Christopher M Dobson

List of Publications by Citations

231 papers	36,210 citations	76 h-index	189 g-index
240 ext. papers	41,730 ext. citations	10.3 avg, IF	7.87 L-index

#	Paper	IF	Citations
231	Protein misfolding, functional amyloid, and human disease. *Annual Review of Biochemistry*, **2006**, 75, 333-66	29.1	5002
230	Protein folding and misfolding. *Nature*, **2003**, 426, 884-90	50.4	3716
229	Inherent toxicity of aggregates implies a common mechanism for protein misfolding diseases. *Nature*, **2002**, 416, 507-11	50.4	2119
228	Protein misfolding, evolution and disease. *Trends in Biochemical Sciences*, **1999**, 24, 329-32	10.3	1690
227	The amyloid state and its association with protein misfolding diseases. *Nature Reviews Molecular Cell Biology*, **2014**, 15, 384-96	48.7	1481
226	Protein aggregation and aggregate toxicity: new insights into protein folding, misfolding diseases and biological evolution. *Journal of Molecular Medicine*, **2003**, 81, 678-99	5.5	1272
225	Protein Misfolding, Amyloid Formation, and Human Disease: A Summary of Progress Over the Last Decade. *Annual Review of Biochemistry*, **2017**, 86, 27-68	29.1	1248
224	Proliferation of amyloid-42 aggregates occurs through a secondary nucleation mechanism. *Proceedings of the National Academy of Sciences of the United States of America*, **2013**, 110, 9758-63	11.5	867
223	An analytical solution to the kinetics of breakable filament assembly. *Science*, **2009**, 326, 1533-7	33.3	804
222	The protofilament structure of insulin amyloid fibrils. *Proceedings of the National Academy of Sciences of the United States of America*, **2002**, 99, 9196-201	11.5	707
221	Direct observation of the interconversion of normal and toxic forms of synuclein. *Cell*, **2012**, 149, 1048-59	36.2	588
220	Mapping long-range interactions in alpha-synuclein using spin-label NMR and ensemble molecular dynamics simulations. *Journal of the American Chemical Society*, **2005**, 127, 476-7	16.4	559
219	A causative link between the structure of aberrant protein oligomers and their toxicity. *Nature Chemical Biology*, **2010**, 6, 140-7	11.7	443
218	Atomic structure and hierarchical assembly of a cross- amyloid fibril. *Proceedings of the National Academy of Sciences of the United States of America*, **2013**, 110, 5468-73	11.5	401
217	Solution conditions determine the relative importance of nucleation and growth processes in synuclein aggregation. *Proceedings of the National Academy of Sciences of the United States of America*, **2014**, 111, 7671-6	11.5	395
216	Lipid vesicles trigger synuclein aggregation by stimulating primary nucleation. *Nature Chemical Biology*, **2015**, 11, 229-34	11.7	355
215	Molecular mechanisms of protein aggregation from global fitting of kinetic models. *Nature Protocols*, **2016**, 11, 252-72	18.8	342

