## Michele Vendruscolo

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Protein Misfolding, Functional Amyloid, and Human Disease. Annual Review of Biochemistry, 2006, 75, 333-366.	5.0	5,737
2	Protein folding and misfolding. Nature, 2003, 426, 884-890.	13.7	4,210
3	Inherent toxicity of aggregates implies a common mechanism for protein misfolding diseases. Nature, 2002, 416, 507-511.	13.7	2,322
4	Protein Misfolding, Amyloid Formation, and Human Disease: A Summary of Progress Over the Last Decade. Annual Review of Biochemistry, 2017, 86, 27-68.	5.0	1,929
5	The amyloid state and its association with protein misfolding diseases. Nature Reviews Molecular Cell Biology, 2014, 15, 384-396.	16.1	1,894
6	Protein misfolding, evolution and disease. Trends in Biochemical Sciences, 1999, 24, 329-332.	3.7	1,858
7	Protein aggregation and aggregate toxicity: new insights into protein folding, misfolding diseases and biological evolution. Journal of Molecular Medicine, 2003, 81, 678-699.	1.7	1,444
8	Proliferation of amyloid-β42 aggregates occurs through a secondary nucleation mechanism. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 9758-9763.	3.3	1,162
9	Instability, unfolding and aggregation of human lysozyme variants underlying amyloid fibrillogenesis. Nature, 1997, 385, 787-793.	13.7	1,061
10	Rationalization of the effects of mutations on peptide andprotein aggregation rates. Nature, 2003, 424, 805-808.	13.7	1,013
11	An Analytical Solution to the Kinetics of Breakable Filament Assembly. Science, 2009, 326, 1533-1537.	6.0	970
12	Hydrodynamic Radii of Native and Denatured Proteins Measured by Pulse Field Gradient NMR Techniquesâ€. Biochemistry, 1999, 38, 16424-16431.	1.2	886
13	Chemical space and biology. Nature, 2004, 432, 824-828.	13.7	862
14	The structural basis of protein folding and its links with human disease. Philosophical Transactions of the Royal Society B: Biological Sciences, 2001, 356, 133-145.	1.8	828
15	Amyloid fibrils from muscle myoglobin. Nature, 2001, 410, 165-166.	13.7	791
16	Parmbsc1: a refined force field for DNA simulations. Nature Methods, 2016, 13, 55-58.	9.0	790
17	Protein Folding: A Perspective from Theory and Experiment. Angewandte Chemie - International Edition, 1998, 37, 868-893.	7.2	778
18	Principles of protein folding, misfolding and aggregation. Seminars in Cell and Developmental Biology, 2004, 15, 3-16.	2.3	772

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19	The folding of hen lysozyme involves partially structured intermediates and multiple pathways. Nature, 1992, 358, 302-307.	13.7	771
20	The protofilament structure of insulin amyloid fibrils. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 9196-9201.	3.3	770
21	Direct Observation of the Interconversion of Normal and Toxic Forms of α-Synuclein. Cell, 2012, 149, 1048-1059.	13.5	755
22	Amyloid formation by globular proteins under native conditions. Nature Chemical Biology, 2009, 5, 15-22.	3.9	746
23	ALS/FTD Mutation-Induced Phase Transition of FUS Liquid Droplets and Reversible Hydrogels into Irreversible Hydrogels Impairs RNP Granule Function. Neuron, 2015, 88, 678-690.	3.8	716
24	Role of Intermolecular Forces in Defining Material Properties of Protein Nanofibrils. Science, 2007, 318, 1900-1903.	6.0	694
25	FUS Phase Separation Is Modulated by a Molecular Chaperone and Methylation of Arginine Cation-Ï€ Interactions. Cell, 2018, 173, 720-734.e15.	13.5	662
26	Mapping Long-Range Interactions in α-Synuclein using Spin-Label NMR and Ensemble Molecular Dynamics Simulations. Journal of the American Chemical Society, 2005, 127, 476-477.	6.6	658
27	Simultaneous determination of protein structure and dynamics. Nature, 2005, 433, 128-132.	13.7	641
28	Amyloid-like Aggregates Sequester Numerous Metastable Proteins with Essential Cellular Functions. Cell, 2011, 144, 67-78.	13.5	604
29	Long-Range Interactions Within a Nonnative Protein. Science, 2002, 295, 1719-1722.	6.0	600
