Christopher M Dobson

List of Publications by Year
in descending order

226 papers	46,083 citations	4388 86 h-index	1980 206 g-index
240 all docs	240 docs citations	240 times ranked	27295 citing authors

#	Article	IF	Citations
1	Protein Misfolding, Functional Amyloid, and Human Disease. Annual Review of Biochemistry, 2006, 75, 333-366.	11.1	5,737
2	Protein folding and misfolding. Nature, 2003, 426, 884-890.	27.8	4,210
3	Inherent toxicity of aggregates implies a common mechanism for protein misfolding diseases. Nature, 2002, 416, 507-511.	27.8	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.	11.1	1,929
5	The amyloid state and its association with protein misfolding diseases. Nature Reviews Molecular Cell Biology, 2014, 15, 384-396.	37.0	1,894
6	Protein misfolding, evolution and disease. Trends in Biochemical Sciences, 1999, 24, 329-332.	7.5	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.	3.9	1,444
8	Proliferation of amyloid-Î²42 aggregates occurs through a secondary nucleation mechanism. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 9758-9763.	7.1	1,162
9	An Analytical Solution to the Kinetics of Breakable Filament Assembly. Science, 2009, 326, 1533-1537.	12.6	970
10	The protofilament structure of insulin amyloid fibrils. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 9196-9201.	7.1	770
11	Direct Observation of the Interconversion of Normal and Toxic Forms of Î±-Synuclein. Cell, 2012, 149, 1048-1059.	28.9	755
12	Mapping Long-Range Interactions in Î±-Synuclein using Spin-Label NMR and Ensemble Molecular Dynamics Simulations. Journal of the American Chemical Society, 2005, 127, 476-477.	13.7	658
13	Solution conditions determine the relative importance of nucleation and growth processes in Î±-synuclein aggregation. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, 7671-7676.	7.1	546
14	Molecular mechanisms of protein aggregation from global fitting of kinetic models. Nature Protocols, 2016, 11, 252-272.	12.0	546
15	Lipid vesicles trigger Î±-synuclein aggregation by stimulating primary nucleation. Nature Chemical Biology, 2015, 11, 229-234.	8.0	532
16	A causative link between the structure of aberrant protein oligomers and their toxicity. Nature Chemical Biology, 2010, 6, 140-147.	8.0	499
17	Structural basis of membrane disruption and cellular toxicity by Î±-synuclein oligomers. Science, 2017, 358, 1440-1443.	12.6	492
18	Atomic structure and hierarchical assembly of a cross-Î² amyloid fibril. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 5468-5473.	7.1	479

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19	Widespread Proteome Remodeling and Aggregation in Aging C.Âelegans. Cell, 2015, 161, 919-932.	28.9	478
20	From Macroscopic Measurements to Microscopic Mechanisms of Protein Aggregation. Journal of Molecular Biology, 2012, 421, 160-171.	4.2	407
21	Differences in nucleation behavior underlie the contrasting aggregation kinetics of the AÎ²40 and AÎ²42 peptides. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, 9384-9389.	7.1	405
22	Structural characterization of toxic oligomers that are kinetically trapped during Î±-synuclein fibril formation. Proceedings of the National Academy of Sciences of the United States of America, 2015, 112, E1994-2003.	7.1	384
23	A molecular chaperone breaks the catalytic cycle that generates toxic AÎ² oligomers. Nature Structural and Molecular Biology, 2015, 22, 207-213.	8.2	373
24	Metastability of Native Proteins and the Phenomenon of Amyloid Formation. Journal of the American Chemical Society, 2011, 133, 14160-14163.	13.7	369
25	Direct observation of the three regions in Î±-synuclein that determine its membrane-bound behaviour. Nature Communications, 2014, 5, 3827.	12.8	357
26	Multiple Tight Phospholipid-Binding Modes of Î±-Synuclein Revealed by Solution NMR Spectroscopy. Journal of Molecular Biology, 2009, 390, 775-790.	4.2	345