214	Widespread Proteome Remodeling and Aggregation in Aging C. elegans. *Cell*, **2015**, 161, 919-32	56.2	333
213	From macroscopic measurements to microscopic mechanisms of protein aggregation. *Journal of Molecular Biology*, **2012**, 421, 160-71	6.5	331
212	Metastability of native proteins and the phenomenon of amyloid formation. *Journal of the American Chemical Society*, **2011**, 133, 14160-3	16.4	305
211	Structural basis of membrane disruption and cellular toxicity by -synuclein oligomers. *Science*, **2017**, 358, 1440-1443	33.3	301
210	Differences in nucleation behavior underlie the contrasting aggregation kinetics of the A40 and A42 peptides. *Proceedings of the National Academy of Sciences of the United States of America*, **2014**, 111, 9384-9	11.5	294
209	ANS binding reveals common features of cytotoxic amyloid species. *ACS Chemical Biology*, **2010**, 5, 735-40	4.9	291
208	Multiple tight phospholipid-binding modes of alpha-synuclein revealed by solution NMR spectroscopy. *Journal of Molecular Biology*, **2009**, 390, 775-90	6.5	281
207	Structural characterization of toxic oligomers that are kinetically trapped during -synuclein fibril formation. *Proceedings of the National Academy of Sciences of the United States of America*, **2015**, 112, E1994-2003	11.5	278
206	A molecular chaperone breaks the catalytic cycle that generates toxic A oligomers. *Nature Structural and Molecular Biology*, **2015**, 22, 207-213	17.6	268
205	Direct observation of the three regions in -synuclein that determine its membrane-bound behaviour. *Nature Communications*, **2014**, 5, 3827	17.4	266
204	The extracellular chaperone clusterin influences amyloid formation and toxicity by interacting with prefibrillar structures. *FASEB Journal*, **2007**, 21, 2312-22	0.9	237
203	Nucleated polymerization with secondary pathways. I. Time evolution of the principal moments. *Journal of Chemical Physics*, **2011**, 135, 065105	3.9	226
202	A camelid antibody fragment inhibits the formation of amyloid fibrils by human lysozyme. *Nature*, **2003**, 424, 783-8	50.4	212
201	Prefibrillar amyloid aggregates could be generic toxins in higher organisms. *Journal of Neuroscience* , **2006**, 26, 8160-7	6.6	199
200	The extracellular chaperone clusterin sequesters oligomeric forms of the amyloid- (1-40) peptide. *Nature Structural and Molecular Biology*, **2011**, 19, 79-83	17.6	198
199	Alpha-Synuclein Oligomers Interact with Metal Ions to Induce Oxidative Stress and Neuronal Death in Parkinson's Disease. *Antioxidants and Redox Signaling*, **2016**, 24, 376-91	8.4	192
198	Widespread aggregation and neurodegenerative diseases are associated with supersaturated proteins. *Cell Reports*, **2013**, 5, 781-90	10.6	182
197	Differential phospholipid binding of alpha-synuclein variants implicated in Parkinson's disease revealed by solution NMR spectroscopy. *Biochemistry*, **2010**, 49, 862-71	3.2	179

196	Structural reorganisation and potential toxicity of oligomeric species formed during the assembly of amyloid fibrils. *PLoS Computational Biology*, **2007**, 3, 1727-38	5	178
195	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	11.5	177
194	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, 10016-21	11.5	167
193	Direct characterization of amyloidogenic oligomers by single-molecule fluorescence. *Proceedings of the National Academy of Sciences of the United States of America*, **2008**, 105, 14424-9	11.5	165
192	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, 7065-70	11.5	164
191	The role of stable α-synuclein oligomers in the molecular events underlying amyloid formation. *Journal of the American Chemical Society*, **2014**, 136, 3859-68	16.4	163
190	Chemical kinetics for drug discovery to combat protein aggregation diseases. *Trends in Pharmacological Sciences*, **2014**, 35, 127-35	13.2	161
189	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, 10328-33	11.5	159
188	Kinetic analysis reveals the diversity of microscopic mechanisms through which molecular chaperones suppress amyloid formation. *Nature Communications*, **2016**, 7, 10948	17.4	153
187	Systematic in vivo analysis of the intrinsic determinants of amyloid Beta pathogenicity. *PLoS Biology*, **2007**, 5, e290	9.7	152
186	Half a century of amyloids: past, present and future. *Chemical Society Reviews*, **2020**, 49, 5473-5509	58.5	142
185	α-Synuclein senses lipid packing defects and induces lateral expansion of lipids leading to membrane remodeling. *Journal of Biological Chemistry*, **2013**, 288, 20883-20895	5.4	141
184	Structural basis of synaptic vesicle assembly promoted by α-synuclein. *Nature Communications*, **2016**, 7, 12563	17.4	139
183	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	11.5	134
182	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	14.3	133
181	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	3.9	132
180	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	11.5	130
179	In situ measurements of the formation and morphology of intracellular α-amyloid fibrils by super-resolution fluorescence imaging. *Journal of the American Chemical Society*, **2011**, 133, 12902-5	16.4	129

