Hilal A Lashuel

List of Publications by Year
in descending order

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128 papers	14,495 citations	28190 55 h-index	20900 115 g-index
158 all docs	158 docs citations	158 times ranked	13605 citing authors

#	Article	IF	Citations
1	Non-monotonic fibril surface occlusion by GFP tags from coarse-grained molecular simulations. Computational and Structural Biotechnology Journal, 2022, 20, 309-321.	1.9	4
2	Revisiting the grammar of Tau aggregation and pathology formation: how new insights from brain pathology are shaping how we study and target Tauopathies. Chemical Society Reviews, 2022, 51, 513-565.	18.7	68
3	Pathological Relevance of Post-Translationally Modified Alpha-Synuclein (pSer87, pSer129, nTyr39) in Idiopathic Parkinsonâ€™s Disease and Multiple System Atrophy. Cells, 2022, 11, 906.	1.8	14
4	Remembering John Q Trojanowski, in his own words: A life dedicated to discovering building blocks and using them to build bridges of knowledge, collaboration, and discovery. Npj Parkinson's Disease, 2022, 8, 43.	2.5	1
5	A NAC domain mutation (E83Q) unlocks the pathogenicity of human alpha-synuclein and recapitulates its pathological diversity. Science Advances, 2022, 8, eabn0044.	4.7	20
6	Structural Basis of Huntingtin Fibril Polymorphism Revealed by Cryogenic Electron Microscopy of Exon 1 HTT Fibrils. Journal of the American Chemical Society, 2022, 144, 10723-10735.	6.6	15
7	On-Demand Nanoliter Sampling Probe for the Collection of Brain Fluid. Analytical Chemistry, 2022, 94, 10415-10426.	3.2	1
8	Monitoring alphaâ€synuclein oligomerization and aggregation using bimolecular fluorescence complementation assays: What you see is not always what you get. Journal of Neurochemistry, 2021, 157, 872-888.	2.1	18
9	Alphaâ€Synuclein oligomerization and aggregation: All models are useful but only if we know what they model. Journal of Neurochemistry, 2021, 157, 891-898.	2.1	5
10	Siteâ€Specific Phosphorylation of Huntingtin Exonâ€…1 Recombinant Proteins Enabled by the Discovery of Novel Kinases. ChemBioChem, 2021, 22, 217-231.	1.3	18
11	Reverse engineering Lewy bodies: how far have we come and how far can we go?. Nature Reviews Neuroscience, 2021, 22, 111-131.	4.9	104
12	Hypoxia Conditioning as a Promising Therapeutic Target in Parkinson's Disease?. Movement Disorders, 2021, 36, 857-861.	2.2	26
13	Correlative light and electron microscopy suggests that mutant huntingtin dysregulates the endolysosomal pathway in presymptomatic Huntingtonâ€™s disease. Acta Neuropathologica Communications, 2021, 9, 70.	2.4	7
14	Enforced dimerization between XBP1s and ATF6f enhances the protective effects of the UPR in models of neurodegeneration. Molecular Therapy, 2021, 29, 1862-1882.	3.7	25
15	Parkinson mice show functional and molecular changes in the gut long before motoric disease onset. Molecular Neurodegeneration, 2021, 16, 34.	4.4	29
16	A New Chemoenzymatic Semisynthetic Approach Provides Insight into the Role of Phosphorylation beyond Exon1 of Huntingtin and Reveals N-Terminal Fragment Length-Dependent Distinct Mechanisms of Aggregation. Journal of the American Chemical Society, 2021, 143, 9798-9812.	6.6	8
17	Alpha-synuclein research: defining strategic moves in the battle against Parkinsonâ€™s disease. Npj Parkinson's Disease, 2021, 7, 65.	2.5	74
18	Investigating Crosstalk Among PTMs Provides Novel Insight Into the Structural Basis Underlying the Differential Effects of Nt17 PTMs on Mutant Httex1 Aggregation. Frontiers in Molecular Biosciences, 2021, 8, 686086.	1.6	8

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19	Lewy body-associated proteins: victims, instigators, or innocent bystanders? The case of AIMP2 and alpha-synuclein. Neurobiology of Disease, 2021, 156, 105417.	2.1	4
20	Pharmacological characterization of mutant huntingtin aggregate-directed PET imaging tracer candidates. Scientific Reports, 2021, 11, 17977.	1.6	16