27	Half a century of amyloids: past, present and future. Chemical Society Reviews, 2020, 49, 5473-5509.	38.1	345
28	ANS Binding Reveals Common Features of Cytotoxic Amyloid Species. ACS Chemical Biology, 2010, 5, 735-740.	3.4	335
29	The extracellular chaperone clusterin influences amyloid formation and toxicity by interacting with prefibrillar structures. FASEB Journal, 2007, 21, 2312-2322.	0.5	285
30	Nucleated polymerization with secondary pathways. I. Time evolution of the principal moments. Journal of Chemical Physics, 2011, 135, 065105.	3.0	270
31	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.	5.4	266
32	Mutations associated with familial Parkinsonâ€™s disease alter the initiation and amplification steps of Î±-synuclein aggregation. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, 10328-10333.	7.1	252
33	Chemical properties of lipids strongly affect the kinetics of the membrane-induced aggregation of Î±-synuclein. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, 7065-7070.	7.1	248
34	Widespread Aggregation and Neurodegenerative Diseases Are Associated with Supersaturated Proteins. Cell Reports, 2013, 5, 781-790.	6.4	245
35	The extracellular chaperone clusterin sequesters oligomeric forms of the amyloid-Î²1â€“40 peptide. Nature Structural and Molecular Biology, 2012, 19, 79-83.	8.2	232
36	A natural product inhibits the initiation of Î±-synuclein aggregation and suppresses its toxicity. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, E1009-E1017.	7.1	231

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37	A camelid antibody fragment inhibits the formation of amyloid fibrils by human lysozyme. Nature, 2003, 424, 783-788.	27.8	227
38	Dynamics of oligomer populations formed during the aggregation of Alzheimerâ€™s AÎ²42 peptide. Nature Chemistry, 2020, 12, 445-451.	13.6	223
39	Prefibrillar Amyloid Aggregates Could Be Generic Toxins in Higher Organisms. Journal of Neuroscience, 2006, 26, 8160-8167.	3.6	222
40	Kinetic analysis reveals the diversity of microscopic mechanisms through which molecular chaperones suppress amyloid formation. Nature Communications, 2016, 7, 10948.	12.8	219
41	The Role of Stable Î±-Synuclein Oligomers in the Molecular Events Underlying Amyloid Formation. Journal of the American Chemical Society, 2014, 136, 3859-3868.	13.7	218
42	Differential Phospholipid Binding of Î±-Synuclein Variants Implicated in Parkinsonâ€™s Disease Revealed by Solution NMR Spectroscopy. Biochemistry, 2010, 49, 862-871.	2.5	208
43	Structural basis of synaptic vesicle assembly promoted by Î±-synuclein. Nature Communications, 2016, 7, 12563.	12.8	203
44	Structural Reorganisation and Potential Toxicity of Oligomeric Species Formed during the Assembly of Amyloid Fibrils. PLoS Computational Biology, 2007, 3, e173.	3.2	194
45	Chemical kinetics for drug discovery to combat protein aggregation diseases. Trends in Pharmacological Sciences, 2014, 35, 127-135.	8.7	191
46	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.	7.1	186
47	Cholesterol catalyses AÎ²42 aggregation through a heterogeneous nucleation pathway in the presence of lipid membranes. Nature Chemistry, 2018, 10, 673-683.	13.6	186
48	Î±-Synuclein Senses Lipid Packing Defects and Induces Lateral Expansion of Lipids Leading to Membrane Remodeling. Journal of Biological Chemistry, 2013, 288, 20883-20895.	3.4	183
49	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.	7.1	181
50	Direct characterization of amyloidogenic oligomers by single-molecule fluorescence. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 14424-14429.	7.1	180
51	An anticancer drug suppresses the primary nucleation reaction that initiates the production of the toxic AÎ²42 aggregates linked with Alzheimerâ€™s disease. Science Advances, 2016, 2, e1501244.	10.3	180