30	Characterization of the nanoscale properties of individual amyloid fibrils. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 15806-15811.	3.3	579
31	Prediction of "Aggregation-prone―and "Aggregation-susceptible―Regions in Proteins Associated with Neurodegenerative Diseases. Journal of Molecular Biology, 2005, 350, 379-392.	2.0	557
32	Solution conditions determine the relative importance of nucleation and growth processes in α-synuclein aggregation. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, 7671-7676.	3.3	546
33	Molecular mechanisms of protein aggregation from global fitting of kinetic models. Nature Protocols, 2016, 11, 252-272.	5.5	546
34	Lipid vesicles trigger α-synuclein aggregation by stimulating primary nucleation. Nature Chemical Biology, 2015, 11, 229-234.	3.9	532
35	Characterization of a partly folded protein by NMR methods: studies on the molten globule state of guinea pig .alphalactalbumin. Biochemistry, 1989, 28, 7-13.	1.2	504
36	Protein structure determination from NMR chemical shifts. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 9615-9620.	3.3	499

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37	A causative link between the structure of aberrant protein oligomers and their toxicity. Nature Chemical Biology, 2010, 6, 140-147.	3.9	499
38	High-resolution molecular structure of a peptide in an amyloid fibril determined by magic angle spinning NMR spectroscopy. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 711-716.	3.3	495
39	Structural basis of membrane disruption and cellular toxicity by α-synuclein oligomers. Science, 2017, 358, 1440-1443.	6.0	492
40	Cryo-electron microscopy structure of an SH3 amyloid fibril and model of the molecular packing. EMBO Journal, 1999, 18, 815-821.	3.5	487
41	Atomic structure and hierarchical assembly of a cross-β amyloid fibril. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 5468-5473.	3.3	479
42	Widespread Proteome Remodeling and Aggregation in Aging C.Âelegans. Cell, 2015, 161, 919-932.	13.5	478
43	The Amyloid-β Pathway in Alzheimer's Disease. Molecular Psychiatry, 2021, 26, 5481-5503.	4.1	478
44	The behaviour of polyamino acids reveals an inverse side chain effect in amyloid structure formation. EMBO Journal, 2002, 21, 5682-5690.	3.5	475
45	Understanding protein folding via free-energy surfaces from theory and experiment. Trends in Biochemical Sciences, 2000, 25, 331-339.	3.7	461
46	Formation of insulin amyloid fibrils followed by FTIR simultaneously with CD and electron microscopy. Protein Science, 2000, 9, 1960-1967.	3.1	453
47	Low-populated folding intermediates of Fyn SH3 characterized by relaxation dispersion NMR. Nature, 2004, 430, 586-590.	13.7	445
48	Three key residues form a critical contact network in a protein folding transition state. Nature, 2001, 409, 641-645.	13.7	423
49	Prediction of Aggregation-Prone Regions in Structured Proteins. Journal of Molecular Biology, 2008, 380, 425-436.	2.0	420
50	From Macroscopic Measurements to Microscopic Mechanisms of Protein Aggregation. Journal of Molecular Biology, 2012, 421, 160-171.	2.0	407
51	Differences in nucleation behavior underlie the contrasting aggregation kinetics of the AÎ <sup>2</sup> 40 and AÎ <sup>2</sup> 42 peptides. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, 9384-9389.	3.3	405
52	Formation and seeding of amyloid fibrils from wild-type hen lysozyme and a peptide fragment from the β-domain. Journal of Molecular Biology, 2000, 300, 541-549.	2.0	395
53	Structural characterization of toxic oligomers that are kinetically trapped during α-synuclein fibril formation. Proceedings of the National Academy of Sciences of the United States of America, 2015, 112, E1994-2003.	3.3	384
54	De novo designed peptide-based amyloid fibrils. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 16052-16057.	3.3	381

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55	Analysis of Main Chain Torsion Angles in Proteins: Prediction of NMR Coupling Constants for Native and Random Coil Conformations. Journal of Molecular Biology, 1996, 255, 494-506.	2.0	379