21	Rethinking protein aggregation and drug discovery in neurodegenerative diseases: Why we need to embrace complexity?. Current Opinion in Chemical Biology, 2021, 64, 67-75.	2.8	40
22	The Nt17 Domain and its Helical Conformation Regulate the Aggregation, Cellular Properties and Neurotoxicity of Mutant Huntingtin Exon 1. Journal of Molecular Biology, 2021, 433, 167222.	2.0	15
23	Fatal attraction â€“ The role of hypoxia when alpha-synuclein gets intimate with mitochondria. Neurobiology of Aging, 2021, 107, 128-141.	1.5	11
24	To target Tau pathologies, we must embrace and reconstruct their complexities. Neurobiology of Disease, 2021, 161, 105536.	2.1	20
25	Nuclear and cytoplasmic huntingtin inclusions exhibit distinct biochemical composition, interactome and ultrastructural properties. Nature Communications, 2021, 12, 6579.	5.8	42
26	Ultrasensitive quantitative measurement of huntingtin phosphorylation at residue S13. Biochemical and Biophysical Research Communications, 2020, 521, 549-554.	1.0	14
27	A simple, versatile and robust centrifugationâ€based filtration protocol for the isolation and quantification of Î±â€synuclein monomers, oligomers and fibrils: Towards improving experimental reproducibility in Î±â€synuclein research. Journal of Neurochemistry, 2020, 153, 103-119.	2.1	44
28	Siteâ€Specific Hyperphosphorylation Inhibits, Rather than Promotes, Tau Fibrillization, Seeding Capacity, and Its Microtubule Binding. Angewandte Chemie, 2020, 132, 4088-4096.	1.6	11
29	Siteâ€Specific Hyperphosphorylation Inhibits, Rather than Promotes, Tau Fibrillization, Seeding Capacity, and Its Microtubule Binding. Angewandte Chemie - International Edition, 2020, 59, 4059-4067.	7.2	56
30	How specific are the conformation-specific Î±-synuclein antibodies? Characterization and validation of 16 Î±-synuclein conformation-specific antibodies using well-characterized preparations of Î±-synuclein monomers, fibrils and oligomers with distinct structures and morphology. Neurobiology of Disease, 2020, 146, 105086.	2.1	95
31	Extent of N-terminus exposure of monomeric alpha-synuclein determines its aggregation propensity. Nature Communications, 2020, 11, 2820.	5.8	99
32	Phosphorylation of the overlooked tyrosine 310 regulates the structure, aggregation, and microtubule- and lipid-binding properties of Tau. Journal of Biological Chemistry, 2020, 295, 7905-7922.	1.6	32
33	Education and research are essential for lasting peace in Yemen. Lancet, The, 2020, 395, 1114.	6.3	1
34	Do Lewy bodies contain alpha-synuclein fibrils? and Does it matter? A brief history and critical analysis of recent reports. Neurobiology of Disease, 2020, 141, 104876.	2.1	71
35	Unraveling the complexity of amyloid polymorphism using gold nanoparticles and cryo-EM. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 6866-6874.	3.3	54
36	The process of Lewy body formation, rather than simply Î±-synuclein fibrillization, is one of the major drivers of neurodegeneration. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 4971-4982.	3.3	422

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37	Pronounced Î±-Synuclein Pathology in a Seeding-Based Mouse Model Is Not Sufficient to Induce Mitochondrial Respiration Deficits in the Striatum and Amygdala. ENeuro, 2020, 7, ENEURO.0110-20.2020.	0.9	8
38	TBK1 phosphorylates mutant Huntingtin and suppresses its aggregation and toxicity in Huntington's disease models. EMBO Journal, 2020, 39, e104671.	3.5	34
39	What about faculty?. ELife, 2020, 9, .	2.8	27
40	Antibodyâ€based methods for the measurement of Î±â€synuclein concentration in human cerebrospinal fluid â€“ method comparison and round robin study. Journal of Neurochemistry, 2019, 149, 126-138.	2.1	44
41	Chronic corticosterone aggravates behavioral and neuronal symptomatology in a mouse model of alpha-synuclein pathology. Neurobiology of Aging, 2019, 83, 11-20.	1.5	32