52	Systematic development of small molecules to inhibit specific microscopic steps of AÎ²42 aggregation in Alzheimerâ€™s disease. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, E200-E208.	7.1	180
53	Systematic In Vivo Analysis of the Intrinsic Determinants of Amyloid Î² Pathogenicity. PLoS Biology, 2007, 5, e290.	5.6	171
54	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.	3.0	166

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55	Toxicity of Protein Oligomers Is Rationalized by a Function Combining Size and Surface Hydrophobicity. ACS Chemical Biology, 2014, 9, 2309-2317.	3.4	166
56	Chemical Kinetics for Bridging Molecular Mechanisms and Macroscopic Measurements of Amyloid Fibril Formation. Annual Review of Physical Chemistry, 2018, 69, 273-298.	10.8	161
57	Direct Observation of Heterogeneous Amyloid Fibril Growth Kinetics via Two-Color Super-Resolution Microscopy. Nano Letters, 2014, 14, 339-345.	9.1	159
58	Interaction of the Molecular Chaperone DNAJB6 with Growing Amyloid-beta 42 (AÎ²42) Aggregates Leads to Sub-stoichiometric Inhibition of Amyloid Formation. Journal of Biological Chemistry, 2014, 289, 31066-31076.	3.4	158
59	Multi-dimensional super-resolution imaging enables surface hydrophobicity mapping. Nature Communications, 2016, 7, 13544.	12.8	152
60	In Situ Measurements of the Formation and Morphology of Intracellular Î²-Amyloid Fibrils by Super-Resolution Fluorescence Imaging. Journal of the American Chemical Society, 2011, 133, 12902-12905.	13.7	151
61	Supersaturation is a major driving force for protein aggregation in neurodegenerative diseases. Trends in Pharmacological Sciences, 2015, 36, 72-77.	8.7	147
62	Binding of the Molecular Chaperone Î±B-Crystallin to AÎ² Amyloid Fibrils Inhibits Fibril Elongation. Biophysical Journal, 2011, 101, 1681-1689.	0.5	143
63	Different soluble aggregates of AÎ²42 can give rise to cellular toxicity through different mechanisms. Nature Communications, 2019, 10, 1541.	12.8	140
64	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.	7.1	137
65	The Interaction of Î±B-Crystallin with Mature Î±-Synuclein Amyloid Fibrils Inhibits Their Elongation. Biophysical Journal, 2010, 98, 843-851.	0.5	136
66	Calcium is a key factor in Î±-synuclein induced neurotoxicity. Journal of Cell Science, 2016, 129, 1792-801.	2.0	136
67	A structural ensemble of a ribosomeâ€“nascent chain complex during cotranslational protein folding. Nature Structural and Molecular Biology, 2016, 23, 278-285.	8.2	135
68	Observation of spatial propagation of amyloid assembly from single nuclei. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 14746-14751.	7.1	134
69	Distinct thermodynamic signatures of oligomer generation in the aggregation of the amyloid-Î² peptide. Nature Chemistry, 2018, 10, 523-531.	13.6	129
70	Secondary nucleation and elongation occur at different sites on Alzheimerâ€™s amyloid-Î² aggregates. Science Advances, 2019, 5, eaau3112.	10.3	127
71	Targeting the Intrinsically Disordered Structural Ensemble of Î±-Synuclein by Small Molecules as a Potential Therapeutic Strategy for Parkinsonâ€™s Disease. PLoS ONE, 2014, 9, e87133.	2.5	126
72	Kinetic fingerprints differentiate the mechanisms of action of anti-AÎ² antibodies. Nature Structural and Molecular Biology, 2020, 27, 1125-1133.	8.2	123

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73	The release of toxic oligomers from Î±-synuclein fibrils induces dysfunction in neuronal cells. Nature Communications, 2021, 12, 1814.	12.8	123
74	Protein Microgels from Amyloid Fibril Networks. ACS Nano, 2015, 9, 43-51.	14.6	121
75	Selective targeting of primary and secondary nucleation pathways in AÎ²42 aggregation using a rational antibody scanning method. Science Advances, 2017, 3, e1700488.	10.3	116
