Christopher M Dobson

List of Publications by Year
in descending order

226 papers	46,083 citations	5126 86 h-index	2289 206 g-index
240 all docs	240 docs citations	240 times ranked	30561 citing authors

#	Article	IF	Citations
1	The Pathological G51D Mutation in Alpha-Synuclein Oligomers Confers Distinct Structural Attributes and Cellular Toxicity. Molecules, 2022, 27, 1293.	1.7	6
2	Systematic Activity Maturation of a Single-Domain Antibody with Non-canonical Amino Acids through Chemical Mutagenesis. Cell Chemical Biology, 2021, 28, 70-77.e5.	2.5	15
3	AÎ² Oligomers Dysregulate Calcium Homeostasis by Mechanosensitive Activation of AMPA and NMDA Receptors. ACS Chemical Neuroscience, 2021, 12, 766-781.	1.7	35
4	Scaling analysis reveals the mechanism and rates of prion replication in vivo. Nature Structural and Molecular Biology, 2021, 28, 365-372.	3.6	22
5	The release of toxic oligomers from Î±-synuclein fibrils induces dysfunction in neuronal cells. Nature Communications, 2021, 12, 1814.	5.8	123
6	Comparative Studies in the A30P and A53T Î±-Synuclein C. elegans Strains to Investigate the Molecular Origins of Parkinson's Disease. Frontiers in Cell and Developmental Biology, 2021, 9, 552549.	1.8	12
7	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.	1.4	34
8	Distinct responses of human peripheral blood cells to different misfolded protein oligomers. Immunology, 2021, 164, 358-371.	2.0	7
9	Two human metabolites rescue a C. elegans model of Alzheimerâ€™s disease via a cytosolic unfolded protein response. Communications Biology, 2021, 4, 843.	2.0	6
10	Exogenous misfolded protein oligomers can cross the intestinal barrier and cause a disease phenotype in C. elegans. Scientific Reports, 2021, 11, 14391.	1.6	6
11	Cytosolic aggregation of mitochondrial proteins disrupts cellular homeostasis by stimulating the aggregation of other proteins. ELife, 2021, 10, .	2.8	49
12	The binding of the small heat-shock protein Î±B-crystallin to fibrils of Î±-synuclein is driven by entropic forces. Proceedings of the National Academy of Sciences of the United States of America, 2021, 118, .	3.3	15
13	A dopamine metabolite stabilizes neurotoxic amyloid-Î² oligomers. Communications Biology, 2021, 4, 19.	2.0	25
14	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.	1.5	65
15	The Hsc70 disaggregation machinery removes monomer units directly from Î±-synuclein fibril ends. Nature Communications, 2021, 12, 5999.	5.8	37
16	The Amyloid Phenomenon and Its Significance in Biology and Medicine. Cold Spring Harbor Perspectives in Biology, 2020, 12, a033878.	2.3	111
17	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.	3.3	115
18	Kinetic fingerprints differentiate the mechanisms of action of anti-AÎ² antibodies. Nature Structural and Molecular Biology, 2020, 27, 1125-1133.	3.6	123