42	Phospho-S129 Alpha-Synuclein Is Present in Human Plasma but Not in Cerebrospinal Fluid as Determined by an Ultrasensitive Immunoassay. Frontiers in Neuroscience, 2019, 13, 889.	1.4	25
43	The Role of Post-translational Modifications on the Energy Landscape of Huntingtin N-Terminus. Frontiers in Molecular Biosciences, 2019, 6, 95.	1.6	19
44	Single Posttranslational Modifications in the Central Repeat Domains of Tau4 Impact its Aggregation and Tubulin Binding. Angewandte Chemie, 2019, 131, 1630-1634.	1.6	11
45	Î±-Synuclein O-GlcNAcylation alters aggregation and toxicity, revealing certain residues as potential inhibitors of Parkinsonâ€™s disease. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 1511-1519.	3.3	156
46	Single Posttranslational Modifications in the Central Repeat Domains of Tau4 Impact its Aggregation and Tubulin Binding. Angewandte Chemie - International Edition, 2019, 58, 1616-1620.	7.2	38
47	Protein Semisynthesis Provides Access to Tau Disease-Associated Post-translational Modifications (PTMs) and Paves the Way to Deciphering the Tau PTM Code in Health and Diseased States. Journal of the American Chemical Society, 2018, 140, 6611-6621.	6.6	82
48	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
49	O2â€09â€04: HARMONIZATION OF IMMUNOCHEMICAL METHODS FOR MEASUREMENT OF Î±â€SYNUCLEIN IN HUMAN CEREBROSPINAL FLUID: A ROUND ROBIN STUDY APPROACH. Alzheimer's and Dementia, 2018, 14, P642.	0.4	0
50	N-terminal Huntingtin (Htt) phosphorylation is a molecular switch regulating Htt aggregation, helical conformation, internalization, and nuclear targeting. Journal of Biological Chemistry, 2018, 293, 18540-18558.	1.6	63
51	Real-Time In Situ Secondary Structure Analysis of Protein Monolayer with Mid-Infrared Plasmonic Nanoantennas. ACS Sensors, 2018, 3, 1109-1117.	4.0	51
52	Generation of Native, Untagged Huntingtin Exon1 Monomer and Fibrils Using a SUMO Fusion Strategy. Journal of Visualized Experiments, 2018, , .	0.2	14
53	Identification and nanomechanical characterization of the fundamental single-strand protofilaments of amyloid Î±-synuclein fibrils. Proceedings of the National Academy of Sciences of the United States of America, 2018, 115, 7230-7235.	3.3	96
54	Resolving molecule-specific information in dynamic lipid membrane processes with multi-resonant infrared metasurfaces. Nature Communications, 2018, 9, 2160.	5.8	176

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55	Glycation potentiates Î±-synuclein-associated neurodegeneration in synucleinopathies. Brain, 2017, 140, 1399-1419.	3.7	153
56	Frontispiece: Mutant Exon1 Huntingtin Aggregation is Regulated by T3 Phosphorylationâ€Induced Structural Changes and Crosstalk between T3 Phosphorylation and Acetylation at K6. Angewandte Chemie - International Edition, 2017, 56, .	7.2	6
57	Frontispiz: Mutant Exon1 Huntingtin Aggregation is Regulated by T3 Phosphorylationâ€Induced Structural Changes and Crosstalk between T3 Phosphorylation and Acetylation at K6. Angewandte Chemie, 2017, 129, .	1.6	1
58	Mutant Exon1 Huntingtin Aggregation is Regulated by T3 Phosphorylationâ€Induced Structural Changes and Crosstalk between T3 Phosphorylation and Acetylation at K6. Angewandte Chemie, 2017, 129, 5286-5291.	1.6	2
59	Mutant Exon1 Huntingtin Aggregation is Regulated by T3 Phosphorylationâ€Induced Structural Changes and Crosstalk between T3 Phosphorylation and Acetylation at K6. Angewandte Chemie - International Edition, 2017, 56, 5202-5207.	7.2	54
60	Membrane scission driven by the PROPPIN Atg18. EMBO Journal, 2017, 36, 3274-3291.	3.5	68
61	Amyloid single-cell cytotoxicity assays by nanomotion detection. Cell Death Discovery, 2017, 3, 17053.	2.0	20
62	Monomeric Huntingtin Exon 1 Has Similar Overall Structural Features for Wild-Type and Pathological Polyglutamine Lengths. Journal of the American Chemical Society, 2017, 139, 14456-14469.	6.6	87