76	Proteome-wide observation of the phenomenon of life on the edge of solubility. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 1015-1020.	7.1	115
77	Detailed Analysis of the Energy Barriers for Amyloid Fibril Growth. Angewandte Chemie - International Edition, 2012, 51, 5247-5251.	13.8	112
78	Trodusquemine enhances AÎ²42 aggregation but suppresses its toxicity by displacing oligomers from cell membranes. Nature Communications, 2019, 10, 225.	12.8	111
79	The Amyloid Phenomenon and Its Significance in Biology and Medicine. Cold Spring Harbor Perspectives in Biology, 2020, 12, a033878.	5.5	111
80	Binding affinity of amyloid oligomers to cellular membranes is a generic indicator of cellular dysfunction in protein misfolding diseases. Scientific Reports, 2016, 6, 32721.	3.3	107
81	Microfluidic Diffusion Analysis of the Sizes and Interactions of Proteins under Native Solution Conditions. ACS Nano, 2016, 10, 333-341.	14.6	105
82	Kinetic diversity of amyloid oligomers. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 12087-12094.	7.1	103
83	Single-Molecule Imaging of Individual Amyloid Protein Aggregates in Human Biofluids. ACS Chemical Neuroscience, 2016, 7, 399-406.	3.5	99
84	A FRET Sensor for Nonâ€Invasive Imaging of Amyloid Formation in Vivo. ChemPhysChem, 2011, 12, 673-680.	2.1	98
85	The small heat shock protein Hsp27 binds Î±-synuclein fibrils, preventing elongation and cytotoxicity. Journal of Biological Chemistry, 2018, 293, 4486-4497.	3.4	97
86	Silk micrococoons for protein stabilisation and molecular encapsulation. Nature Communications, 2017, 8, 15902.	12.8	96
87	Hsp70 Inhibits the Nucleation and Elongation of Tau and Sequesters Tau Aggregates with High Affinity. ACS Chemical Biology, 2018, 13, 636-646.	3.4	96
88	Defining Î±-synuclein species responsible for Parkinsonâ€™s disease phenotypes in mice. Journal of Biological Chemistry, 2019, 294, 10392-10406.	3.4	96
89	Small-molecule sequestration of amyloid-Î² as a drug discovery strategy for Alzheimerâ€™s disease. Science Advances, 2020, 6, .	10.3	95
90	Conserved C-Terminal Charge Exerts a Profound Influence on the Aggregation Rate of Î±-Synuclein. Journal of Molecular Biology, 2011, 411, 329-333.	4.2	92

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91	Nucleated polymerization with secondary pathways. III. Equilibrium behavior and oligomer populations. Journal of Chemical Physics, 2011, 135, 065107.	3.0	92
92	Molecular determinants of the aggregation behavior of Î±â€•and Î²â€‰synuclein. Protein Science, 2008, 17, 887-898.	7.6	91
93	Towards a structural biology of the hydrophobic effect in protein folding. Scientific Reports, 2016, 6, 28285.	3.3	91
94	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.	7.1	91
95	Physical determinants of the self-replication of protein fibrils. Nature Physics, 2016, 12, 874-880.	16.7	90
96	Heteronuclear NMR investigations of dynamic regions of intact Escherichia coli ribosomes. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 10949-10954.	7.1	87
97	Multistep Inhibition of Î±-Synuclein Aggregation and Toxicity in Vitro and in Vivo by Trodusquemine. ACS Chemical Biology, 2018, 13, 2308-2319.	3.4	86
98	Î±2-Macroglobulin and Haptoglobin Suppress Amyloid Formation by Interacting with Prefibrillar Protein Species. Journal of Biological Chemistry, 2009, 284, 4246-4254.	3.4	85
99	The Extracellular Chaperone Clusterin Potently Inhibits Human Lysozyme Amyloid Formation by Interacting with Prefibrillar Species. Journal of Molecular Biology, 2007, 369, 157-167.	4.2	84
100	A protein homeostasis signature in healthy brains recapitulates tissue vulnerability to Alzheimerâ€™s disease. Science Advances, 2016, 2, e1600947.	10.3	84
101	Rare Individual Amyloid-Î² Oligomers Act on Astrocytes to Initiate Neuronal Damage. Biochemistry, 2014, 53, 2442-2453.	2.5	83
