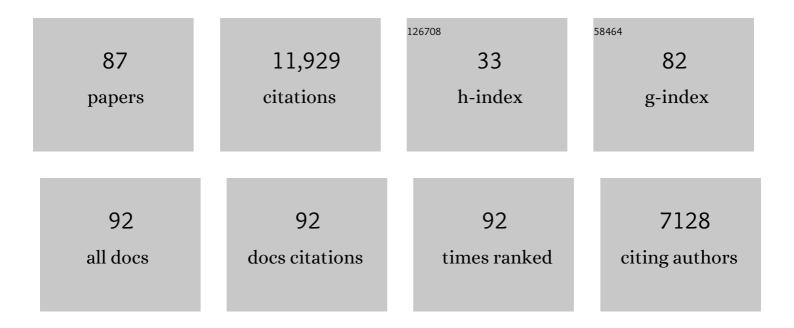
## David Westaway

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Prion-like strain effects in tauopathies. Cell and Tissue Research, 2023, 392, 179-199.	1.5	13
2	Distinct populations of highly potent TAU seed conformers in rapidly progressing Alzheimer's disease. Science Translational Medicine, 2022, 14, eabg0253.	5.8	26
3	Prion protein with a mutant N-terminal octarepeat region undergoes cobalamin-dependent assembly into high–molecular weight complexes. Journal of Biological Chemistry, 2022, 298, 101770.	1.6	4
4	Susceptibility of Beavers to Chronic Wasting Disease. Biology, 2022, 11, 667.	1.3	1
5	Investigating CRISPR/Cas9 gene drive for production of disease-preventing prion gene alleles. PLoS ONE, 2022, 17, e0269342.	1.1	2
6	Quaternary Structure Changes for PrPSc Predate PrPC Downregulation and Neuronal Death During Progression of Experimental Scrapie Disease. Molecular Neurobiology, 2021, 58, 375-390.	1.9	8
7	Murine models of tauopathies. , 2021, , 505-515.		0
8	Multisite interactions of prions with membranes and native nanodiscs. Chemistry and Physics of Lipids, 2021, 236, 105063.	1.5	11
9	Genetic Depletion of Amylin/Calcitonin Receptors Improves Memory and Learning in Transgenic Alzheimer's Disease Mouse Models. Molecular Neurobiology, 2021, 58, 5369-5382.	1.9	7
10	Pathologic tau conformer ensembles induce dynamic, liquid-liquid phase separation events at the nuclear envelope. BMC Biology, 2021, 19, 199.	1.7	23
11	Cellular Biology of Tau Diversity and Pathogenic Conformers. Frontiers in Neurology, 2020, 11, 590199.	1.1	12
12	Metabolomic study of disease progression in scrapie prion infected mice; validation of a novel method for brain metabolite extraction. Metabolomics, 2020, 16, 72.	1.4	2
13	Diverse, evolving conformer populations drive distinct phenotypes in frontotemporal lobar degeneration caused by the same MAPT-P301L mutation. Acta Neuropathologica, 2020, 139, 1045-1070.	3.9	17
14	Short amylin receptor antagonist peptides improve memory deficits in Alzheimer's disease mouse model. Scientific Reports, 2019, 9, 10942.	1.6	25
15	Nascent β Structure in the Elongated Hydrophobic Region of a Gerstmann–Strässler–Scheinker PrP Allele. Journal of Molecular Biology, 2019, 431, 2599-2611.	2.0	2
16	Proteasomal Inhibition Redirects the PrP-Like Shadoo Protein to the Nucleus. Molecular Neurobiology, 2019, 56, 7888-7904.	1.9	2
17	Application of high-throughput, capillary-based Western analysis to modulated cleavage of the cellular prion protein. Journal of Biological Chemistry, 2019, 294, 2642-5291.	1.6	20
18	Teaching case 3-2019: Are nuclear clefts or invaginations the niche of intranuclear inclusions in		2

FTLD-TDP?. , 2019, 38, 97-99.

