

John H Viles

List of Publications by Year in descending order

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Version: 2024-02-01

50
papers

4,719
citations

126858

33
h-index

189801

50
g-index

51
all docs

51
docs citations

51
times ranked

4897
citing authors

#	ARTICLE	IF	CITATIONS
1	Cross-seeding of WT amyloid- β 2 with Arctic but not Italian familial mutants accelerates fibril formation in Alzheimer's disease. <i>Journal of Biological Chemistry</i> , 2022, 298, 102071.	1.6	8
2	Therapeutic potential for amyloid surface inhibitor: only amyloid- β oligomers formed by secondary nucleation disrupt lipid membrane integrity. <i>FEBS Journal</i> , 2022, 289, 6767-6781.	2.2	7
3	3D-visualization of amyloid- β oligomer interactions with lipid membranes by cryo-electron tomography. <i>Chemical Science</i> , 2021, 12, 6896-6907.	3.7	31
4	Copper ²⁺ Binding to β -Synuclein. Histidine50 Can Form a Ternary Complex with Cu ²⁺ at the N-Terminus but Not a Macrochelate. <i>Inorganic Chemistry</i> , 2019, 58, 15580-15589.	1.9	9
5	Amyloid- β oligomers have a profound detergent-like effect on lipid membrane bilayers, imaged by atomic force and electron microscopy. <i>Journal of Biological Chemistry</i> , 2019, 294, 7566-7572.	1.6	112
6	Serum Albumin's Protective Inhibition of Amyloid- β Fiber Formation Is Suppressed by Cholesterol, Fatty Acids and Warfarin. <i>Journal of Molecular Biology</i> , 2018, 430, 919-934.	2.0	24
7	Copper Redox Cycling Inhibits A β 2 Fibre Formation and Promotes Fibre Fragmentation, while Generating a Dityrosine A β 2 Dimer. <i>Scientific Reports</i> , 2018, 8, 16190.	1.6	46
8	Prion protein stabilizes amyloid- β (A β) oligomers and enhances A β neurotoxicity in a Drosophila model of Alzheimer's disease. <i>Journal of Biological Chemistry</i> , 2018, 293, 13090-13099.	1.6	29
9	N-terminally Truncated Amyloid- β (11-40/42) Cofibrillizes with its Full-length Counterpart: Implications for Alzheimer's Disease. <i>Angewandte Chemie - International Edition</i> , 2017, 56, 9816-9819.	7.2	25
10	Ion Channel Formation by Amyloid- β 242 Oligomers but Not Amyloid- β 240 in Cellular Membranes. <i>Journal of Biological Chemistry</i> , 2017, 292, 1404-1413.	1.6	159
11	N-terminally Truncated Amyloid- β (11-40/42) Cofibrillizes with its Full-length Counterpart: Implications for Alzheimer's Disease. <i>Angewandte Chemie</i> , 2017, 129, 9948-9951.	1.6	7
12	Endocytosis of the tachykinin neuropeptide, neurokinin B, in astrocytes and its role in cellular copper uptake. <i>Journal of Inorganic Biochemistry</i> , 2016, 162, 319-325.	1.5	13
13	The Rapid Exchange of Zinc ²⁺ Enables Trace Levels to Profoundly Influence Amyloid- β Misfolding and Dominates Assembly Outcomes in Cu ²⁺ /Zn ²⁺ Mixtures. <i>Journal of Molecular Biology</i> , 2016, 428, 2832-2846.	2.0	25
14	Methionine oxidation reduces lag-times for amyloid- β (1-40) fiber formation but generates highly fragmented fibers. <i>Biochimica Et Biophysica Acta - Proteins and Proteomics</i> , 2016, 1864, 1260-1269.	1.1	20
15	A Comparison of Three Fluorophores for the Detection of Amyloid Fibers and Prefibrillar Oligomeric Assemblies. ThT (Thioflavin T); ANS (1-Anilinonaphthalene-8-sulfonic Acid); and bisANS (4,4'-Dianilino-1,1'-binaphthyl-5,5'-disulfonic Acid). <i>Biochemistry</i> , 2015, 54, 4297-4306.	1.2	143
16	Cu ²⁺ accentuates distinct misfolding of A β (1-40) and A β (1-42) peptides, and potentiates membrane disruption. <i>Biochemical Journal</i> , 2015, 466, 233-242.	1.7	56
17	Amyloid β Protein and Alzheimer's Disease: When Computer Simulations Complement Experimental Studies. <i>Chemical Reviews</i> , 2015, 115, 3518-3563.	23.0	530
18	Truncated Amyloid- β (11-40/42) from Alzheimer Disease Binds Cu ²⁺ with a Femtomolar Affinity and Influences Fiber Assembly. <i>Journal of Biological Chemistry</i> , 2015, 290, 27791-27802.	1.6	53