63	A user's guide for Î±â€synuclein biomarker studies in biological fluids: Perianalytical considerations. Movement Disorders, 2017, 32, 1117-1130.	2.2	54
64	Phosphorylation of huntingtin at residue T3 is decreased in Huntingtonâ€™s disease and modulates mutant huntingtin protein conformation. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, E10809-E10818.	3.3	63
65	Discovery and characterization of stable and toxic Tau/phospholipid oligomeric complexes. Nature Communications, 2017, 8, 1678.	5.8	117
66	Polyglutamine expansion affects huntingtin conformation in multiple Huntingtonâ€™s disease models. Scientific Reports, 2017, 7, 5070.	1.6	32
67	Activation of the STING-Dependent Type I Interferon Response Reduces Microglial Reactivity and Neuroinflammation. Neuron, 2017, 96, 1290-1302.e6.	3.8	107
68	Nanoplasmonic mid-infrared biosensor for in vitro protein secondary structure detection. Light: Science and Applications, 2017, 6, e17029-e17029.	7.7	93
69	Microtubuleâ€Binding R3 Fragment from Tau Selfâ€Assembles into Giant Multistranded Amyloid Ribbons. Angewandte Chemie - International Edition, 2016, 55, 618-622.	7.2	43
70	A New Cagedâ€Glutamine Derivative as a Tool To Control the Assembly of Glutamineâ€Containing Amyloidogenic Peptides. ChemBioChem, 2016, 17, 2353-2360.	1.3	8
71	An Intein-based Strategy for the Production of Tag-free Huntingtin Exon 1 Proteins Enables New Insights into the Polyglutamine Dependence of Httex1 Aggregation and Fibril Formation. Journal of Biological Chemistry, 2016, 291, 12074-12086.	1.6	30
72	Semisynthetic and in Vitro Phosphorylation of Alpha-Synuclein at Y39 Promotes Functional Partly Helical Membrane-Bound States Resembling Those Induced by PD Mutations. ACS Chemical Biology, 2016, 11, 2428-2437.	1.6	64

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73	Induction of de novo Î±-synuclein fibrillization in a neuronal model for Parkinsonâ€™s disease. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, E912-21.	3.3	95
74	Health hazards of methylammonium lead iodide based perovskites: cytotoxicity studies. Toxicology Research, 2016, 5, 407-419.	0.9	113
75	Semisynthesis and Enzymatic Preparation of Post-translationally Modified Î±-Synuclein. Methods in Molecular Biology, 2016, 1345, 3-20.	0.4	25
76	Influence of the Î²â€sheet Content on the Mechanical Properties of Aggregates during Amyloid Fibrillization. Angewandte Chemie, 2015, 127, 2492-2496.	1.6	22
77	Detection of huntingtin exon 1 phosphorylation by Phos-Tag SDS-PAGE: Predominant phosphorylation on threonine 3 and regulation by IKKÎ². Biochemical and Biophysical Research Communications, 2015, 463, 1317-1322.	1.0	21
78	Elucidating the Role of Site-Specific Nitration of Î±-Synuclein in the Pathogenesis of Parkinsonâ€™s Disease via Protein Semisynthesis and Mutagenesis. Journal of the American Chemical Society, 2015, 137, 5041-5052.	6.6	131
79	Parkinson Disease Mutant E46K Enhances Î±-Synuclein Phosphorylation in Mammalian Cell Lines, in Yeast, and in Vivo. Journal of Biological Chemistry, 2015, 290, 9412-9427.	1.6	52
80	Structural differences of amyloid-Î² fibrils revealed by antibodies from phage display. BMC Biotechnology, 2015, 15, 57.	1.7	12
81	Photobiomodulation Suppresses Alpha-Synuclein-Induced Toxicity in an AAV-Based Rat Genetic Model of Parkinsonâ€™s Disease. PLoS ONE, 2015, 10, e0140880.	1.1	62
82	c-Abl phosphorylates Î±-synuclein and regulates its degradation: implication for Î±-synuclein clearance and contribution to the pathogenesis of Parkinson's disease. Human Molecular Genetics, 2014, 23, 2858-2879.	1.4	176
83	The novel Parkinson's disease linked mutation G51D attenuates in vitro aggregation and membrane binding of Â-synuclein, and enhances its secretion and nuclear localization in cells. Human Molecular Genetics, 2014, 23, 4491-4509.	1.4	194
84	The H50Q Mutation Enhances Î±-Synuclein Aggregation, Secretion, and Toxicity. Journal of Biological Chemistry, 2014, 289, 21856-21876.	1.6	152