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19	A novel Gerstmann-StrÃ <b>u</b> ssler-Scheinker disease mutation defines a precursor for amyloidogenic 8 kDa PrP fragments and reveals N-terminal structural changes shared by other GSS alleles. PLoS Pathogens, 2018, 14, e1006826.	2.1	16
20	Cyclic AC253, a novel amylin receptor antagonist, improves cognitive deficits in a mouse model of Alzheimer's disease. Alzheimer's and Dementia: Translational Research and Clinical Interventions, 2017, 3, 44-56.	1.8	24
21	Propagation of Misfolded Proteins in Neurodegeneration: Insights and Cautions from the Study of Prion Disease Prototypes. , 2017, , 203-223.		0
22	Role of microglial amylin receptors in mediating beta amyloid (Aβ)-induced inflammation. Journal of Neuroinflammation, 2017, 14, 199.	3.1	41
23	The CNS in inbred transgenic models of 4-repeat Tauopathy develops consistent tau seeding capacity yet focal and diverse patterns of protein deposition. Molecular Neurodegeneration, 2017, 12, 72.	4.4	14
24	Substitutions of PrP N-terminal histidine residues modulate scrapie disease pathogenesis and incubation time in transgenic mice. PLoS ONE, 2017, 12, e0188989.	1.1	11
25	Tollâ€like receptorâ€mediated immune response inhibits prion propagation. Clia, 2016, 64, 937-951.	2.5	18
26	Inhibition of the FKBP family of peptidyl prolyl isomerases induces abortive translocation and degradation of the cellular prion protein. Molecular Biology of the Cell, 2016, 27, 757-767.	0.9	21
27	A Common Phenotype Polymorphism in Mammalian Brains Defined by Concomitant Production of Prolactin and Growth Hormone. PLoS ONE, 2016, 11, e0149410.	1.1	3
28	Octarepeat region flexibility impacts prion function, endoproteolysis and disease manifestation. EMBO Molecular Medicine, 2015, 7, 339-356.	3.3	26
29	The Standard Scrapie Cell Assay: Development, Utility and Prospects. Viruses, 2015, 7, 180-198.	1.5	11
30	Interaction between Shadoo and PrP Affects the PrP-Folding Pathway. Journal of Virology, 2015, 89, 6287-6293.	1.5	15
31	Bioenergetic Mechanisms in Astrocytes May Contribute to Amyloid Plaque Deposition and Toxicity. Journal of Biological Chemistry, 2015, 290, 12504-12513.	1.6	63
32	Neuroprotective properties of the PrP-like Shadoo glycoprotein assessed in the middle cerebral artery occlusion model of ischemia. Prion, 2015, 9, 376-393.	0.9	4
33	Prion Infectivity Plateaus and Conversion to Symptomatic Disease Originate from Falling Precursor Levels and Increased Levels of Oligomeric PrP <sup>Sc</sup> Species. Journal of Virology, 2015, 89, 12418-12426.	1.5	33
34	Synergistic associations of catechol-O-methyltransferase and brain-derived neurotrophic factor with executive function in agingÂare selective and modified by apolipoprotein E. Neurobiology of Aging, 2015, 36, 249-256.	1.5	21
35	Basic Prion Science "Spreads―Insight. PLoS Pathogens, 2015, 11, e1005092.	2.1	0
36	IDE (rs6583817) polymorphism and pulse pressure are independently and interactively associated with level and change in executive function in older adults Psychology and Aging, 2014, 29, 418-430.	1.4	26

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37	Endoproteolytic processing of the mammalian prion glycoprotein family. FEBS Journal, 2014, 281, 862-876.	2.2	25
38	APOE and COMT polymorphisms are complementary biomarkers of status, stability, and transitions in normal aging and early mild cognitive impairment. Frontiers in Aging Neuroscience, 2014, 6, 236.	1.7	32
39	Prion disease tempo determined by host-dependent substrate reduction. Journal of Clinical Investigation, 2014, 124, 847-858.	3.9	59