56	The fundamentals of protein folding: bringing together theory and experiment. Current Opinion in Structural Biology, 1999, 9, 92-101.	2.6	375
57	Kinetic partitioning of protein folding and aggregation. Nature Structural Biology, 2002, 9, 137-143.	9.7	373
58	A molecular chaperone breaks the catalytic cycle that generates toxic AÎ <sup>2</sup> oligomers. Nature Structural and Molecular Biology, 2015, 22, 207-213.	3.6	373
59	Metastability of Native Proteins and the Phenomenon of Amyloid Formation. Journal of the American Chemical Society, 2011, 133, 14160-14163.	6.6	369
60	Direct observation of the three regions in α-synuclein that determine its membrane-bound behaviour. Nature Communications, 2014, 5, 3827.	5.8	357
61	Prefibrillar Amyloid Protein Aggregates Share Common Features of Cytotoxicity. Journal of Biological Chemistry, 2004, 279, 31374-31382.	1.6	346
62	Multiple Tight Phospholipid-Binding Modes of α-Synuclein Revealed by Solution NMR Spectroscopy. Journal of Molecular Biology, 2009, 390, 775-790.	2.0	345
63	Half a century of amyloids: past, present and future. Chemical Society Reviews, 2020, 49, 5473-5509.	18.7	345
64	Molecular recycling within amyloid fibrils. Nature, 2005, 436, 554-558.	13.7	342
65	The CamSol Method of Rational Design of Protein Mutants with Enhanced Solubility. Journal of Molecular Biology, 2015, 427, 478-490.	2.0	341
66	Structure of an Intermediate State in Protein Folding and Aggregation. Science, 2012, 336, 362-366.	6.0	339
67	ANS Binding Reveals Common Features of Cytotoxic Amyloid Species. ACS Chemical Biology, 2010, 5, 735-740.	1.6	335
68	Protein-misfolding diseases: Getting out of shape. Nature, 2002, 418, 729-730.	13.7	334
69	RNA Granules Hitchhike on Lysosomes for Long-Distance Transport, Using Annexin A11 as a Molecular Tether. Cell, 2019, 179, 147-164.e20.	13.5	327
70	Determination of Secondary Structure Populations in Disordered States of Proteins Using Nuclear Magnetic Resonance Chemical Shifts. Biochemistry, 2012, 51, 2224-2231.	1.2	316
71	Prediction of the Absolute Aggregation Rates of Amyloidogenic Polypeptide Chains. Journal of Molecular Biology, 2004, 341, 1317-1326.	2.0	307
72	The Zyggregator method for predicting protein aggregation propensities. Chemical Society Reviews, 2008, 37, 1395.	18.7	307

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73	Stabilization of neurotoxic Alzheimer amyloid-β oligomers by protein engineering. Proceedings of the National Academy of Sciences of the United States of America, 2010, 107, 15595-15600.	3.3	304
74	The concept of a random coil: Residual structure in peptides and denatured proteins. Folding & Design, 1996, 1, R95-R106.	4.5	296
75	Structural and Dynamical Properties of a Denatured Protein. Heteronuclear 3D NMR Experiments and Theoretical Simulations of Lysozyme in 8 M Ureaâ€. Biochemistry, 1997, 36, 8977-8991.	1.2	296
76	The importance of sequence diversity in the aggregation and evolution of proteins. Nature, 2005, 438, 878-881.	13.7	291
77	The extracellular chaperone clusterin influences amyloid formation and toxicity by interacting with prefibrillar structures. FASEB Journal, 2007, 21, 2312-2322.	0.2	285
78	Principles of protein structural ensemble determination. Current Opinion in Structural Biology, 2017, 42, 106-116.	2.6	285
79	Myoglobin forms amyloid fibrils by association of unfolded polypeptide segments. Proceedings of the National Academy of Sciences of the United States of America, 2003, 100, 15463-15468.	3.3	270
80	Nucleated polymerization with secondary pathways. I. Time evolution of the principal moments. Journal of Chemical Physics, 2011, 135, 065105.	1.2	270
81	Studies of the aggregation of mutant proteins in vitro provide insights into the genetics of amyloid diseases. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 16419-16426.	3.3	268
82	Location of Aluminum in Substituted Calcium Silicate Hydrate (C-S-H) Gels as Determined by 29Si and 27Al NMR and EELS. Journal of the American Ceramic Society, 1993, 76, 2285-2288.	1.9	266