85	Oneâ€Pot Semisynthesis of Exonâ€…1 of the Huntingtin Protein: New Tools for Elucidating the Role of Posttranslational Modifications in the Pathogenesis of Huntingtonâ€™s Disease. Angewandte Chemie - International Edition, 2014, 53, 1928-1933.	7.2	48
86	One-pot total chemical synthesis of human Î±-synuclein. Chemical Communications, 2013, 49, 9254.	2.2	40
87	Synthetic polyubiquitinated Î±-Synuclein reveals important insights into the roles of the ubiquitin chain in regulating its pathophysiology. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 17726-17731.	3.3	130
88	Alpha-synuclein Post-translational Modifications as Potential Biomarkers for Parkinson Disease and Other Synucleinopathies. Molecular and Cellular Proteomics, 2013, 12, 3543-3558.	2.5	159
89	Oxidative and nitrative alphaâ€synuclein modifications and proteostatic stress: implications for disease mechanisms and interventions in synucleinopathies. Journal of Neurochemistry, 2013, 125, 491-511.	2.1	116
90	The many faces of Î±-synuclein: from structure and toxicity to therapeutic target. Nature Reviews Neuroscience, 2013, 14, 38-48.	4.9	1,322

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91	Discovery of a Novel Aggregation Domain in the Huntingtin Protein: Implications for the Mechanisms of Htt Aggregation and Toxicity. Angewandte Chemie - International Edition, 2013, 52, 562-567.	7.2	11
92	Polo-like kinase 2 regulates selective autophagic Î±-synuclein clearance and suppresses its toxicity in vivo. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, E3945-54.	3.3	160
93	Mimicking Phosphorylation at Serine 87 Inhibits the Aggregation of Human Î±-Synuclein and Protects against Its Toxicity in a Rat Model of Parkinson's Disease. Journal of Neuroscience, 2012, 32, 1536-1544.	1.7	84
94	Characterization of Semisynthetic and Naturally NÎ±-Acetylated Î±-Synuclein in Vitro and in Intact Cells. Journal of Biological Chemistry, 2012, 287, 28243-28262.	1.6	148
95	Elucidating the Role of C-Terminal Post-Translational Modifications Using Protein Semisynthesis Strategies: Î±-Synuclein Phosphorylation at Tyrosine 125. Journal of the American Chemical Society, 2012, 134, 5196-5210.	6.6	95
96	Chemical Strategies for Controlling Protein Folding and Elucidating the Molecular Mechanisms of Amyloid Formation and Toxicity. Journal of Molecular Biology, 2012, 421, 204-236.	2.0	27
97	The Size of the Proteasomal Substrate Determines Whether Its Degradation Will Be Mediated by Mono- or Polyubiquitylation. Molecular Cell, 2012, 48, 87-97.	4.5	141
98	Î±-Synuclein in Central Nervous System and from Erythrocytes, Mammalian Cells, and Escherichia coli Exists Predominantly as Disordered Monomer. Journal of Biological Chemistry, 2012, 287, 15345-15364.	1.6	466
99	Phosphorylation of Î±-Synuclein at Y125 and S129 Alters Its Metal Binding Properties: Implications for Understanding the Role of Î±-Synuclein in the Pathogenesis of Parkinsonâ€™s Disease and Related Disorders. ACS Chemical Neuroscience, 2011, 2, 667-675.	1.7	97
100	Towards Elucidation of the Role of Ubiquitination in the Pathogenesis of Parkinsonâ€™s Disease with Semisynthetic Ubiquitinated Î±â€Synuclein. Angewandte Chemie - International Edition, 2011, 50, 405-409.	7.2	109
101	Amyloidogenic Proteinâ€“Membrane Interactions: Mechanistic Insight from Model Systems. Angewandte Chemie - International Edition, 2010, 49, 5628-5654.	7.2	529
102	Amyloid-Î² Aggregates Cause Alterations of Astrocytic Metabolic Phenotype: Impact on Neuronal Viability. Journal of Neuroscience, 2010, 30, 3326-3338.	1.7	252
103	Phosphorylation at S87 Is Enhanced in Synucleinopathies, Inhibits Î±-Synuclein Oligomerization, and Influences Synuclein-Membrane Interactions. Journal of Neuroscience, 2010, 30, 3184-3198.	1.7	271