178	Toxicity of protein oligomers is rationalized by a function combining size and surface hydrophobicity. *ACS Chemical Biology*, **2014**, 9, 2309-17	4.9	128
177	Direct observation of heterogeneous amyloid fibril growth kinetics via two-color super-resolution microscopy. *Nano Letters*, **2014**, 14, 339-45	11.5	127
176	Cholesterol catalyses A42 aggregation through a heterogeneous nucleation pathway in the presence of lipid membranes. *Nature Chemistry*, **2018**, 10, 673-683	17.6	126
175	Supersaturation is a major driving force for protein aggregation in neurodegenerative diseases. *Trends in Pharmacological Sciences*, **2015**, 36, 72-7	13.2	122
174	Binding of the molecular chaperone B-crystallin to A amyloid fibrils inhibits fibril elongation. *Biophysical Journal*, **2011**, 101, 1681-9	2.9	122
173	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, 12479-84	11.5	121
172	The interaction of alphaB-crystallin with mature alpha-synuclein amyloid fibrils inhibits their elongation. *Biophysical Journal*, **2010**, 98, 843-51	2.9	120
171	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, 14746-51	11.5	108
170	Ca2+ is a key factor in synuclein-induced neurotoxicity. *Journal of Cell Science*, **2016**, 129, 1792-801	5.3	106
169	Interaction of the molecular chaperone DNAJB6 with growing amyloid-beta 42 (A42) aggregates leads to sub-stoichiometric inhibition of amyloid formation. *Journal of Biological Chemistry*, **2014**, 289, 31066-76	5.4	106
168	Dynamics of oligomer populations formed during the aggregation of Alzheimer's A42 peptide. *Nature Chemistry*, **2020**, 12, 445-451	17.6	103
167	Chemical Kinetics for Bridging Molecular Mechanisms and Macroscopic Measurements of Amyloid Fibril Formation. *Annual Review of Physical Chemistry*, **2018**, 69, 273-298	15.7	98
166	Targeting the intrinsically disordered structural ensemble of synuclein by small molecules as a potential therapeutic strategy for Parkinson's disease. *PLoS ONE*, **2014**, 9, e87133	3.7	98
165	Multi-dimensional super-resolution imaging enables surface hydrophobicity mapping. *Nature Communications*, **2016**, 7, 13544	17.4	97
164	A structural ensemble of a ribosome-nascent chain complex during cotranslational protein folding. *Nature Structural and Molecular Biology*, **2016**, 23, 278-285	17.6	96
163	Protein microgels from amyloid fibril networks. *ACS Nano*, **2015**, 9, 43-51	16.7	94
162	Distinct thermodynamic signatures of oligomer generation in the aggregation of the amyloid- peptide. *Nature Chemistry*, **2018**, 10, 523-531	17.6	89
161	Detailed analysis of the energy barriers for amyloid fibril growth. *Angewandte Chemie - International Edition*, **2012**, 51, 5247-51	16.4	88