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19	Structural analysis of the starfish SALMFamide neuropeptides S1 and S2: The N-terminal region of S2 facilitates self-association. <i>Biochimica Et Biophysica Acta - Proteins and Proteomics</i> , 2014, 1844, 358-365.	1.1	6
20	Developing predictive rules for coordination geometry from visible circular dichroism of copper(II) and nickel(II) ions in histidine and amide main-chain complexes. <i>FEBS Journal</i> , 2014, 281, 3945-3954.	2.2	35
21	Bioactivity and structural properties of chimeric analogs of the starfish SALMFamide neuropeptides S1 and S2. <i>Biochimica Et Biophysica Acta - Proteins and Proteomics</i> , 2014, 1844, 1842-1850.	1.1	4
22	Copper(II) Sequentially Loads onto the N-Terminal Amino Group of the Cellular Prion Protein before the Individual Octarepeats. <i>Biochemistry</i> , 2014, 53, 3934-3939.	1.2	15
23	The cellular prion protein traps Alzheimer's A β in an oligomeric form and disassembles amyloid fibers. <i>FASEB Journal</i> , 2013, 27, 1847-1858.	0.2	89
24	Human Serum Albumin Can Regulate Amyloid- β Peptide Fiber Growth in the Brain Interstitium. <i>Journal of Biological Chemistry</i> , 2012, 287, 28163-28168.	1.6	134
25	Methionine Oxidation Perturbs the Structural Core of the Prion Protein and Suggests a Generic Misfolding Pathway. <i>Journal of Biological Chemistry</i> , 2012, 287, 28263-28275.	1.6	48
26	Metal ions and amyloid fiber formation in neurodegenerative diseases. Copper, zinc and iron in Alzheimer's, Parkinson's and prion diseases. <i>Coordination Chemistry Reviews</i> , 2012, 256, 2271-2284.	9.5	339
27	The Synucleins Are a Family of Redox-Active Copper Binding Proteins. <i>Biochemistry</i> , 2011, 50, 37-47.	1.2	66
28	Copper(II)-Induced Secondary Structure Changes and Reduced Folding Stability of the Prion Protein. <i>Journal of Molecular Biology</i> , 2011, 410, 369-382.	2.0	52
29	Substoichiometric Levels of Cu ²⁺ Ions Accelerate the Kinetics of Fiber Formation and Promote Cell Toxicity of Amyloid- β from Alzheimer Disease. <i>Journal of Biological Chemistry</i> , 2010, 285, 41533-41540.	1.6	174
30	Dynamics of a truncated prion protein, PrP(113-231), from ¹⁵ N NMR relaxation: Order parameters calculated and slow conformational fluctuations localized to a distinct region. <i>Protein Science</i> , 2009, 18, 410-423.	3.1	28
31	Evaluation of Copper ²⁺ Affinities for the Prion Protein. <i>Biochemistry</i> , 2009, 48, 8929-8931.	1.2	40
32	Copper(II) Binding to Amyloid- β Fibrils of Alzheimer's Disease Reveals a Picomolar Affinity: Stoichiometry and Coordination Geometry Are Independent of A β Oligomeric Form. <i>Biochemistry</i> , 2009, 48, 4388-4402.	1.2	198
33	Amyloid β -Cu ²⁺ Complexes in both Monomeric and Fibrillar Forms Do Not Generate H ₂ O ₂ Catalytically but Quench Hydroxyl Radicals. <i>Biochemistry</i> , 2008, 47, 11653-11664.	1.2	113
34	Manganese Binding to the Prion Protein. <i>Journal of Biological Chemistry</i> , 2008, 283, 12831-12839.	1.6	90
35	Deconvoluting the Cu ²⁺ Binding Modes of Full-length Prion Protein*. <i>Journal of Biological Chemistry</i> , 2008, 283, 1870-1881.	1.6	97
36	Copper and the structural biology of the prion protein. <i>Biochemical Society Transactions</i> , 2008, 36, 1288-1292.	1.6	68