102	Structural Ensembles of Membrane-bound Î±-Synuclein Reveal the Molecular Determinants of Synaptic Vesicle Affinity. Scientific Reports, 2016, 6, 27125.	3.3	83
103	Mapping Surface Hydrophobicity of Î±-Synuclein Oligomers at the Nanoscale. Nano Letters, 2018, 18, 7494-7501.	9.1	83
104	Fast Flow Microfluidics and Single-Molecule Fluorescence for the Rapid Characterization of Î±-Synuclein Oligomers. Analytical Chemistry, 2015, 87, 8818-8826.	6.5	81
105	Protein homeostasis of a metastable subproteome associated with Alzheimerâ€™s disease. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, E5703-E5711.	7.1	77
106	Membrane lipid composition and its physicochemical properties define cell vulnerability to aberrant protein oligomers. Journal of Cell Science, 2012, 125, 2416-27.	2.0	75
107	Amyloid-Î² Oligomers are Sequestered by both Intracellular and Extracellular Chaperones. Biochemistry, 2012, 51, 9270-9276.	2.5	75
108	A transcriptional signature of Alzheimerâ€™s disease is associated with a metastable subproteome at risk for aggregation. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, 4753-4758.	7.1	74

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109	Ultrasensitive Measurement of Ca²⁺ Influx into Lipid Vesicles Induced by Protein Aggregates. Angewandte Chemie - International Edition, 2017, 56, 7750-7754.	13.8	72
110	Spatial Persistence of Angular Correlations in Amyloid Fibrils. Physical Review Letters, 2006, 96, 238301.	7.8	71
111	Sequestration of the AÎ² Peptide Prevents Toxicity and Promotes Degradation In Vivo. PLoS Biology, 2010, 8, e1000334.	5.6	70
112	Kinetic modelling indicates that fast-translating codons can coordinate cotranslational protein folding by avoiding misfolded intermediates. Nature Communications, 2014, 5, 2988.	12.8	69
113	Detergent-like Interaction of Congo Red with the Amyloid Î² Peptide. Biochemistry, 2010, 49, 1358-1360.	2.5	66
114	A Simple Lattice Model That Captures Protein Folding, Aggregation and Amyloid Formation. PLoS ONE, 2014, 9, e85185.	2.5	66
115	Experimental free energy surfaces reveal the mechanisms of maintenance of protein solubility. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 21057-21062.	7.1	65
116	Î²-Synuclein suppresses both the initiation and amplification steps of Î±-synuclein aggregation via competitive binding to surfaces. Scientific Reports, 2016, 6, 36010.	3.3	65
117	Scaling behaviour and rate-determining steps in filamentous self-assembly. Chemical Science, 2017, 8, 7087-7097.	7.4	65
118	Observation of an Î±-synuclein liquid droplet state and its maturation into Lewy body-like assemblies. Journal of Molecular Cell Biology, 2021, 13, 282-294.	3.3	65
119	C-terminal truncation of Î±-synuclein promotes amyloid fibril amplification at physiological pH. Chemical Science, 2018, 9, 5506-5516.	7.4	64
120	Frequency Factors in a Landscape Model of Filamentous Protein Aggregation. Physical Review Letters, 2010, 104, 228101.	7.8	63
121	Nanoscopic insights into seeding mechanisms and toxicity of Î±-synuclein species in neurons. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, 3815-3819.	7.1	63
122	The contribution of biophysical and structural studies of protein self-assembly to the design of therapeutic strategies for amyloid diseases. Neurobiology of Disease, 2018, 109, 178-190.	4.4	62
123	Nanobodies raised against monomeric É'-synuclein inhibit fibril formation and destabilize toxic oligomeric species. BMC Biology, 2017, 15, 57.	3.8	61
124	Proteasome-targeted nanobodies alleviate pathology and functional decline in an Î±-synuclein-based Parkinsonâ€™s disease model. Npj Parkinson's Disease, 2018, 4, 25.	5.3	61
125	Rational design of a conformation-specific antibody for the quantification of AÎ² oligomers. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 13509-13518.	7.1	61
