s patient cerebrospinal fluid on AÎ²1-42 toxicity and uptake. Cell Stress and Chaperones, 2010, 15, 115-121.	1.2	51
74	Protein Chemistry of Amyloid Fibrils and Chaperones: Implications for Amyloid Formation and Disease. Current Chemical Biology, 2010, 4, 89-98.	0.2	3
75	Extracellular Chaperones. Topics in Current Chemistry, 2010, , 1.	4.0	1
76	ANS Binding Reveals Common Features of Cytotoxic Amyloid Species. ACS Chemical Biology, 2010, 5, 735-740.	1.6	335
77	Protein Chemistry of Amyloid Fibrils and Chaperones: Implications for Amyloid Formation and Disease. Current Chemical Biology, 2010, 4, 89-98.	0.2	8
78	Î±2-Macroglobulin and Haptoglobin Suppress Amyloid Formation by Interacting with Prefibrillar Protein Species. Journal of Biological Chemistry, 2009, 284, 4246-4254.	1.6	85
79	Structural Characterization of Clusterin-Chaperone Client Protein Complexes. Journal of Biological Chemistry, 2009, 284, 21920-21927.	1.6	70
80	Therapeutic Targets in Extracellular Protein Deposition Diseases. Current Medicinal Chemistry, 2009, 16, 2855-2866.	1.2	19
81	Chapter 6 The Chaperone Action of Clusterin and Its Putative Role in Quality Control of Extracellular Protein Folding. Advances in Cancer Research, 2009, 104, 89-114.	1.9	59
82	Clusterin Interacts with Paclitaxel and Confer Paclitaxel Resistance in Ovarian Cancer. Neoplasia, 2008, 10, 964-IN7.	2.3	50
83	Potential roles of abundant extracellular chaperones in the control of amyloid formation and toxicity. Molecular BioSystems, 2008, 4, 42-52.	2.9	124
84	Protease Activation of Î±₂-Macroglobulin Modulates a Chaperone-like Action with Broad Specificity. Biochemistry, 2008, 47, 1176-1185.	1.2	82
85	Extracellular Chaperones and Amyloids. , 2008, , 283-315.		6
86	The extracellular chaperone clusterin influences amyloid formation and toxicity by interacting with prefibrillar structures. FASEB Journal, 2007, 21, 2312-2322.	0.2	285
87	The Extracellular Chaperone Clusterin Potently Inhibits Human Lysozyme Amyloid Formation by Interacting with Prefibrillar Species. Journal of Molecular Biology, 2007, 369, 157-167.	2.0	84
88	Quality control of protein folding in extracellular space. EMBO Reports, 2005, 6, 1131-1136.	2.0	110
89	The Acute Phase Protein Haptoglobin Is a Mammalian Extracellular Chaperone with an Action Similar to Clusterinâ€. Biochemistry, 2005, 44, 10914-10925.	1.2	101