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19	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.	3.7	30
20	Direct measurement of lipid membrane disruption connects kinetics and toxicity of AÎ²42 aggregation. Nature Structural and Molecular Biology, 2020, 27, 886-891.	3.6	38
21	Amelioration of aggregate cytotoxicity by catalytic conversion of protein oligomers into amyloid fibrils. Nanoscale, 2020, 12, 18663-18672.	2.8	13
22	A rationally designed bicyclic peptide remodels AÎ²42 aggregation in vitro and reduces its toxicity in a worm model of Alzheimerâ€™s disease. Scientific Reports, 2020, 10, 15280.	1.6	15
23	Structural Characterization of Covalently Stabilized Human Cystatin C Oligomers. International Journal of Molecular Sciences, 2020, 21, 5860.	1.8	3
24	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.	3.3	49
25	Trodusquemine displaces protein misfolded oligomers from cell membranes and abrogates their cytotoxicity through a generic mechanism. Communications Biology, 2020, 3, 435.	2.0	44
26	Biophysical studies of protein misfolding and aggregation in in vivo models of Alzheimer's and Parkinson's diseases â€“ ERRATUM. Quarterly Reviews of Biophysics, 2020, 53, e13.	2.4	7
27	Small-molecule sequestration of amyloid-Î² as a drug discovery strategy for Alzheimerâ€™s disease. Science Advances, 2020, 6, .	4.7	95
28	Kinetic diversity of amyloid oligomers. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 12087-12094.	3.3	103
29	Assessing motor-related phenotypes of Caenorhabditis elegans with the wide field-of-view nematode tracking platform. Nature Protocols, 2020, 15, 2071-2106.	5.5	23
30	Biophysical studies of protein misfolding and aggregation inin vivomodels of Alzheimer's and Parkinson's diseases. Quarterly Reviews of Biophysics, 2020, 53, e22.	2.4	13
31	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.	3.3	61
32	The Influence of Pathogenic Mutations in Î±-Synuclein on Biophysical and Structural Characteristics of Amyloid Fibrils. ACS Nano, 2020, 14, 5213-5222.	7.3	58
33	A Cell- and Tissue-Specific Weakness of the Protein Homeostasis System Underlies Brain Vulnerability to Protein Aggregation. IScience, 2020, 23, 100934.	1.9	9
34	Half a century of amyloids: past, present and future. Chemical Society Reviews, 2020, 49, 5473-5509.	18.7	345
35	Rationally Designed Antibodies as Research Tools to Study the Structureâ€“Toxicity Relationship of Amyloid-Î² Oligomers. International Journal of Molecular Sciences, 2020, 21, 4542.	1.8	12
36	A Role of Cholesterol in Modulating the Binding of Î±-Synuclein to Synaptic-Like Vesicles. Frontiers in Neuroscience, 2020, 14, 18.	1.4	30

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37	ThX â€“ a next-generation probe for the early detection of amyloid aggregates. Chemical Science, 2020, 11, 4578-4583.	3.7	43
38	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.	1.6	47
39	Transthyretin Inhibits Primary and Secondary Nucleations of Amyloid-Î² Peptide Aggregation and Reduces the Toxicity of Its Oligomers. Biomacromolecules, 2020, 21, 1112-1125.	2.6	59
40	Dynamics of oligomer populations formed during the aggregation of Alzheimerâ€™s AÎ²42 peptide. Nature Chemistry, 2020, 12, 445-451.	6.6	223
41	Screening of small molecules using the inhibition of oligomer formation in Î±-synuclein aggregation as a selection parameter. Communications Chemistry, 2020, 3, .	2.0	27
42	Differential Interactome and Innate Immune Response Activation of Two Structurally Distinct Misfolded Protein Oligomers. ACS Chemical Neuroscience, 2019, 10, 3464-3478.	1.7	7
43	Enhancement of the Anti-Aggregation Activity of a Molecular Chaperone Using a Rationally Designed Post-Translational Modification. ACS Central Science, 2019, 5, 1417-1424.	5.3	18
44	Bacterial production and direct functional screening of expanded molecular libraries for discovering inhibitors of protein aggregation. Science Advances, 2019, 5, eaax5108.	4.7	12
45	Probing the dynamic stalk region of the ribosome using solution NMR. Scientific Reports, 2019, 9, 13528.	1.6	10
46	Chemical and mechanistic analysis of photodynamic inhibition of Alzheimer's Î²-amyloid aggregation. Chemical Communications, 2019, 55, 1152-1155.	2.2	19
47	Fast Fluorescence Lifetime Imaging Reveals the Aggregation Processes of Î±-Synuclein and Polyglutamine in Aging Caenorhabditis elegans. ACS Chemical Biology, 2019, 14, 1628-1636.	1.6	30
48	Defining Î±-synuclein species responsible for Parkinsonâ€™s disease phenotypes in mice. Journal of Biological Chemistry, 2019, 294, 10392-10406.	1.6	96
49	Expression of the amyloid-Î² peptide in a single pair of C. elegans sensory neurons modulates the associated behavioural response. PLoS ONE, 2019, 14, e0217746.	1.1	10
50	The Toxicity of Misfolded Protein Oligomers Is Independent of Their Secondary Structure. ACS Chemical Biology, 2019, 14, 1593-1600.	1.6	34
51	Secondary nucleation and elongation occur at different sites on Alzheimerâ€™s amyloid-Î² aggregates. Science Advances, 2019, 5, eaau3112.	4.7	127
52	Identifying A- and P-site locations on ribosome-protected mRNA fragments using Integer Programming. Scientific Reports, 2019, 9, 6256.	1.6	18
53	Probing the Origin of the Toxicity of Oligomeric Aggregates of Î±-Synuclein with Antibodies. ACS Chemical Biology, 2019, 14, 1352-1362.	1.6	33
54	The metastability of the proteome of spinal motor neurons underlies their selective vulnerability in ALS. Neuroscience Letters, 2019, 704, 89-94.	1.0	22

