Hilal A Lashuel

List of Publications by Year in Descending Order

131 papers	10,913 citations	48 h-index	104 g-index
158 ext. papers	12,932 ext. citations	9.6 avg, IF	6.61 L-index

#	Paper	IF	Citations
131	Non-monotonic fibril surface occlusion by GFP tags from coarse-grained molecular simulations.. *Computational and Structural Biotechnology Journal*, **2022**, 20, 309-321	6.8	0
130	A NAC domain mutation (E83Q) unlocks the pathogenicity of human alpha-synuclein and recapitulates its pathological diversity.. *Science Advances*, **2022**, 8, eabn0044	14.3	0
129	Nuclear and cytoplasmic huntingtin inclusions exhibit distinct biochemical composition, interactome and ultrastructural properties. *Nature Communications*, **2021**, 12, 6579	17.4	4
128	To target Tau pathologies, we must embrace and reconstruct their complexities. *Neurobiology of Disease*, **2021**, 161, 105536	7.5	3
127	Correlative light and electron microscopy suggests that mutant huntingtin dysregulates the endolysosomal pathway in presymptomatic Huntington's disease. *Acta Neuropathologica Communications*, **2021**, 9, 70	7.3	1
126	Enforced dimerization between XBP1s and ATF6f enhances the protective effects of the UPR in models of neurodegeneration. *Molecular Therapy*, **2021**, 29, 1862-1882	11.7	8
125	Parkinson mice show functional and molecular changes in the gut long before motoric disease onset. *Molecular Neurodegeneration*, **2021**, 16, 34	19	5
124	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	16.4	3
123	Alpha-synuclein research: defining strategic moves in the battle against Parkinson's disease. *Npj Parkinson's Disease*, **2021**, 7, 65	9.7	12
122	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	5.6	3
121	Monitoring alpha-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	6	8
120	Alpha-Synuclein oligomerization and aggregation: All models are useful but only if we know what they model: This is the reply to a comment "Alpha-synuclein oligomerization and aggregation: A model will always be a model" on the original article "Monitoring alpha-synuclein oligomerization and aggregation using bimolecular fluorescence complementation assays: What you see is not	6	4
119	Site-Specific Phosphorylation of Huntingtin Exon 1 Recombinant Proteins Enabled by the Discovery of Novel Kinases. *ChemBioChem*, **2021**, 22, 217-231	3.8	8
118	Reverse engineering Lewy bodies: how far have we come and how far can we go?. *Nature Reviews Neuroscience*, **2021**, 22, 111-131	13.5	47
117	Hypoxia Conditioning as a Promising Therapeutic Target in Parkinson's Disease?. *Movement Disorders*, **2021**, 36, 857-861	7	8
116	Lewy body-associated proteins: victims, instigators, or innocent bystanders? The case of AIMP2 and alpha-synuclein. *Neurobiology of Disease*, **2021**, 156, 105417	7.5	0
115	Pharmacological characterization of mutant huntingtin aggregate-directed PET imaging tracer candidates. *Scientific Reports*, **2021**, 11, 17977	4.9	4

114	Rethinking protein aggregation and drug discovery in neurodegenerative diseases: Why we need to embrace complexity?. *Current Opinion in Chemical Biology*, **2021**, 64, 67-75	9.7	14
113	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	6.5	2
112	Fatal attraction - The role of hypoxia when alpha-synuclein gets intimate with mitochondria. *Neurobiology of Aging*, **2021**, 107, 128-141	5.6	1
111	Extent of N-terminus exposure of monomeric alpha-synuclein determines its aggregation propensity. *Nature Communications*, **2020**, 11, 2820	17.4	39
110	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	5.4	15
109	Education and research are essential for lasting peace in Yemen. *Lancet, The*, **2020**, 395, 1114	40	0
108	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	7.5	36
107	Pronounced -Synuclein Pathology in a Seeding-Based Mouse Model Is Not Sufficient to Induce Mitochondrial Respiration Deficits in the Striatum and Amygdala. *ENeuro*, **2020**, 7,	3.9	6
106	TBK1 phosphorylates mutant Huntingtin and suppresses its aggregation and toxicity in Huntington's disease models. *EMBO Journal*, **2020**, 39, e104671	13	15
105	What about faculty?. *ELife*, **2020**, 9,	8.9	14
104	The process of Lewy body formation, rather than simply -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	11.5	193
103	Ultrasensitive quantitative measurement of huntingtin phosphorylation at residue S13. *Biochemical and Biophysical Research Communications*, **2020**, 521, 549-554	3.4	8
102	A simple, versatile and robust centrifugation-based filtration protocol for the isolation and quantification of -synuclein monomers, oligomers and fibrils: Towards improving experimental reproducibility in -synuclein research. *Journal of Neurochemistry*, **2020**, 153, 103-119	6	20
101	Site-Specific Hyperphosphorylation Inhibits, Rather than Promotes, Tau Fibrillization, Seeding Capacity, and Its Microtubule Binding. *Angewandte Chemie*, **2020**, 132, 4088-4096	3.6	8
100	Site-Specific Hyperphosphorylation Inhibits, Rather than Promotes, Tau Fibrillization, Seeding Capacity, and Its Microtubule Binding. *Angewandte Chemie - International Edition*, **2020**, 59, 4059-4067	16.4	29
99	How specific are the conformation-specific -synuclein antibodies? Characterization and validation of 16 -synuclein conformation-specific antibodies using well-characterized preparations of -synuclein monomers, fibrils and oligomers with distinct structures and morphology. *Neurobiology of Disease*, **2020**, 146, 105086	7.5	31
98	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	11.5	27
97	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	5.1	14