83	Life on the edge: a link between gene expression levels and aggregation rates of human proteins. Trends in Biochemical Sciences, 2007, 32, 204-206.	3.7	266
84	Alpha-Synuclein Oligomers Interact with Metal Ions to Induce Oxidative Stress and Neuronal Death in Parkinson's Disease. Antioxidants and Redox Signaling, 2016, 24, 376-391.	2.5	266
85	Demonstration by NMR of folding domains in lysozyme. Nature, 1991, 349, 633-636.	13.7	265
86	Characterization of the Oligomeric States of Insulin in Self-Assembly and Amyloid Fibril Formation by Mass Spectrometry. Biophysical Journal, 2000, 79, 1053-1065.	0.2	258
87	Mutational analysis of acylphosphatase suggests the importance of topology and contact order in protein folding. Nature Structural Biology, 1999, 6, 1005-1009.	9.7	257
88	Mutations associated with familial Parkinson's disease alter the initiation and amplification steps of α-synuclein aggregation. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, 10328-10333.	3.3	252
89	Molecular conformation of a peptide fragment of transthyretin in an amyloid fibril. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 16748-16753.	3.3	249
90	A Highly Amyloidogenic Region of Hen Lysozyme. Journal of Molecular Biology, 2004, 340, 1153-1165.	2.0	248

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91	Chemical properties of lipids strongly affect the kinetics of the membrane-induced aggregation of α-synuclein. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, 7065-7070.	3.3	248
92	Widespread Aggregation and Neurodegenerative Diseases Are Associated with Supersaturated Proteins. Cell Reports, 2013, 5, 781-790.	2.9	245
93	Short amino acid stretches can mediate amyloid formation in globular proteins: The Src homology 3 (SH3) case. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 7258-7263.	3.3	241
94	A residue-specific NMR view of the non-cooperative unfolding of a molten globule. Nature Structural Biology, 1997, 4, 630-634.	9.7	236
95	A kinetic study of β-lactoglobulin amyloid fibril formation promoted by urea. Protein Science, 2009, 11, 2417-2426.	3.1	233
96	The formation of spherulites by amyloid fibrils of bovine insulin. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 14420-14424.	3.3	232
97	The extracellular chaperone clusterin sequesters oligomeric forms of the amyloid-β1â^'40 peptide. Nature Structural and Molecular Biology, 2012, 19, 79-83.	3.6	232
98	A natural product inhibits the initiation of α-synuclein aggregation and suppresses its toxicity. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, E1009-E1017.	3.3	231
99	Recovery of protein structure from contact maps. Folding & Design, 1997, 2, 295-306.	4.5	230
100	A camelid antibody fragment inhibits the formation of amyloid fibrils by human lysozyme. Nature, 2003, 424, 783-788.	13.7	227
101	Heat Shock Protein 70 Inhibits α-Synuclein Fibril Formation via Preferential Binding to Prefibrillar Species. Journal of Biological Chemistry, 2005, 280, 14733-14740.	1.6	223
102	Fast and Accurate Predictions of Protein NMR Chemical Shifts from Interatomic Distances. Journal of the American Chemical Society, 2009, 131, 13894-13895.	6.6	223
103	Dynamics of oligomer populations formed during the aggregation of Alzheimer's Aβ42 peptide. Nature Chemistry, 2020, 12, 445-451.	6.6	223
104	Prefibrillar Amyloid Aggregates Could Be Generic Toxins in Higher Organisms. Journal of Neuroscience, 2006, 26, 8160-8167.	1.7	222
105	Conformation of GroEL-bound α-lactalbumin probed by mass spectrometry. Nature, 1994, 372, 646-651.	13.7	221
106	Kinetic analysis reveals the diversity of microscopic mechanisms through which molecular chaperones suppress amyloid formation. Nature Communications, 2016, 7, 10948.	5.8	219
107	The Role of Stable α-Synuclein Oligomers in the Molecular Events Underlying Amyloid Formation. Journal of the American Chemical Society, 2014, 136, 3859-3868.	6.6	218