160	Molecular determinants of the aggregation behavior of alpha- and beta-synuclein. *Protein Science*, **2008**, 17, 887-98	6.3	84
159	Nucleated polymerization with secondary pathways. III. Equilibrium behavior and oligomer populations. *Journal of Chemical Physics*, **2011**, 135, 065107	3.9	82
158	Selective targeting of primary and secondary nucleation pathways in A□42 aggregation using a rational antibody scanning method. *Science Advances*, **2017**, 3, e1700488	14.3	81
157	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, 10949-54	11.5	80
156	Conserved C-terminal charge exerts a profound influence on the aggregation rate of □synuclein. *Journal of Molecular Biology*, **2011**, 411, 329-33	6.5	76
155	A FRET sensor for non-invasive imaging of amyloid formation in vivo. *ChemPhysChem*, **2011**, 12, 673-680	3.2	76
154	Single-Molecule Imaging of Individual Amyloid Protein Aggregates in Human Biofluids. *ACS Chemical Neuroscience*, **2016**, 7, 399-406	5.7	75
153	Secondary nucleation and elongation occur at different sites on Alzheimer's amyloid-□aggregates. *Science Advances*, **2019**, 5, eaau3112	14.3	74
152	The extracellular chaperone clusterin potently inhibits human lysozyme amyloid formation by interacting with prefibrillar species. *Journal of Molecular Biology*, **2007**, 369, 157-67	6.5	74
151	Physical determinants of the self-replication of protein fibrils. *Nature Physics*, **2016**, 12, 874-880	16.2	73
150	Binding affinity of amyloid oligomers to cellular membranes is a generic indicator of cellular dysfunction in protein misfolding diseases. *Scientific Reports*, **2016**, 6, 32721	4.9	73
149	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	11.5	72
148	alpha2-Macroglobulin and haptoglobin suppress amyloid formation by interacting with prefibrillar protein species. *Journal of Biological Chemistry*, **2009**, 284, 4246-54	5.4	72
147	Membrane lipid composition and its physicochemical properties define cell vulnerability to aberrant protein oligomers. *Journal of Cell Science*, **2012**, 125, 2416-27	5.3	72
146	Different soluble aggregates of A□42 can give rise to cellular toxicity through different mechanisms. *Nature Communications*, **2019**, 10, 1541	17.4	71
145	Spatial persistence of angular correlations in amyloid fibrils. *Physical Review Letters*, **2006**, 96, 238301	7.4	69
144	Trodusquemine enhances A□aggregation but suppresses its toxicity by displacing oligomers from cell membranes. *Nature Communications*, **2019**, 10, 225	17.4	69
143	A protein homeostasis signature in healthy brains recapitulates tissue vulnerability to Alzheimer's disease. *Science Advances*, **2016**, 2, e1600947	14.3	68

142	Rare individual amyloid-β oligomers act on astrocytes to initiate neuronal damage. *Biochemistry*, **2014**, 53, 2442-53	3.2	68
141	Fast flow microfluidics and single-molecule fluorescence for the rapid characterization of α-synuclein oligomers. *Analytical Chemistry*, **2015**, 87, 8818-26	7.8	65
140	Silk micrococoons for protein stabilisation and molecular encapsulation. *Nature Communications*, **2017**, 8, 15902	17.4	65
139	Amyloid-β oligomers are sequestered by both intracellular and extracellular chaperones. *Biochemistry*, **2012**, 51, 9270-6	3.2	65
138	The Amyloid Phenomenon and Its Significance in Biology and Medicine. *Cold Spring Harbor Perspectives in Biology*, **2020**, 12,	10.2	65
137	The small heat shock protein Hsp27 binds α-synuclein fibrils, preventing elongation and cytotoxicity. *Journal of Biological Chemistry*, **2018**, 293, 4486-4497	5.4	64
136	A transcriptional signature of Alzheimer'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, 4753-8	11.5	64
135	Hsp70 Inhibits the Nucleation and Elongation of Tau and Sequesters Tau Aggregates with High Affinity. *ACS Chemical Biology*, **2018**, 13, 636-646	4.9	63
134	Sequestration of the Abeta peptide prevents toxicity and promotes degradation in vivo. *PLoS Biology*, **2010**, 8, e1000334	9.7	63
133	Towards a structural biology of the hydrophobic effect in protein folding. *Scientific Reports*, **2016**, 6, 28285	4.9	62
132	Structural Ensembles of Membrane-bound α-Synuclein Reveal the Molecular Determinants of Synaptic Vesicle Affinity. *Scientific Reports*, **2016**, 6, 27125	4.9	62
131	Microfluidic Diffusion Analysis of the Sizes and Interactions of Proteins under Native Solution Conditions. *ACS Nano*, **2016**, 10, 333-41	16.7	61
130	Detergent-like interaction of Congo red with the amyloid beta peptide. *Biochemistry*, **2010**, 49, 1358-60	3.2	61
129	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, 21057-62	11.5	59
128	Nanoscopic insights into seeding mechanisms and toxicity of α-synuclein species in neurons. *Proceedings of the National Academy of Sciences of the United States of America*, **2016**, 113, 3815-9	11.5	57
127	Defining α-synuclein species responsible for Parkinson's disease phenotypes in mice. *Journal of Biological Chemistry*, **2019**, 294, 10392-10406	5.4	55
126	Kinetic diversity of amyloid oligomers. *Proceedings of the National Academy of Sciences of the United States of America*, **2020**, 117, 12087-12094	11.5	55
125	Frequency factors in a landscape model of filamentous protein aggregation. *Physical Review Letters* , **2010**, 104, 228101	7.4	55