126	Modulation of electrostatic interactions to reveal a reaction network unifying the aggregation behaviour of the AÎ²42 peptide and its variants. Chemical Science, 2017, 8, 4352-4362.	7.4	60

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127	Phage display and kinetic selection of antibodies that specifically inhibit amyloid self-replication. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, 6444-6449.	7.1	60
128	Transthyretin Inhibits Primary and Secondary Nucleations of Amyloid-Î² Peptide Aggregation and Reduces the Toxicity of Its Oligomers. Biomacromolecules, 2020, 21, 1112-1125.	5.4	59
129	Single-Molecule Characterization of the Interactions between Extracellular Chaperones and Toxic Î±-Synuclein Oligomers. Cell Reports, 2018, 23, 3492-3500.	6.4	59
130	The Influence of Pathogenic Mutations in Î±-Synuclein on Biophysical and Structural Characteristics of Amyloid Fibrils. ACS Nano, 2020, 14, 5213-5222.	14.6	58
131	Latent analysis of unmodified biomolecules and their complexes in solution with attomole detection sensitivity. Nature Chemistry, 2015, 7, 802-809.	13.6	56
132	Structural characterization of the interaction of Î±-synuclein nascent chains with the ribosomal surface and trigger factor. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, 5012-5017.	7.1	54
133	SAR by kinetics for drug discovery in protein misfolding diseases. Proceedings of the National Academy of Sciences of the United States of America, 2018, 115, 10245-10250.	7.1	54
134	Massively parallel C. elegans tracking provides multi-dimensional fingerprints for phenotypic discovery. Journal of Neuroscience Methods, 2018, 306, 57-67.	2.5	52
135	Nanoscopic Characterisation of Individual Endogenous Protein Aggregates in Human Neuronal Cells. ChemBioChem, 2018, 19, 2033-2038.	2.6	52
136	Thermodynamic and kinetic design principles for amyloid-aggregation inhibitors. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 24251-24257.	7.1	49
137	Cytosolic aggregation of mitochondrial proteins disrupts cellular homeostasis by stimulating the aggregation of other proteins. ELife, 2021, 10, .	6.0	49
138	Twisting Transition between Crystalline and Fibrillar Phases of Aggregated Peptides. Physical Review Letters, 2012, 109, 158101.	7.8	48
139	Quantitative thermophoretic study of disease-related protein aggregates. Scientific Reports, 2016, 6, 22829.	3.3	48
140	Inhibition of Î±-Synuclein Fibril Elongation by Hsp70 Is Governed by a Kinetic Binding Competition between Î±-Synuclein Species. Biochemistry, 2017, 56, 1177-1180.	2.5	47
141	The N-terminal Acetylation of Î±-Synuclein Changes the Affinity for Lipid Membranes but not the Structural Properties of the Bound State. Scientific Reports, 2020, 10, 204.	3.3	47
142	Hydrophobicity and Conformational Change as Mechanistic Determinants for Nonspecific Modulators of Amyloid Î² Self-Assembly. Biochemistry, 2012, 51, 126-137.	2.5	46
143	Intrinsic Determinants of Neurotoxic Aggregate Formation by the Amyloid Î² Peptide. Biophysical Journal, 2010, 98, 1677-1684.	0.5	45
144	Monomeric and fibrillar Î±-synuclein exert opposite effects on the catalytic cycle that promotes the proliferation of AÎ²42 aggregates. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, 8005-8010.	7.1	45

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145	Direct Observation of Oligomerization by Single Molecule Fluorescence Reveals a Multistep Aggregation Mechanism for the Yeast Prion Protein Ure2. Journal of the American Chemical Society, 2018, 140, 2493-2503.	13.7	44
146	Trodusquemine displaces protein misfolded oligomers from cell membranes and abrogates their cytotoxicity through a generic mechanism. Communications Biology, 2020, 3, 435.	4.4	44
147	Enzymatically Active Microgels from Self-Assembling Protein Nanofibrils for Microflow Chemistry. ACS Nano, 2015, 9, 5772-5781.	14.6	43