96	The Role of Post-translational Modifications on the Energy Landscape of Huntingtin N-Terminus. *Frontiers in Molecular Biosciences*, **2019**, 6, 95	5.6	10
95	Antibody-based methods for the measurement of -synuclein concentration in human cerebrospinal fluid - method comparison and round robin study. *Journal of Neurochemistry*, **2019**, 149, 126-138	6	26
94	Chronic corticosterone aggravates behavioral and neuronal symptomatology in a mouse model of alpha-synuclein pathology. *Neurobiology of Aging*, **2019**, 83, 11-20	5.6	11
93	Single Posttranslational Modifications in the Central Repeat Domains of Tau4 Impact its Aggregation and Tubulin Binding. *Angewandte Chemie*, **2019**, 131, 1630-1634	3.6	10
92	-Synuclein O-GlcNAcylation alters aggregation and toxicity, revealing certain residues as potential inhibitors of Parkinson's disease. *Proceedings of the National Academy of Sciences of the United States of America*, **2019**, 116, 1511-1519	11.5	91
91	Single Posttranslational Modifications in the Central Repeat Domains of Tau4 Impact its Aggregation and Tubulin Binding. *Angewandte Chemie - International Edition*, **2019**, 58, 1616-1620	16.4	26
90	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	16.4	54
89	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
88	Resolving molecule-specific information in dynamic lipid membrane processes with multi-resonant infrared metasurfaces. *Nature Communications*, **2018**, 9, 2160	17.4	103
87	O2-09-04: HARMONIZATION OF IMMUNOCHEMICAL METHODS FOR MEASUREMENT OF -SYNUCLEIN IN HUMAN CEREBROSPINAL FLUID: A ROUND ROBIN STUDY APPROACH **2018**, 14, P642-P642		
86	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	5.4	36
85	Real-Time In Situ Secondary Structure Analysis of Protein Monolayer with Mid-Infrared Plasmonic Nanoantennas. *ACS Sensors*, **2018**, 3, 1109-1117	9.2	30
84	Generation of Native, Untagged Huntingtin Exon1 Monomer and Fibrils Using a SUMO Fusion Strategy. *Journal of Visualized Experiments*, **2018**,	1.6	7
83	Identification and nanomechanical characterization of the fundamental single-strand protofilaments of amyloid -synuclein fibrils. *Proceedings of the National Academy of Sciences of the United States of America*, **2018**, 115, 7230-7235	11.5	51
82	Glycation potentiates -synuclein-associated neurodegeneration in synucleinopathies. *Brain*, **2017**, 140, 1399-1419	11.2	96
81	Frontispiece: Mutant Exon1 Huntingtin Aggregation is Regulated by T3 Phosphorylation-Induced Structural Changes and Crosstalk between T3 Phosphorylation and Acetylation at K6. *Angewandte Chemie - International Edition*, **2017**, 56,	16.4	3
80	Frontispiz: Mutant Exon1 Huntingtin Aggregation is Regulated by T3 Phosphorylation-Induced Structural Changes and Crosstalk between T3 Phosphorylation and Acetylation at K6. *Angewandte Chemie*, **2017**, 129,	3.6	1
79	Mutant Exon1 Huntingtin Aggregation is Regulated by T3 Phosphorylation-Induced Structural Changes and Crosstalk between T3 Phosphorylation and Acetylation at K6. *Angewandte Chemie*, **2017**, 129, 5286-5291	3.6	2