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55	Different soluble aggregates of AÎ²42 can give rise to cellular toxicity through different mechanisms. Nature Communications, 2019, 10, 1541.	5.8	140
56	Increased Secondary Nucleation Underlies Accelerated Aggregation of the Four-Residue N-Terminally Truncated AÎ²42 Species AÎ²5â€“42. ACS Chemical Neuroscience, 2019, 10, 2374-2384.	1.7	16
57	Supersaturated proteins are enriched at synapses and underlie cell and tissue vulnerability in Alzheimer's disease. Heliyon, 2019, 5, e02589.	1.4	23
58	Lipid Dynamics and Phase Transition within Î±-Synuclein Amyloid Fibrils. Journal of Physical Chemistry Letters, 2019, 10, 7872-7877.	2.1	43
59	A metastable subproteome underlies inclusion formation in muscle proteinopathies. Acta Neuropathologica Communications, 2019, 7, 197.	2.4	16
60	Trodusquemine enhances AÎ²42 aggregation but suppresses its toxicity by displacing oligomers from cell membranes. Nature Communications, 2019, 10, 225.	5.8	111
61	Dynamics and Control of Peptide Self-Assembly and Aggregation. Advances in Experimental Medicine and Biology, 2019, 1174, 1-33.	0.8	6
62	Bifunctional fluorescent probes for detection of amyloid aggregates and reactive oxygen species. Royal Society Open Science, 2018, 5, 171399.	1.1	11
63	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
64	Molecular determinants of the interaction of EGCG with ordered and disordered proteins. Biopolymers, 2018, 109, e23117.	1.2	30
65	Exploring the role of postâ€translational modifications in regulating Î±â€synuclein interactions by studying the effects of phosphorylation on nanobody binding. Protein Science, 2018, 27, 1262-1274.	3.1	25
66	Massively parallel C. elegans tracking provides multi-dimensional fingerprints for phenotypic discovery. Journal of Neuroscience Methods, 2018, 306, 57-67.	1.3	52
67	The small heat shock protein Hsp27 binds Î±-synuclein fibrils, preventing elongation and cytotoxicity. Journal of Biological Chemistry, 2018, 293, 4486-4497.	1.6	97
68	Optical Structural Analysis of Individual Î±â€Synuclein Oligomers. Angewandte Chemie - International Edition, 2018, 57, 4886-4890.	7.2	40
69	Optical Structural Analysis of Individual Î±â€Synuclein Oligomers. Angewandte Chemie, 2018, 130, 4980-4984.	1.6	0
70	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.	6.6	44
71	Microfluidic Diffusion Platform for Characterizing the Sizes of Lipid Vesicles and the Thermodynamics of Proteinâ€“Lipid Interactions. Analytical Chemistry, 2018, 90, 3284-3290.	3.2	20
72	Hsp70 Inhibits the Nucleation and Elongation of Tau and Sequesters Tau Aggregates with High Affinity. ACS Chemical Biology, 2018, 13, 636-646.	1.6	96