148	Lipid Dynamics and Phase Transition within Î±-Synuclein Amyloid Fibrils. Journal of Physical Chemistry Letters, 2019, 10, 7872-7877.	4.6	43
149	ThX â€“ a next-generation probe for the early detection of amyloid aggregates. Chemical Science, 2020, 11, 4578-4583.	7.4	43
150	Stabilization and Characterization of Cytotoxic AÎ²₄₀ Oligomers Isolated from an Aggregation Reaction in the Presence of Zinc Ions. ACS Chemical Neuroscience, 2018, 9, 2959-2971.	3.5	42
151	Expression in Drosophila of Tandem Amyloid Î² Peptides Provides Insights into Links between Aggregation and Neurotoxicity. Journal of Biological Chemistry, 2012, 287, 20748-20754.	3.4	40
152	Optical Structural Analysis of Individual Î±â€Synuclein Oligomers. Angewandte Chemie - International Edition, 2018, 57, 4886-4890.	13.8	40
153	Microfluidic deposition for resolving single-molecule protein architecture and heterogeneity. Nature Communications, 2018, 9, 3890.	12.8	40
154	Nucleation-conversion-polymerization reactions of biological macromolecules with prenucleation clusters. Physical Review E, 2014, 89, 032712.	2.1	39
155	Quantifying Co-Oligomer Formation by Î±-Synuclein. ACS Nano, 2018, 12, 10855-10866.	14.6	38
156	Direct measurement of lipid membrane disruption connects kinetics and toxicity of AÎ²42 aggregation. Nature Structural and Molecular Biology, 2020, 27, 886-891.	8.2	38
157	The Hsc70 disaggregation machinery removes monomer units directly from Î±-synuclein fibril ends. Nature Communications, 2021, 12, 5999.	12.8	37
158	A Fragment-Based Method of Creating Small-Molecule Libraries to Target the Aggregation of Intrinsically Disordered Proteins. ACS Combinatorial Science, 2016, 18, 144-153.	3.8	35
159	AÎ² Oligomers Dysregulate Calcium Homeostasis by Mechanosensitive Activation of AMPA and NMDA Receptors. ACS Chemical Neuroscience, 2021, 12, 766-781.	3.5	35
160	Determination of the structures of distinct transition state ensembles for a Î²-sheet peptide with parallel folding pathways. Journal of Chemical Physics, 2002, 117, 9510-9517.	3.0	34
161	The Toxicity of Misfolded Protein Oligomers Is Independent of Their Secondary Structure. ACS Chemical Biology, 2019, 14, 1593-1600.	3.4	34
162	Squalamine and Its Derivatives Modulate the Aggregation of Amyloid-Î² and Î±-Synuclein and Suppress the Toxicity of Their Oligomers. Frontiers in Neuroscience, 2021, 15, 680026.	2.8	34

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163	Probing the Origin of the Toxicity of Oligomeric Aggregates of Î±-Synuclein with Antibodies. ACS Chemical Biology, 2019, 14, 1352-1362.	3.4	33
164	Structure of a low-population intermediate state in the release of an enzyme product. ELife, 2015, 4, .	6.0	33
165	Hamiltonian Dynamics of Protein Filament Formation. Physical Review Letters, 2016, 116, 038101.	7.8	32
166	Kinetic barriers to Î±-synuclein protofilament formation and conversion into mature fibrils. Chemical Communications, 2018, 54, 7854-7857.	4.1	31
167	Molecular determinants of the interaction of EGCG with ordered and disordered proteins. Biopolymers, 2018, 109, e23117.	2.4	30
168	Fast Fluorescence Lifetime Imaging Reveals the Aggregation Processes of Î±-Synuclein and Polyglutamine in Aging Caenorhabditis elegans. ACS Chemical Biology, 2019, 14, 1628-1636.	3.4	30
169	The extent of protein hydration dictates the preference for heterogeneous or homogeneous nucleation generating either parallel or antiparallel Î²-sheet Î±-synuclein aggregates. Chemical Science, 2020, 11, 11902-11914.	7.4	30
170	A Role of Cholesterol in Modulating the Binding of Î±-Synuclein to Synaptic-Like Vesicles. Frontiers in Neuroscience, 2020, 14, 18.	2.8	30
171	Synthesis of Nonequilibrium Supramolecular Peptide Polymers on a Microfluidic Platform. Journal of the American Chemical Society, 2016, 138, 9589-9596.	13.7	27
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