124	Protein homeostasis of a metastable subproteome associated with Alzheimer's disease. *Proceedings of the National Academy of Sciences of the United States of America*, **2017**, 114, E5703-E5711	11.5	53
123	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	11.5	52
122	Multistep Inhibition of Synuclein Aggregation and Toxicity in Vitro and in Vivo by Trodusquemine. *ACS Chemical Biology*, **2018**, 13, 2308-2319	4.9	52
121	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	7.5	51
120	Ultrasensitive Measurement of Ca Influx into Lipid Vesicles Induced by Protein Aggregates. *Angewandte Chemie - International Edition*, **2017**, 56, 7750-7754	16.4	51
119	Kinetic modelling indicates that fast-translating codons can coordinate cotranslational protein folding by avoiding misfolded intermediates. *Nature Communications*, **2014**, 5, 2988	17.4	50
118	A simple lattice model that captures protein folding, aggregation and amyloid formation. *PLoS ONE* , **2014**, 9, e85185	3.7	50
117	Twisting transition between crystalline and fibrillar phases of aggregated peptides. *Physical Review Letters*, **2012**, 109, 158101	7.4	47
116	Nanobodies raised against monomeric ?-synuclein inhibit fibril formation and destabilize toxic oligomeric species. *BMC Biology*, **2017**, 15, 57	7.3	46
115	Inhibition of Synuclein Fibril Elongation by Hsp70 Is Governed by a Kinetic Binding Competition between Synuclein Species. *Biochemistry*, **2017**, 56, 1177-1180	3.2	45
114	Synuclein suppresses both the initiation and amplification steps of Synuclein aggregation via competitive binding to surfaces. *Scientific Reports*, **2016**, 6, 36010	4.9	45
113	Latent analysis of unmodified biomolecules and their complexes in solution with attomole detection sensitivity. *Nature Chemistry*, **2015**, 7, 802-9	17.6	44
112	Intrinsic determinants of neurotoxic aggregate formation by the amyloid beta peptide. *Biophysical Journal*, **2010**, 98, 1677-84	2.9	44
111	Scaling behaviour and rate-determining steps in filamentous self-assembly. *Chemical Science*, **2017**, 8, 7087-7097	9.4	43
110	Modulation of electrostatic interactions to reveal a reaction network unifying the aggregation behaviour of the A42 peptide and its variants. *Chemical Science*, **2017**, 8, 4352-4362	9.4	42
109	Single-Molecule Characterization of the Interactions between Extracellular Chaperones and Toxic Synuclein Oligomers. *Cell Reports*, **2018**, 23, 3492-3500	10.6	42
108	Mapping Surface Hydrophobicity of Synuclein Oligomers at the Nanoscale. *Nano Letters*, **2018**, 18, 7494-7501	11.5	42
107	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	11.5	41