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73	Distinct thermodynamic signatures of oligomer generation in the aggregation of the amyloid-Î² peptide. Nature Chemistry, 2018, 10, 523-531.	6.6	129
74	Microfluidic approaches for probing amyloid assembly and behaviour. Lab on A Chip, 2018, 18, 999-1016.	3.1	27
75	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.	2.1	62
76	Automated Behavioral Analysis of Large C. elegans Populations Using a Wide Field-of-view Tracking Platform. Journal of Visualized Experiments, 2018, , .	0.2	7
77	Microfluidic deposition for resolving single-molecule protein architecture and heterogeneity. Nature Communications, 2018, 9, 3890.	5.8	40
78	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.	3.3	54
79	Mapping Surface Hydrophobicity of Î±-Synuclein Oligomers at the Nanoscale. Nano Letters, 2018, 18, 7494-7501.	4.5	83
80	Quantifying Co-Oligomer Formation by Î±-Synuclein. ACS Nano, 2018, 12, 10855-10866.	7.3	38
81	Proteasome-targeted nanobodies alleviate pathology and functional decline in an Î±-synuclein-based Parkinsonâ€™s disease model. Npj Parkinson's Disease, 2018, 4, 25.	2.5	61
82	C-terminal truncation of Î±-synuclein promotes amyloid fibril amplification at physiological pH. Chemical Science, 2018, 9, 5506-5516.	3.7	64
83	Cooperative Assembly of Hsp70 Subdomain Clusters. Biochemistry, 2018, 57, 3641-3649.	1.2	13
84	Kinetic barriers to Î±-synuclein protofilament formation and conversion into mature fibrils. Chemical Communications, 2018, 54, 7854-7857.	2.2	31
85	Multistep Inhibition of Î±-Synuclein Aggregation and Toxicity in Vitro and in Vivo by Trodusquemine. ACS Chemical Biology, 2018, 13, 2308-2319.	1.6	86
86	Nanoscopic Characterisation of Individual Endogenous Protein Aggregates in Human Neuronal Cells. ChemBioChem, 2018, 19, 2033-2038.	1.3	52
87	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.	1.7	42
88	Structural differences between toxic and nontoxic HypF-N oligomers. Chemical Communications, 2018, 54, 8637-8640.	2.2	25
89	Cholesterol catalyses AÎ²42 aggregation through a heterogeneous nucleation pathway in the presence of lipid membranes. Nature Chemistry, 2018, 10, 673-683.	6.6	186
90	Single-Molecule Characterization of the Interactions between Extracellular Chaperones and Toxic Î±-Synuclein Oligomers. Cell Reports, 2018, 23, 3492-3500.	2.9	59

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91	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.	3.3	231
92	Inhibition of Î±-Synuclein Fibril Elongation by Hsp70 Is Governed by a Kinetic Binding Competition between Î±-Synuclein Species. Biochemistry, 2017, 56, 1177-1180.	1.2	47
93	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
94	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
95	Amyloid-like Fibrils from an Î±-Helical Transmembrane Protein. Biochemistry, 2017, 56, 3225-3233.	1.2	19
96	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.	3.7	60
97	Direct Conversion of an Enzyme from Native-like to Amyloid-like Aggregates within Inclusion Bodies. Biophysical Journal, 2017, 112, 2540-2551.	0.2	9
98	Selective targeting of primary and secondary nucleation pathways in AÎ²42 aggregation using a rational antibody scanning method. Science Advances, 2017, 3, e1700488.	4.7	116
99	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.	3.3	60
100	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.	3.3	180
101	Gradient-free determination of isoelectric points of proteins on chip. Physical Chemistry Chemical Physics, 2017, 19, 23060-23067.	1.3	25
102	Scaling behaviour and rate-determining steps in filamentous self-assembly. Chemical Science, 2017, 8, 7087-7097.	3.7	65
103	Silk micrococoons for protein stabilisation and molecular encapsulation. Nature Communications, 2017, 8, 15902.	5.8	96
104	Structural basis of membrane disruption and cellular toxicity by Î±-synuclein oligomers. Science, 2017, 358, 1440-1443.	6.0	492
105	Inhibiting the Ca 2+ Influx Induced by Human CSF. Cell Reports, 2017, 21, 3310-3316.	2.9	20
106	Delivery of Native Proteins into C. elegans Using a Transduction Protocol Based on Lipid Vesicles. Scientific Reports, 2017, 7, 15045.	1.6	16
107	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.	3.3	77
108	Ultrasensitive Measurement of Ca²⁺ Influx into Lipid Vesicles Induced by Protein Aggregates. Angewandte Chemie, 2017, 129, 7858-7862.	1.6	9