108	Effects of α-tubulin acetylation on microtubule structure and stability. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 10366-10371.	3.3	216

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109	Structural Determinants of Protein Dynamics: Analysis of 15N NMR Relaxation Measurements for Main-Chain and Side-Chain Nuclei of Hen Egg White Lysozyme. Biochemistry, 1995, 34, 4041-4055.	1.2	211
110	Dependence on solution conditions of aggregation and amyloid formation by an SH3 domain. Journal of Molecular Biology, 2001, 311, 325-340.	2.0	208
111	Local cooperativity in the unfolding of an amyloidogenic variant of human lysozyme. Nature Structural Biology, 2002, 9, 308-315.	9.7	208
112	Differential Phospholipid Binding of α-Synuclein Variants Implicated in Parkinson's Disease Revealed by Solution NMR Spectroscopy. Biochemistry, 2010, 49, 862-871.	1.2	208
113	Following protein folding in real time using NMR spectroscopy. Nature Structural Biology, 1995, 2, 865-870.	9.7	206
114	Characterisation of protein unfolding by NMR diffusion measurements. Journal of Biomolecular NMR, 1997, 10, 199-203.	1.6	206
115	Structural basis of synaptic vesicle assembly promoted by α-synuclein. Nature Communications, 2016, 7, 12563.	5.8	203
116	Different Subdomains are Most Protected From Hydrogen Exchange in the Molten Globule and Native States of Human α-Lactalbumin. Journal of Molecular Biology, 1995, 253, 651-657.	2.0	200
117	Ostwald's rule of stages governs structural transitions and morphology of dipeptide supramolecular polymers. Nature Communications, 2014, 5, 5219.	5.8	197
118	pE-DB: a database of structural ensembles of intrinsically disordered and of unfolded proteins. Nucleic Acids Research, 2014, 42, D326-D335.	6.5	195
119	Structural Reorganisation and Potential Toxicity of Oligomeric Species Formed during the Assembly of Amyloid Fibrils. PLoS Computational Biology, 2007, 3, e173.	1.5	194
120	Quenched disorder, memory, and self-organization. Physical Review E, 1996, 53, R13-R16.	0.8	193
121	Amyloid Fibril Formation by Bovine Milk κ-Casein and Its Inhibition by the Molecular Chaperones αS- and β-Casein. Biochemistry, 2005, 44, 17027-17036.	1.2	193
122	Progressive Changes in the Structure of Hardened C3S Cement Pastes due to Carbonation. Journal of the American Ceramic Society, 1991, 74, 2891-2896.	1.9	191
123	Chemical kinetics for drug discovery to combat protein aggregation diseases. Trends in Pharmacological Sciences, 2014, 35, 127-135.	4.0	191
124	MobiDB 3.0: more annotations for intrinsic disorder, conformational diversity and interactions in proteins. Nucleic Acids Research, 2018, 46, D471-D476.	6.5	190
125	Determination of the Free Energy Landscape of α-Synuclein Using Spin Label Nuclear Magnetic Resonance Measurements. Journal of the American Chemical Society, 2009, 131, 18314-18326.	6.6	187
126	Kinetics and thermodynamics of amyloid formation from direct measurements of fluctuations in fibril mass. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 10016-10021.	3.3	186

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127	Cholesterol catalyses Al <sup>2</sup> 42 aggregation through a heterogeneous nucleation pathway in the presence of lipid membranes. Nature Chemistry, 2018, 10, 673-683.	6.6	186
128	Ultrastructural Organization of Amyloid Fibrils byAtomic Force Microscopy. Biophysical Journal, 2000, 79, 3282-3293.	0.2	185
129	α-Synuclein Senses Lipid Packing Defects and Induces Lateral Expansion of Lipids Leading to Membrane Remodeling. Journal of Biological Chemistry, 2013, 288, 20883-20895.	1.6	183
130	Protein amyloids develop an intrinsic fluorescence signature during aggregation. Analyst, The, 2013, 138, 2156.	1.7	182
131	Kinetic model of the aggregation of alpha-synuclein provides insights into prion-like spreading. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, E1206-15.	3.3	181
132	Proline isomerism in staphylococcal nuclease characterized by NMR and site-directed mutagenesis. Nature, 1987, 329, 266-268.	13.7	180