78	Mutant Exon1 Huntingtin Aggregation is Regulated by T3 Phosphorylation-Induced Structural Changes and Crosstalk between T3 Phosphorylation and Acetylation at K6. *Angewandte Chemie - International Edition*, **2017**, 56, 5202-5207	16.4	38
77	Membrane scission driven by the PROPPIN Atg18. *EMBO Journal*, **2017**, 36, 3274-3291	13	39
76	Amyloid single-cell cytotoxicity assays by nanomotion detection. *Cell Death Discovery*, **2017**, 3, 17053	6.9	13
75	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	16.4	54
74	A user's guide for □synuclein biomarker studies in biological fluids: Perianalytical considerations. *Movement Disorders*, **2017**, 32, 1117-1130	7	35
73	Phosphorylation of huntingtin at residue T3 is decreased in Huntington'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	11.5	33
72	Discovery and characterization of stable and toxic Tau/phospholipid oligomeric complexes. *Nature Communications*, **2017**, 8, 1678	17.4	77
71	Polyglutamine expansion affects huntingtin conformation in multiple Huntington's disease models. *Scientific Reports*, **2017**, 7, 5070	4.9	22
70	Activation of the STING-Dependent Type I Interferon Response Reduces Microglial Reactivity and Neuroinflammation. *Neuron*, **2017**, 96, 1290-1302.e6	13.9	65
69	Nanoplasmonic mid-infrared biosensor for protein secondary structure detection. *Light: Science and Applications*, **2017**, 6, e17029	16.7	66
68	Semisynthesis and Enzymatic Preparation of Post-translationally Modified □Synuclein. *Methods in Molecular Biology*, **2016**, 1345, 3-20	1.4	12
67	A New Caged-Glutamine Derivative as a Tool To Control the Assembly of Glutamine-Containing Amyloidogenic Peptides. *ChemBioChem*, **2016**, 17, 2353-2360	3.8	6
66	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, 12074-86	5.4	25
65	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, 2428-37	4.9	39
64	Induction of de novo □synuclein fibrillization in a neuronal model for Parkinson's disease. *Proceedings of the National Academy of Sciences of the United States of America*, **2016**, 113, E912-21	11.5	69
63	Health hazards of methylammonium lead iodide based perovskites: cytotoxicity studies. *Toxicology Research*, **2016**, 5, 407-419	2.6	82
62	Microtubule-Binding R3 Fragment from Tau Self-Assembles into Giant Multistranded Amyloid Ribbons. *Angewandte Chemie - International Edition*, **2016**, 55, 618-22	16.4	33
61	Detection of huntingtin exon 1 phosphorylation by Phos-Tag SDS-PAGE: Predominant phosphorylation on threonine 3 and regulation by IKK□*Biochemical and Biophysical Research Communications*, **2015**, 463, 1317-22	3.4	15

60	Elucidating the Role of Site-Specific Nitration of α-Synuclein in the Pathogenesis of Parkinson's Disease via Protein Semisynthesis and Mutagenesis. *Journal of the American Chemical Society*, **2015**, 137, 5041-52	16.4	81
59	Parkinson disease mutant E46K enhances α-synuclein phosphorylation in mammalian cell lines, in yeast, and in vivo. *Journal of Biological Chemistry*, **2015**, 290, 9412-27	5.4	41
58	Structural differences of amyloid-β fibrils revealed by antibodies from phage display. *BMC Biotechnology*, **2015**, 15, 57	3.5	8
57	Influence of the β-Sheet Content on the Mechanical Properties of Aggregates during Amyloid Fibrillization. *Angewandte Chemie*, **2015**, 127, 2492-2496	3.6	15
56	Photobiomodulation Suppresses Alpha-Synuclein-Induced Toxicity in an AAV-Based Rat Genetic Model of Parkinson's Disease. *PLoS ONE*, **2015**, 10, e0140880	3.7	35
55	c-Abl phosphorylates α-synuclein and regulates its degradation: implication for α-synuclein clearance and contribution to the pathogenesis of Parkinson's disease. *Human Molecular Genetics*, **2014**, 23, 2858-79	5.6	126
54	The novel Parkinson's disease linked mutation G51D attenuates in vitro aggregation and membrane binding of α-synuclein, and enhances its secretion and nuclear localization in cells. *Human Molecular Genetics*, **2014**, 23, 4491-509	5.6	153
53	The H50Q mutation enhances α-synuclein aggregation, secretion, and toxicity. *Journal of Biological Chemistry*, **2014**, 289, 21856-76	5.4	126
52	One-pot semisynthesis of exon 1 of the Huntingtin protein: new tools for elucidating the role of posttranslational modifications in the pathogenesis of Huntington's disease. *Angewandte Chemie - International Edition*, **2014**, 53, 1928-33	16.4	39
51	One-Pot Semisynthesis of Exon 1 of the Huntingtin Protein: New Tools for Elucidating the Role of Posttranslational Modifications in the Pathogenesis of Huntington's Disease. *Angewandte Chemie*, **2014**, 126, 1959-1964	3.6	5
50	One-pot total chemical synthesis of human α-synuclein. *Chemical Communications*, **2013**, 49, 9254-6	5.8	31
49	Synthetic polyubiquitinated α-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, 17726-31	11.5	108
48	Alpha-synuclein post-translational modifications as potential biomarkers for Parkinson disease and other synucleinopathies. *Molecular and Cellular Proteomics*, **2013**, 12, 3543-58	7.6	106
47	Oxidative and nitrative alpha-synuclein modifications and proteostatic stress: implications for disease mechanisms and interventions in synucleinopathies. *Journal of Neurochemistry*, **2013**, 125, 491-511	6	102
46	The many faces of α-synuclein: from structure and toxicity to therapeutic target. *Nature Reviews Neuroscience*, **2013**, 14, 38-48	13.5	975
45	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, 562-7	16.4	9
44	Polo-like kinase 2 regulates selective autophagic α-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	11.5	125
43	Elucidating the role of C-terminal post-translational modifications using protein semisynthesis strategies: α-synuclein phosphorylation at tyrosine 125. *Journal of the American Chemical Society*, **2012**, 134, 5196-210	16.4	75