104	Role of post-translational modifications in modulating the structure, function and toxicity of Î±-synuclein. Progress in Brain Research, 2010, 183, 115-145.	0.9	283
105	Phosphorylation of Synucleins by Members of the Polo-like Kinase Family. Journal of Biological Chemistry, 2010, 285, 2807-2822.	1.6	204
106	Amyloids Go Genomic: Insights Regarding the Sequence Determinants of Prion Formation from Genomeâ€Wide Studies. ChemBioChem, 2009, 10, 1951-1954.	1.3	5
107	Highly Efficient and Chemoselective Peptide Ubiquitylation. Angewandte Chemie - International Edition, 2009, 48, 8090-8094.	7.2	241
108	Structural Properties of Pore-Forming Oligomers of Î±-Synuclein. Journal of the American Chemical Society, 2009, 131, 17482-17489.	6.6	191

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109	Switchâ€Peptides: Design and Characterization of Controllable Superâ€Amyloidâ€Forming Hostâ€“Guest Peptides as Tools for Identifying Antiâ€Amyloid Agents. ChemBioChem, 2008, 9, 2104-2112.	1.3	24
110	Switch Peptide via Staudinger Reaction. Organic Letters, 2008, 10, 5243-5246.	2.4	38
111	Phosphorylation at Ser-129 but Not the Phosphomimics S129E/D Inhibits the Fibrillation of Î±-Synuclein. Journal of Biological Chemistry, 2008, 283, 16895-16905.	1.6	302
112	The Ratio of Monomeric to Aggregated Forms of AÎ²40 and AÎ²42 Is an Important Determinant of Amyloid-Î² Aggregation, Fibrillogenesis, and Toxicity. Journal of Biological Chemistry, 2008, 283, 28176-28189.	1.6	237
113	Inhibition of Î±-Synuclein Fibrillization by Dopamine Is Mediated by Interactions with Five C-Terminal Residues and with E83 in the NAC Region. PLoS ONE, 2008, 3, e3394.	1.1	106
114	The Impact of the E46K Mutation on the Properties of Î±-Synuclein in Its Monomeric and Oligomeric States. Biochemistry, 2007, 46, 7107-7118.	1.2	198
115	Switch-peptides as folding precursors in self-assembling peptides and amyloid fibrillogenesis. Biopolymers, 2007, 88, 239-252.	1.2	38
116	Disruption of Amyloid-Derived Peptide Assemblies through the Controlled Induction of a Î²-Sheet to Î±-Helix Transformation: Application of the Switch Concept. Angewandte Chemie - International Edition, 2007, 46, 2681-2684.	7.2	67
117	Are amyloid diseases caused by protein aggregates that mimic bacterial pore-forming toxins?. Quarterly Reviews of Biophysics, 2006, 39, 167-201.	2.4	365
118	Switch-Peptides: From Conformational Studies to Alzheimer's Disease. Chimia, 2006, 60, 199-202.	0.3	17
119	A century-old debate on protein aggregation and neurodegeneration enters the clinic. Nature, 2006, 443, 774-779.	13.7	621
120	Molecular Electron Microscopy Approaches to Elucidating the Mechanisms of Protein Fibrillogenesis. , 2005, 299, 081-102.		14
121	In Vitro Preparation of Prefibrillar Intermediates of Amyloid-Î² and Î±-Synuclein. , 2005, 299, 019-034.		20
122	Membrane Permeabilization: A Common Mechanism in Protein-Misfolding Diseases. Science of Aging Knowledge Environment: SAGE KE, 2005, 2005, pe28-pe28.	0.9	33
123	AÎ² Protofibrils Possess a Stable Core Structure Resistant to Hydrogen Exchangeâ€. Biochemistry, 2003, 42, 14092-14098.	1.2	127
124	Mixtures of Wild-type and a Pathogenic (E22G) Form of AÎ²40 in Vitro Accumulate Protofibrils, Including Amyloid Pores. Journal of Molecular Biology, 2003, 332, 795-808.	2.0	229
125	New Class of Inhibitors of Amyloid-Î² Fibril Formation. Journal of Biological Chemistry, 2002, 277, 42881-42890.	1.6	133
126	Î±-Synuclein, Especially the Parkinson's Disease-associated Mutants, Forms Pore-like Annular and Tubular Protofibrils. Journal of Molecular Biology, 2002, 322, 1089-1102.	2.0	772

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127	Amyloid pores from pathogenic mutations. Nature, 2002, 418, 291-291.	13.7	1,182
128	Protofilaments, Filaments, Ribbons, and Fibrils from Peptidomimetic Self-Assembly:Â Implications for Amyloid Fibril Formation and Materials Science. Journal of the American Chemical Society, 2000, 122, 5262-5277.	6.6	286