133	Functionalised amyloid fibrils for roles in cell adhesion. Biomaterials, 2008, 29, 1553-1562.	5.7	180
134	Direct characterization of amyloidogenic oligomers by single-molecule fluorescence. Proceedings of the United States of America, 2008, 105, 14424-14429.	3.3	180
135	Metainference: A Bayesian inference method for heterogeneous systems. Science Advances, 2016, 2, e1501177.	4.7	180
136	An anticancer drug suppresses the primary nucleation reaction that initiates the production of the toxic Aβ42 aggregates linked with Alzheimer's disease. Science Advances, 2016, 2, e1501244.	4.7	180
137	Systematic development of small molecules to inhibit specific microscopic steps of Aβ42 aggregation in Alzheimer's disease. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, E200-E208.	3.3	180
138	Widespread occurrence of the droplet state of proteins in the human proteome. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 33254-33262.	3.3	178
139	Investigation of protein folding by mass spectrometry. FASEB Journal, 1996, 10, 93-101.	0.2	175
140	Cucurbit[8]uril and Blue-Box: High-Energy Water Release Overwhelms Electrostatic Interactions. Journal of the American Chemical Society, 2013, 135, 14879-14888.	6.6	174
141	Systematic In Vivo Analysis of the Intrinsic Determinants of Amyloid Î <sup>2</sup> Pathogenicity. PLoS Biology, 2007, 5, e290.	2.6	171
142	Folding of a four-helix bundle: studies of acyl-coenzyme A binding protein. Biochemistry, 1995, 34, 7217-7224.	1.2	169
143	Molecular dynamics simulations with replica-averaged structural restraints generate structural ensembles according to the maximum entropy principle. Journal of Chemical Physics, 2013, 138, 094112.	1.2	169
144	Amyloid Fibril Formation by Lens Crystallin Proteins and Its Implications for Cataract Formation. Journal of Biological Chemistry, 2004, 279, 3413-3419.	1.6	166

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145	Exploring amyloid formation by a de novo design. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 4435-4440.	3.3	166
146	Nucleated polymerization with secondary pathways. II. Determination of self-consistent solutions to growth processes described by non-linear master equations. Journal of Chemical Physics, 2011, 135, 065106.	1.2	166
147	Toxicity of Protein Oligomers Is Rationalized by a Function Combining Size and Surface Hydrophobicity. ACS Chemical Biology, 2014, 9, 2309-2317.	1.6	166
148	Structure and Properties of a Complex of α-Synuclein and a Single-Domain Camelid Antibody. Journal of Molecular Biology, 2010, 402, 326-343.	2.0	164
149	Chemical Kinetics for Bridging Molecular Mechanisms and Macroscopic Measurements of Amyloid Fibril Formation. Annual Review of Physical Chemistry, 2018, 69, 273-298.	4.8	161
150	Toward a Description of the Conformations of Denatured States of Proteins. Comparison of a Random Coil Model with NMR Measurements. The Journal of Physical Chemistry, 1996, 100, 2661-2666.	2.9	160
151	Protein misfolding and disease: from the test tube to the organism. Current Opinion in Chemical Biology, 2008, 12, 25-31.	2.8	160
152	Intermolecular Structure Determination of Amyloid Fibrils with Magic-Angle Spinning and Dynamic Nuclear Polarization NMR. Journal of the American Chemical Society, 2011, 133, 13967-13974.	6.6	160
153	A refined solution structure of hen lysozyme determined using residual dipolar coupling data. Protein Science, 2001, 10, 677-688.	3.1	159
154	Direct Observation of Heterogeneous Amyloid Fibril Growth Kinetics via Two-Color Super-Resolution Microscopy. Nano Letters, 2014, 14, 339-345.	4.5	159
155	Interaction of the Molecular Chaperone DNAJB6 with Growing Amyloid-beta 42 (Aβ42) Aggregates Leads to Sub-stoichiometric Inhibition of Amyloid Formation. Journal of Biological Chemistry, 2014, 289, 31066-31076.	1.6	158
156	Formation of Mixed Fibrils Demonstrates the Generic Nature and Potential Utility of Amyloid Nanostructures. Journal of the American Chemical Society, 2000, 122, 12707-12713.	6.6	155