106	Hydrophobicity and conformational change as mechanistic determinants for nonspecific modulators of amyloid -self-assembly. *Biochemistry*, **2012**, 51, 126-37	3.2	40
105	Structural characterization of the interaction of -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, 5012-7	11.5	40
104	The release of toxic oligomers from -synuclein fibrils induces dysfunction in neuronal cells. *Nature Communications*, **2021**, 12, 1814	17.4	39
103	Proteasome-targeted nanobodies alleviate pathology and functional decline in an -synuclein-based Parkinson's disease model. *Npj Parkinson's Disease*, **2018**, 4, 25	9.7	38
102	Quantitative thermophoretic study of disease-related protein aggregates. *Scientific Reports*, **2016**, 6, 22829	4.9	37
101	Enzymatically Active Microgels from Self-Assembling Protein Nanofibrils for Microflow Chemistry. *ACS Nano*, **2015**, 9, 5772-81	16.7	36
100	Massively parallel C. elegans tracking provides multi-dimensional fingerprints for phenotypic discovery. *Journal of Neuroscience Methods*, **2018**, 306, 57-67	3	35
99	Kinetic fingerprints differentiate the mechanisms of action of anti-A -antibodies. *Nature Structural and Molecular Biology*, **2020**, 27, 1125-1133	17.6	35
98	Nucleation-conversion-polymerization reactions of biological macromolecules with prenucleation clusters. *Physical Review E*, **2014**, 89, 032712	2.4	34
97	Expression in drosophila of tandem amyloid -peptides provides insights into links between aggregation and neurotoxicity. *Journal of Biological Chemistry*, **2012**, 287, 20748-54	5.4	34
96	C-terminal truncation of -synuclein promotes amyloid fibril amplification at physiological pH. *Chemical Science*, **2018**, 9, 5506-5516	9.4	34
95	Stabilization and Characterization of Cytotoxic A -Oligomers Isolated from an Aggregation Reaction in the Presence of Zinc Ions. *ACS Chemical Neuroscience*, **2018**, 9, 2959-2971	5.7	33
94	Determination of the structures of distinct transition state ensembles for a -sheet peptide with parallel folding pathways. *Journal of Chemical Physics*, **2002**, 117, 9510-9517	3.9	33
93	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	11.5	32
92	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	16.4	31
91	Quantifying Co-Oligomer Formation by -Synuclein. *ACS Nano*, **2018**, 12, 10855-10866	16.7	30
90	A Fragment-Based Method of Creating Small-Molecule Libraries to Target the Aggregation of Intrinsically Disordered Proteins. *ACS Combinatorial Science*, **2016**, 18, 144-53	3.9	29
89	Small-molecule sequestration of amyloid- as a drug discovery strategy for Alzheimer's disease. *Science Advances*, **2020**, 6,	14.3	28

88	Transthyretin Inhibits Primary and Secondary Nucleations of Amyloid-Peptide Aggregation and Reduces the Toxicity of Its Oligomers. *Biomacromolecules*, **2020**, 21, 1112-1125	6.9	28
87	Optical Structural Analysis of Individual Synuclein Oligomers. *Angewandte Chemie - International Edition*, **2018**, 57, 4886-4890	16.4	27
86	Monomeric and fibrillar synuclein exert opposite effects on the catalytic cycle that promotes the proliferation of A42 aggregates. *Proceedings of the National Academy of Sciences of the United States of America*, **2017**, 114, 8005-8010	11.5	27
85	Rational design of a conformation-specific antibody for the quantification of Aoligomers. *Proceedings of the National Academy of Sciences of the United States of America*, **2020**, 117, 13509-13518	11.5	26
84	Structure of a low-population intermediate state in the release of an enzyme product. *ELife*, **2015**, 4,	8.9	25
83	Observation of an synuclein liquid droplet state and its maturation into Lewy body-like assemblies. *Journal of Molecular Cell Biology*, **2021**, 13, 282-294	6.3	25
82	The Toxicity of Misfolded Protein Oligomers Is Independent of Their Secondary Structure. *ACS Chemical Biology*, **2019**, 14, 1593-1600	4.9	24
81	The Influence of Pathogenic Mutations in Synuclein on Biophysical and Structural Characteristics of Amyloid Fibrils. *ACS Nano*, **2020**, 14, 5213-5222	16.7	24
80	Trodusquemine displaces protein misfolded oligomers from cell membranes and abrogates their cytotoxicity through a generic mechanism. *Communications Biology*, **2020**, 3, 435	6.7	23
79	The N-terminal Acetylation of Synuclein Changes the Affinity for Lipid Membranes but not the Structural Properties of the Bound State. *Scientific Reports*, **2020**, 10, 204	4.9	22
78	Hamiltonian Dynamics of Protein Filament Formation. *Physical Review Letters*, **2016**, 116, 038101	7.4	22
77	Nanoscopic Characterisation of Individual Endogenous Protein Aggregates in Human Neuronal Cells. *ChemBioChem*, **2018**, 19, 2033-2038	3.8	21
76	Structural differences between toxic and nontoxic HypF-N oligomers. *Chemical Communications*, **2018**, 54, 8637-8640	5.8	21
75	Synthesis of Nonequilibrium Supramolecular Peptide Polymers on a Microfluidic Platform. *Journal of the American Chemical Society*, **2016**, 138, 9589-96	16.4	21
74	Probing the Origin of the Toxicity of Oligomeric Aggregates of Synuclein with Antibodies. *ACS Chemical Biology*, **2019**, 14, 1352-1362	4.9	20
73	Molecular determinants of the interaction of EGCG with ordered and disordered proteins. *Biopolymers*, **2018**, 109, e23117	2.2	20
72	Particle-Based Monte-Carlo Simulations of Steady-State Mass Transport at Intermediate Péclet Numbers. *International Journal of Nonlinear Sciences and Numerical Simulation*, **2016**, 17, 175-183	1.8	20
71	A rationally designed six-residue swap generates comparability in the aggregation behavior of synuclein and synuclein. *Biochemistry*, **2012**, 51, 8771-8	3.2	20