40	Infectious Prions Accumulate to High Levels in Non Proliferative C2C12 Myotubes. PLoS Pathogens, 2013, 9, e1003755.	2.1	21
41	The Prion Protein Modulates A-type K+ Currents Mediated by Kv4.2 Complexes through Dipeptidyl Aminopeptidase-like Protein 6. Journal of Biological Chemistry, 2013, 288, 37241-37255.	1.6	25
42	The P's and Q's of cellular PrP-Al <sup>2</sup> interactions. Prion, 2012, 6, 359-363.	0.9	10
43	Shadoo/PrP (Sprn0/0/Prnp0/0) double knockout mice. Prion, 2012, 6, 420-424.	0.9	9
44	Knockout of the prion protein (PrP)-like <i>Sprn</i> gene does not produce embryonic lethality in combination with PrP <sup>C</sup> -deficiency. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 9035-9040.	3.3	58
45	Beta Amyloid-Induced Depression of Hippocampal Long-Term Potentiation Is Mediated through the Amylin Receptor. Journal of Neuroscience, 2012, 32, 17401-17406.	1.7	58
46	RGG Repeats of PrP-like Shadoo Protein Bind Nucleic Acids. Biochemistry, 2012, 51, 9029-9031.	1.2	12
47	LIV-1 ZIP Ectodomain Shedding in Prion-Infected Mice Resembles Cellular Response to Transition Metal Starvation. Journal of Molecular Biology, 2012, 422, 556-574.	2.0	32
48	The isotropic fractionator provides evidence for differential loss of hippocampal neurons in two mouse models of Alzheimer's disease. Molecular Neurodegeneration, 2012, 7, 58.	4.4	28
49	Prion inhibition with multivalent PrPSc binding compounds. Biomaterials, 2012, 33, 6808-6822.	5.7	15
50	Actions of β-Amyloid Protein on Human Neurons Are Expressed through the Amylin Receptor. American Journal of Pathology, 2011, 178, 140-149.	1.9	73
51	Family reunion – The ZIP/prion gene family. Progress in Neurobiology, 2011, 93, 405-420.	2.8	33
52	Biological properties of the PrP-like Shadoo protein. Frontiers in Bioscience - Landmark, 2011, 16, 1505.	3.0	19
53	The PrP-Like Proteins Shadoo and Doppel. Topics in Current Chemistry, 2011, 305, 225-256.	4.0	29
54	Aβ Inhibition of Ionic Conductance in Mouse Basal Forebrain Neurons Is Dependent upon the Cellular Prion Protein PrP <sup>C</sup> . Journal of Neuroscience, 2011, 31, 16292-16297.	1.7	30

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55	Down-Regulation of Shadoo in Prion Infections Traces a Pre-Clinical Event Inversely Related to PrPSc Accumulation. PLoS Pathogens, 2011, 7, e1002391.	2.1	34
56	lonic mechanisms of action of prion protein fragment PrP(106–126) in rat basal forebrain neurons. Journal of Neuroscience Research, 2010, 88, 2217-2227.	1.3	11
57	Wildâ€ŧype Shadoo proteins convert to amyloidâ€ŀike forms under native conditions. Journal of Neurochemistry, 2010, 113, 92-104.	2.1	27
58	Prionet Canada: A Network of Centres of Excellence for Research into Prions and Prion Diseases. Journal of Toxicology and Environmental Health - Part A: Current Issues, 2009, 72, 1000-1007.	1.1	1
59	Interactome Analyses Identify Ties of PrPC and Its Mammalian Paralogs to Oligomannosidic N-Glycans and Endoplasmic Reticulum-Derived Chaperones. PLoS Pathogens, 2009, 5, e1000608.	2.1	108
60	Frequent Missense and Insertion/Deletion Polymorphisms in the Ovine Shadoo Gene Parallel Species-Specific Variation in PrP. PLoS ONE, 2009, 4, e6538.	1.1	13
61	Evolutionary Descent of Prion Genes from the ZIP Family of Metal Ion Transporters. PLoS ONE, 2009, 4, e7208.	1.1	108
62	The in Vivo Brain Interactome of the Amyloid Precursor Protein. Molecular and Cellular Proteomics, 2008, 7, 15-34.	2.5	143