42	Chemical strategies for controlling protein folding and elucidating the molecular mechanisms of amyloid formation and toxicity. *Journal of Molecular Biology*, **2012**, 421, 204-36	6.5	24
41	The size of the proteasomal substrate determines whether its degradation will be mediated by mono- or polyubiquitylation. *Molecular Cell*, **2012**, 48, 87-97	17.6	119
40	Synuclein in central nervous system and from erythrocytes, mammalian cells, and Escherichia coli exists predominantly as disordered monomer. *Journal of Biological Chemistry*, **2012**, 287, 15345-64	5.4	375
39	Mimicking phosphorylation at serine 87 inhibits the aggregation of human synuclein and protects against its toxicity in a rat model of Parkinson's disease. *Journal of Neuroscience*, **2012**, 32, 1536-44	6.6	64
38	Characterization of semisynthetic and naturally N acetylated synuclein in vitro and in intact cells: implications for aggregation and cellular properties of synuclein. *Journal of Biological Chemistry*, **2012**, 287, 28243-62	5.4	121
37	Towards Elucidation of the Role of Ubiquitination in the Pathogenesis of Parkinson Disease with Semisynthetic Ubiquitinated Synuclein. *Angewandte Chemie*, **2011**, 123, 425-429	3.6	21
36	Towards elucidation of the role of ubiquitination in the pathogenesis of Parkinson's disease with semisynthetic ubiquitinated synuclein. *Angewandte Chemie - International Edition*, **2011**, 50, 405-9	16.4	97
35	Phosphorylation of Synuclein at Y125 and S129 alters its metal binding properties: implications for understanding the role of Synuclein in the pathogenesis of Parkinson's Disease and related disorders. *ACS Chemical Neuroscience*, **2011**, 2, 667-75	5.7	76
34	Amyloid-beta aggregates cause alterations of astrocytic metabolic phenotype: impact on neuronal viability. *Journal of Neuroscience*, **2010**, 30, 3326-38	6.6	207
33	Phosphorylation at S87 is enhanced in synucleinopathies, inhibits alpha-synuclein oligomerization, and influences synuclein-membrane interactions. *Journal of Neuroscience*, **2010**, 30, 3184-98	6.6	207
32	Role of post-translational modifications in modulating the structure, function and toxicity of alpha-synuclein: implications for Parkinson's disease pathogenesis and therapies. *Progress in Brain Research*, **2010**, 183, 115-45	2.9	239
31	Phosphorylation of synucleins by members of the Polo-like kinase family. *Journal of Biological Chemistry*, **2010**, 285, 2807-22	5.4	170
30	Amyloidogenic protein-membrane interactions: mechanistic insight from model systems. *Angewandte Chemie - International Edition*, **2010**, 49, 5628-54	16.4	456
29	Amyloids go genomic: insights regarding the sequence determinants of prion formation from genome-wide studies. *ChemBioChem*, **2009**, 10, 1951-4	3.8	5
28	Highly efficient and chemoselective peptide ubiquitylation. *Angewandte Chemie - International Edition*, **2009**, 48, 8090-4	16.4	227
27	Structural properties of pore-forming oligomers of alpha-synuclein. *Journal of the American Chemical Society*, **2009**, 131, 17482-9	16.4	162
26	Switch peptide via Staudinger reaction. *Organic Letters*, **2008**, 10, 5243-6	6.2	37
25	Phosphorylation at Ser-129 but not the phosphomimics S129E/D inhibits the fibrillation of alpha-synuclein. *Journal of Biological Chemistry*, **2008**, 283, 16895-905	5.4	240