70	Gradient-free determination of isoelectric points of proteins on chip. *Physical Chemistry Chemical Physics*, **2017**, 19, 23060-23067	3.6	19
69	Lipid Dynamics and Phase Transition within Synuclein Amyloid Fibrils. *Journal of Physical Chemistry Letters*, **2019**, 10, 7872-7877	6.4	19
68	Microfluidic deposition for resolving single-molecule protein architecture and heterogeneity. *Nature Communications*, **2018**, 9, 3890	17.4	19
67	Force generation by the growth of amyloid aggregates. *Proceedings of the National Academy of Sciences of the United States of America*, **2015**, 112, 9524-9	11.5	18
66	ThX - a next-generation probe for the early detection of amyloid aggregates. *Chemical Science*, **2020**, 11, 4578-4583	9.4	18
65	Exploring the role of post-translational modifications in regulating synuclein interactions by studying the effects of phosphorylation on nanobody binding. *Protein Science*, **2018**, 27, 1262-1274	6.3	18
64	Effect of molecular chaperones on aberrant protein oligomers in vitro: super-versus sub-stoichiometric chaperone concentrations. *Biological Chemistry*, **2016**, 397, 401-15	4.5	18
63	Fast Fluorescence Lifetime Imaging Reveals the Aggregation Processes of Synuclein and Polyglutamine in Aging. *ACS Chemical Biology*, **2019**, 14, 1628-1636	4.9	17
62	The metastability of the proteome of spinal motor neurons underlies their selective vulnerability in ALS. *Neuroscience Letters*, **2019**, 704, 89-94	3.3	17
61	Supersaturated proteins are enriched at synapses and underlie cell and tissue vulnerability in Alzheimer's disease. *Heliyon*, **2019**, 5, e02589	3.6	17
60	Microfluidic Diffusion Platform for Characterizing the Sizes of Lipid Vesicles and the Thermodynamics of Protein-Lipid Interactions. *Analytical Chemistry*, **2018**, 90, 3284-3290	7.8	16
59	Structural Effects of Two Camelid Nanobodies Directed to Distinct C-Terminal Epitopes on Synuclein. *Biochemistry*, **2016**, 55, 3116-22	3.2	16
58	Microfluidic approaches for probing amyloid assembly and behaviour. *Lab on A Chip*, **2018**, 18, 999-1016	7.2	15
57	Structure-Free Validation of Residual Dipolar Coupling and Paramagnetic Relaxation Enhancement Measurements of Disordered Proteins. *Biochemistry*, **2015**, 54, 6876-86	3.2	15
56	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	11.5	15
55	Amyloid-like Fibrils from an Helical Transmembrane Protein. *Biochemistry*, **2017**, 56, 3225-3233	3.2	14
54	Inhibiting the Ca Influx Induced by Human CSF. *Cell Reports*, **2017**, 21, 3310-3316	10.6	14
53	Measuring the kinetics of amyloid fibril elongation using quartz crystal microbalances. *Methods in Molecular Biology*, **2012**, 849, 101-19	1.4	14

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