63	The prion protein family: Diversity, rivalry, and dysfunction. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2007, 1772, 654-672.	1.8	102
64	The CNS glycoprotein Shadoo has PrPC-like protective properties and displays reduced levels in prion infections. EMBO Journal, 2007, 26, 4038-4050.	3.5	114
65	Cortical Neuronal and Glial Pathology in TgTauP301L Transgenic Mice. American Journal of Pathology, 2006, 169, 1365-1375.	1.9	68
66	The Expanding Universe of Prion Diseases. PLoS Pathogens, 2006, 2, e26.	2.1	115
67	Genetic Mapping of Activity Determinants within Cellular Prion Proteins. Journal of Biological Chemistry, 2004, 279, 55443-55454.	1.6	55
68	The PrP-like Protein Doppel Binds Copper. Journal of Biological Chemistry, 2003, 278, 8888-8896.	1.6	39
69	Mouse models of Alzheimer's disease: The long and filamentous road. Neurological Research, 2003, 25, 590-600.	0.6	49
70	Mapping Cu(II) Binding Sites in Prion Proteins by Diethyl Pyrocarbonate Modification and Matrix-assisted Laser Desorption Ionization-Time of Flight (MALDI-TOF) Mass Spectrometric Footprinting. Journal of Biological Chemistry, 2002, 277, 1981-1990.	1.6	139
71	A cluster of familial Creutzfeldt-Jakob disease mutations recapitulate conserved residues in Doppel: a case of molecular mimicry?. FEBS Letters, 2002, 532, 21-26.	1.3	4
72	Mammalian prion proteins: enigma, variation and vaccination. Trends in Biochemical Sciences, 2002, 27, 301-307.	3.7	25

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73	Early-onset Amyloid Deposition and Cognitive Deficits in Transgenic Mice Expressing a Double Mutant Form of Amyloid Precursor Protein 695. Journal of Biological Chemistry, 2001, 276, 21562-21570.	1.6	820
74	Examining the zinc binding site of the amyloid- $\hat{l}^2$ peptide. FEBS Journal, 2000, 267, 6692-6698.	0.2	117
75	Aβ peptide immunization reduces behavioural impairment and plaques in a model of Alzheimer's disease. Nature, 2000, 408, 979-982.	13.7	1,472
76	Copper(II)-induced Conformational Changes and Protease Resistance in Recombinant and Cellular PrP. Journal of Biological Chemistry, 2000, 275, 19121-19131.	1.6	144
77	Doppel Is an N-Glycosylated, Glycosylphosphatidylinositol-anchored Protein. Journal of Biological Chemistry, 2000, 275, 26834-26841.	1.6	124
78	Ataxia in prion protein (PrP)-deficient mice is associated with upregulation of the novel PrP-like protein doppel. Journal of Molecular Biology, 1999, 292, 797-817.	2.0	479
79	Mutant presenilins of Alzheimer's disease increase production of 42-residue amyloid β-protein in both transfected cells and transgenic mice. Nature Medicine, 1997, 3, 67-72.	15.2	1,271
80	The cellular prion protein binds copper in vivo. Nature, 1997, 390, 684-687.	13.7	1,170
81	Degeneration of skeletal muscle, peripheral nerves, and the central nervous system in transgenic mice overexpressing wild-type prion proteins. Cell, 1994, 76, 117-129.	13.5	335
82	Paradoxical shortening of scrapie incubation times by expression of prion protein transgenes derived from long incubation period mice. Neuron, 1991, 7, 59-68.	3.8	139
83	Transgenetic studies implicate interactions between homologous PrP isoforms in scrapie prion replication. Cell, 1990, 63, 673-686.	13.5	877
84	Distinct prion proteins in short and long scrapie incubation period mice. Cell, 1987, 51, 651-662.	13.5	493
85	Molecular Cloning of a Human Prion Protein cDNA. DNA and Cell Biology, 1986, 5, 315-324.	5.1	331
86	Linkage of prion protein and scrapie incubation time genes. Cell, 1986, 46, 503-511.	13.5	416
87	A cellular gene encodes scrapie PrP 27-30 protein. Cell, 1985, 40, 735-746.	13.5	1,490