24	The ratio of monomeric to aggregated forms of Abeta40 and Abeta42 is an important determinant of amyloid-beta aggregation, fibrillogenesis, and toxicity. *Journal of Biological Chemistry*, **2008**, 283, 28176-89	5.4	197
23	Switch-peptides: design and characterization of controllable super-amyloid-forming host-guest peptides as tools for identifying anti-amyloid agents. *ChemBioChem*, **2008**, 9, 2104-12	3.8	22
22	Inhibition of alpha-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	3.7	88
21	The impact of the E46K mutation on the properties of alpha-synuclein in its monomeric and oligomeric states. *Biochemistry*, **2007**, 46, 7107-18	3.2	178
20	Switch-peptides as folding precursors in self-assembling peptides and amyloid fibrillogenesis. *Biopolymers*, **2007**, 88, 239-52	2.2	37
19	Disruption of amyloid-derived peptide assemblies through the controlled induction of a beta-sheet to alpha-helix transformation: application of the switch concept. *Angewandte Chemie - International Edition*, **2007**, 46, 2681-4	16.4	60
18	Are amyloid diseases caused by protein aggregates that mimic bacterial pore-forming toxins?. *Quarterly Reviews of Biophysics*, **2006**, 39, 167-201	7	325
17	Switch-Peptides: From Conformational Studies to Alzheimer's Disease. *Chimia*, **2006**, 60, 199-202	1.3	16
16	A century-old debate on protein aggregation and neurodegeneration enters the clinic. *Nature*, **2006**, 443, 774-9	50.4	554
15	Molecular electron microscopy approaches to elucidating the mechanisms of protein fibrillogenesis. *Methods in Molecular Biology*, **2005**, 299, 81-101	1.4	10
14	In vitro preparation of prefibrillar intermediates of amyloid-beta and alpha-synuclein. *Methods in Molecular Biology*, **2005**, 299, 19-33	1.4	19
13	Membrane permeabilization: a common mechanism in protein-misfolding diseases. *Science of Aging Knowledge Environment: SAGE KE*, **2005**, 2005, pe28		29
12	Abeta protofibrils possess a stable core structure resistant to hydrogen exchange. *Biochemistry*, **2003**, 42, 14092-8	3.2	116
11	Mixtures of wild-type and a pathogenic (E22G) form of Abeta40 in vitro accumulate protofibrils, including amyloid pores. *Journal of Molecular Biology*, **2003**, 332, 795-808	6.5	213
10	Neurodegenerative disease: amyloid pores from pathogenic mutations. *Nature*, **2002**, 418, 291	50.4	1046
9	New class of inhibitors of amyloid-beta fibril formation. Implications for the mechanism of pathogenesis in Alzheimer's disease. *Journal of Biological Chemistry*, **2002**, 277, 42881-90	5.4	115
8	Alpha-synuclein, especially the Parkinson's disease-associated mutants, forms pore-like annular and tubular protofibrils. *Journal of Molecular Biology*, **2002**, 322, 1089-102	6.5	687
7	Protofilaments, filaments, ribbons, and fibrils from peptidomimetic self-assembly: implications for amyloid fibril formation and materials science. *Journal of the American Chemical Society*, **2000**, 122, 5262-77	16.4	267

List of Publications

6	Nuclear and cytoplasmic huntingtin inclusions exhibit distinct biochemical composition, interactome and ultrastructural properties	5
5	N-terminal phosphorylation of Huntingtin: A molecular switch for regulating Htt aggregation, helical conformation, internalization and nuclear targeting	1
4	The making of a Lewy body: the role of alpha-synuclein post-fibrillization modifications in regulating the formation and the maturation of pathological inclusions.	19
3	The process of Lewy body formation, rather than simply alpha-synuclein fibrillization, is the major driver of neurodegeneration in synucleinopathies	3
2	The Nt17 domain and its helical conformation regulate the aggregation, cellular properties and neurotoxicity of mutant huntingtin exon 1	3
1	The structural basis of huntingtin (Htt) fibril polymorphism, revealed by cryo-EM of exon 1 Htt fibrils	1