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37	NMR characterization of the pH 4 β^2 -intermediate of the prion protein: the N-terminal half of the protein remains unstructured and retains a high degree of flexibility. <i>Biochemical Journal</i> , 2007, 401, 533-540.	1.7	36
38	Empirical rules for rationalising visible circular dichroism of Cu ²⁺ and Ni ²⁺ -histidine complexes: Applications to the prion protein. <i>FEBS Letters</i> , 2007, 581, 1430-1434.	1.3	61
39	Fragment length influences affinity for Cu ²⁺ and Ni ²⁺ binding to His96 or His111 of the prion protein and spectroscopic evidence for a multiple histidine binding only at low pH. <i>Biochemical Journal</i> , 2007, 404, 393-402.	1.7	69
40	Prion protein does not redox-silence Cu ²⁺ , but is a sacrificial quencher of hydroxyl radicals. <i>Free Radical Biology and Medicine</i> , 2007, 42, 79-89.	1.3	74
41	A survey of diamagnetic probes for copper ²⁺ binding to the prion protein. 1H NMR solution structure of the palladium ²⁺ bound single octarepeat. <i>Dalton Transactions</i> , 2006, , 509-518.	1.6	25
42	Solution 1H NMR investigation of Zn ²⁺ and Cd ²⁺ binding to amyloid-beta peptide (A β^2) of Alzheimer's disease. <i>Biochimica Et Biophysica Acta - Proteins and Proteomics</i> , 2006, 1764, 246-256.	1.1	154
43	Probing Copper ²⁺ Binding to the Prion Protein Using Diamagnetic Nickel ²⁺ and 1H NMR: The Unstructured N terminus Facilitates the Coordination of Six Copper ²⁺ Ions at Physiological Concentrations. <i>Journal of Molecular Biology</i> , 2005, 346, 1393-1407.	2.0	148
44	Preferential Cu ²⁺ Coordination by His96 and His111 Induces β^2 -Sheet Formation in the Unstructured Amyloidogenic Region of the Prion Protein. <i>Journal of Biological Chemistry</i> , 2004, 279, 32018-32027.	1.6	218
45	Copper Binding to the Amyloid- β^2 (A β^2) Peptide Associated with Alzheimer's Disease. <i>Journal of Biological Chemistry</i> , 2004, 279, 18169-18177.	1.6	414
46	Copper Binding to the Octarepeats of the Prion Protein. <i>Journal of Biological Chemistry</i> , 2003, 278, 6795-6802.	1.6	191
47	Local Structural Plasticity of the Prion Protein. Analysis of NMR Relaxation Dynamics. <i>Biochemistry</i> , 2001, 40, 2743-2753.	1.2	171
48	Potential bias in NMR relaxation data introduced by peak intensity analysis and curve fitting methods. <i>Journal of Biomolecular NMR</i> , 2001, 21, 1-9.	1.6	44
49	Involvement of a lysine residue in the N-terminal Ni ²⁺ and Cu ²⁺ binding site of serum albumins. Comparison with Co ²⁺ , Cd ²⁺ and Al ³⁺ . <i>FEBS Journal</i> , 1994, 220, 193-200.	0.2	184
50	Direct detection of albumin in human blood plasma by proton NMR spectroscopy. Complexation of nickel ²⁺ . <i>Journal of the American Chemical Society</i> , 1993, 115, 9285-9286.	6.6	36