157	Determination of an Ensemble of Structures Representing the Denatured State of the Bovine Acyl-Coenzyme A Binding Protein. Journal of the American Chemical Society, 2004, 126, 3291-3299.	6.6	155
158	Protein Aggregation and Its Consequences for Human Disease. Protein and Peptide Letters, 2006, 13, 219-227.	0.4	154
159	A tau homeostasis signature is linked with the cellular and regional vulnerability of excitatory neurons to tau pathology. Nature Neuroscience, 2019, 22, 47-56.	7.1	154
160	The Folding Kinetics and Thermodynamics of the Fyn-SH3 Domainâ€. Biochemistry, 1998, 37, 2529-2537.	1.2	152
161	Multi-dimensional super-resolution imaging enables surface hydrophobicity mapping. Nature Communications, 2016, 7, 13544.	5.8	152
162	Rapid collapse and slow structural reorganisation during the refolding of bovine α-lactalbumin. Journal of Molecular Biology, 1999, 288, 673-688.	2.0	151

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163	In Situ Measurements of the Formation and Morphology of Intracellular β-Amyloid Fibrils by Super-Resolution Fluorescence Imaging. Journal of the American Chemical Society, 2011, 133, 12902-12905.	6.6	151
164	Physicochemical principles that regulate the competition between functional and dysfunctional association of proteins. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 10159-10164.	3.3	148
165	ALS mutations in FUS cause neuronal dysfunction and death in Caenorhabditis elegans by a dominant gain-of-function mechanism. Human Molecular Genetics, 2012, 21, 1-9.	1.4	148
166	Altered aggregation properties of mutant Î <sup>3</sup> -crystallins cause inherited cataract. EMBO Journal, 2002, 21, 6005-6014.	3.5	147
167	Supersaturation is a major driving force for protein aggregation in neurodegenerative diseases. Trends in Pharmacological Sciences, 2015, 36, 72-77.	4.0	147
168	A Labelâ€Free, Quantitative Assay of Amyloid Fibril Growth Based on Intrinsic Fluorescence. ChemBioChem, 2013, 14, 846-850.	1.3	145
169	Structural characterization of a highly–ordered â€~molten globule' at low pH. Nature Structural and Molecular Biology, 1994, 1, 23-29.	3.6	144
170	Binding of the Molecular Chaperone αB-Crystallin to Aβ Amyloid Fibrils Inhibits Fibril Elongation. Biophysical Journal, 2011, 101, 1681-1689.	0.2	143
171	Protein Aggregation in Crowded Environments. Journal of the American Chemical Society, 2010, 132, 5170-5175.	6.6	142
172	Amyloid Fibril Formation Can Proceed from Different Conformations of a Partially Unfolded Protein. Biophysical Journal, 2005, 89, 4201-4210.	0.2	141
173	Using NMR Chemical Shifts as Structural Restraints in Molecular Dynamics Simulations of Proteins. Structure, 2010, 18, 923-933.	1.6	141
174	The S/T-Rich Motif in the DNAJB6 Chaperone Delays Polyglutamine Aggregation and the Onset of Disease in a Mouse Model. Molecular Cell, 2016, 62, 272-283.	4.5	140
175	Different soluble aggregates of Aβ42 can give rise to cellular toxicity through different mechanisms. Nature Communications, 2019, 10, 1541.	5.8	140
176	Temperature dependent molecular motion of a tyrosine residue of ferrocytochromeC. FEBS Letters, 1976, 70, 96-100.	1.3	138
177	Molecular mechanisms used by chaperones to reduce the toxicity of aberrant protein oligomers. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 12479-12484.	3.3	137
178	Pairwise contact potentials are unsuitable for protein folding. Journal of Chemical Physics, 1998, 109, 11101-11108.	1.2	136
179	Relation between native ensembles and experimental structures of proteins. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 10901-10906.	3.3	136
180	The Interaction of αB-Crystallin with Mature α-Synuclein Amyloid Fibrils Inhibits Their Elongation. Biophysical Journal, 2010, 98, 843-851.	0.2	136

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181	Calcium is a key factor in α-synuclein induced neurotoxicity. Journal of Cell Science, 2016, 129, 1792-801.	1.2	136
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