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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.	7.2	72
110	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.	3.3	45
111	Nanobodies raised against monomeric É'-synuclein inhibit fibril formation and destabilize toxic oligomeric species. BMC Biology, 2017, 15, 57.	1.7	61
112	Synthesis of Nonequilibrium Supramolecular Peptide Polymers on a Microfluidic Platform. Journal of the American Chemical Society, 2016, 138, 9589-9596.	6.6	27
113	Structural basis of synaptic vesicle assembly promoted by Î±-synuclein. Nature Communications, 2016, 7, 12563.	5.8	203
114	Multi-dimensional super-resolution imaging enables surface hydrophobicity mapping. Nature Communications, 2016, 7, 13544.	5.8	152
115	Kinetic analysis reveals the diversity of microscopic mechanisms through which molecular chaperones suppress amyloid formation. Nature Communications, 2016, 7, 10948.	5.8	219
116	Towards a structural biology of the hydrophobic effect in protein folding. Scientific Reports, 2016, 6, 28285.	1.6	91
117	Quantitative thermophoretic study of disease-related protein aggregates. Scientific Reports, 2016, 6, 22829.	1.6	48
118	Structural Ensembles of Membrane-bound Î±-Synuclein Reveal the Molecular Determinants of Synaptic Vesicle Affinity. Scientific Reports, 2016, 6, 27125.	1.6	83
119	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.	3.3	54
120	Structural Effects of Two Camelid Nanobodies Directed to Distinct C-Terminal Epitopes on Î±-Synuclein. Biochemistry, 2016, 55, 3116-3122.	1.2	22
121	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.	3.3	74
122	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.	3.3	252
123	Protein Aggregateâ€Ligand Binding Assays Based on Microfluidic Diffusional Separation. ChemBioChem, 2016, 17, 1920-1924.	1.3	11
124	Physical determinants of the self-replication of protein fibrils. Nature Physics, 2016, 12, 874-880.	6.5	90
125	Hamiltonian Dynamics of Protein Filament Formation. Physical Review Letters, 2016, 116, 038101.	2.9	32
126	Binding affinity of amyloid oligomers to cellular membranes is a generic indicator of cellular dysfunction in protein misfolding diseases. Scientific Reports, 2016, 6, 32721.	1.6	107

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127	A protein homeostasis signature in healthy brains recapitulates tissue vulnerability to Alzheimerâ€™s disease. Science Advances, 2016, 2, e1600947.	4.7	84
128	Î²-Synuclein suppresses both the initiation and amplification steps of Î±-synuclein aggregation via competitive binding to surfaces. Scientific Reports, 2016, 6, 36010.	1.6	65
129	Particle-Based Monte-Carlo Simulations of Steady-State Mass Transport at Intermediate PÃ©clet Numbers. International Journal of Nonlinear Sciences and Numerical Simulation, 2016, 17, 175-183.	0.4	27
130	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.	3.3	248
131	Effect of molecular chaperones on aberrant protein oligomers in vitro: super-versus sub-stoichiometric chaperone concentrations. Biological Chemistry, 2016, 397, 401-415.	1.2	19
132	Single-Molecule Imaging of Individual Amyloid Protein Aggregates in Human Biofluids. ACS Chemical Neuroscience, 2016, 7, 399-406.	1.7	99
133	Calcium is a key factor in Î±-synuclein induced neurotoxicity. Journal of Cell Science, 2016, 129, 1792-801.	1.2	136
134	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.	3.3	63
135	Automated Ex Situ Assays of Amyloid Formation on a Microfluidic Platform. Biophysical Journal, 2016, 110, 555-560.	0.2	15
136	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
137	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.	4.7	180
138	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
139	A structural ensemble of a ribosomeâ€“nascent chain complex during cotranslational protein folding. Nature Structural and Molecular Biology, 2016, 23, 278-285.	3.6	135
140	Molecular mechanisms of protein aggregation from global fitting of kinetic models. Nature Protocols, 2016, 11, 252-272.	5.5	546
141	Microfluidic Diffusion Analysis of the Sizes and Interactions of Proteins under Native Solution Conditions. ACS Nano, 2016, 10, 333-341.	7.3	105
142	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
143	Structure-Free Validation of Residual Dipolar Coupling and Paramagnetic Relaxation Enhancement Measurements of Disordered Proteins. Biochemistry, 2015, 54, 6876-6886.	1.2	19
144	Enzymatically Active Microgels from Self-Assembling Protein Nanofibrils for Microflow Chemistry. ACS Nano, 2015, 9, 5772-5781.	7.3	43

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145	A molecular chaperone breaks the catalytic cycle that generates toxic Aî² oligomers. Nature Structural and Molecular Biology, 2015, 22, 207-213.	3.6	373
146	Lipid vesicles trigger Î±-synuclein aggregation by stimulating primary nucleation. Nature Chemical Biology, 2015, 11, 229-234.	3.9	532
147	Supersaturation is a major driving force for protein aggregation in neurodegenerative diseases. Trends in Pharmacological Sciences, 2015, 36, 72-77.	4.0	147
148	Protein Microgels from Amyloid Fibril Networks. ACS Nano, 2015, 9, 43-51.	7.3	121
149	Force generation by the growth of amyloid aggregates. Proceedings of the National Academy of Sciences of the United States of America, 2015, 112, 9524-9529.	3.3	25
150	Widespread Proteome Remodeling and Aggregation in Aging C.Âelegans. Cell, 2015, 161, 919-932.	13.5	478
151	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.	3.3	384
152	Fast Flow Microfluidics and Single-Molecule Fluorescence for the Rapid Characterization of Î±-Synuclein Oligomers. Analytical Chemistry, 2015, 87, 8818-8826.	3.2	81
153	Latent analysis of unmodified biomolecules and their complexes in solution with attomole detection sensitivity. Nature Chemistry, 2015, 7, 802-809.	6.6	56
154	Structure of a low-population intermediate state in the release of an enzyme product. ELife, 2015, 4, .	2.8	33
155	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.	1.1	126
156	Kinetic modelling indicates that fast-translating codons can coordinate cotranslational protein folding by avoiding misfolded intermediates. Nature Communications, 2014, 5, 2988.	5.8	69
157	Nucleation-conversion-polymerization reactions of biological macromolecules with prenucleation clusters. Physical Review E, 2014, 89, 032712.	0.8	39
158	Rare Individual Amyloid-Î² Oligomers Act on Astrocytes to Initiate Neuronal Damage. Biochemistry, 2014, 53, 2442-2453.	1.2	83
159	Chemical kinetics for drug discovery to combat protein aggregation diseases. Trends in Pharmacological Sciences, 2014, 35, 127-135.	4.0	191
160	The amyloid state and its association with protein misfolding diseases. Nature Reviews Molecular Cell Biology, 2014, 15, 384-396.	16.1	1,894
161	Direct Observation of Heterogeneous Amyloid Fibril Growth Kinetics via Two-Color Super-Resolution Microscopy. Nano Letters, 2014, 14, 339-345.	4.5	159
162	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.	1.6	158

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163	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.	3.3	405
164	Toxicity of Protein Oligomers Is Rationalized by a Function Combining Size and Surface Hydrophobicity. ACS Chemical Biology, 2014, 9, 2309-2317.	1.6	166
165	Oligomer-targeting with a conformational antibody fragment promotes toxicity in AÎ²-expressing flies. Acta Neuropathologica Communications, 2014, 2, 43.	2.4	10
166	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.	3.3	546
167	The Role of Stable Î±-Synuclein Oligomers in the Molecular Events Underlying Amyloid Formation. Journal of the American Chemical Society, 2014, 136, 3859-3868.	6.6	218
168	Direct observation of the three regions in Î±-synuclein that determine its membrane-bound behaviour. Nature Communications, 2014, 5, 3827.	5.8	357
169	A Simple Lattice Model That Captures Protein Folding, Aggregation and Amyloid Formation. PLoS ONE, 2014, 9, e85185.	1.1	66
170	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.	3.3	479
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