Fabrizio Chiti

List of Publications by Year
in descending order

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80	14,793	94269	62479
papers	citations	37 h-index	80 g-index

80	80	80	13930
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Sphingosine 1â€phosphate attenuates neuronal dysfunction induced by amyloidâ€Î² oligomers through endocytic internalization of <scp>NMDA</scp> receptors. FEBS Journal, 2023, 290, 112-133.	2.2	4
2	Squalamine and trodusquemine: two natural products for neurodegenerative diseases, from physical chemistry to the clinic. Natural Product Reports, 2022, 39, 742-753.	5.2	27
3	Mechanosensitivity of N-methyl-D-aspartate receptors (NMDAR) is the key through which amyloid beta oligomers activate them. Neural Regeneration Research, 2022, 17, 1263.	1.6	1
4	Small molecule protein binding to correct cellular folding or stabilize the native state against misfolding and aggregation. Current Opinion in Structural Biology, 2022, 72, 267-278.	2.6	21
5	Editorial overview: Folding and binding. Current Opinion in Structural Biology, 2022, , 102359.	2.6	1
6	A Brain-Permeable Aminosterol Regulates Cell Membranes to Mitigate the Toxicity of Diverse Pore-Forming Agents. ACS Chemical Neuroscience, 2022, 13, 1219-1231.	1.7	7
7	Conversion of the Native N-Terminal Domain of TDP-43 into a Monomeric Alternative Fold with Lower Aggregation Propensity. Molecules, 2022, 27, 4309.	1.7	3
8	Full-length TDP-43 and its C-terminal domain form filaments inÂvitro having non-amyloid properties. Amyloid: the International Journal of Experimental and Clinical Investigation: the Official Journal of the International Society of Amyloidosis, 2021, 28, 56-65.	1.4	6
9	AÎ² Oligomers Dysregulate Calcium Homeostasis by Mechanosensitive Activation of AMPA and NMDA Receptors. ACS Chemical Neuroscience, 2021, 12, 766-781.	1.7	35
10	The release of toxic oligomers from Î±-synuclein fibrils induces dysfunction in neuronal cells. Nature Communications, 2021, 12, 1814.	5.8	123
11	Gold Nanostars Bioconjugation for Selective Targeting and SERS Detection of Biofluids. Nanomaterials, 2021, 11, 665.	1.9	11
12	Urea titration of a lipase from Pseudomonas sp. reveals four different conformational states, with a stable partially folded state explaining its high aggregation propensity. International Journal of Biological Macromolecules, 2021, 174, 32-41.	3.6	5
13	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
14	Distinct responses of human peripheral blood cells to different misfolded protein oligomers. Immunology, 2021, 164, 358-371.	2.0	7
15	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
16	Quantitative Measurement of the Affinity of Toxic and Nontoxic Misfolded Protein Oligomers for Lipid Bilayers and of its Modulation by Lipid Composition and Trodusquemine. ACS Chemical Neuroscience, 2021, 12, 3189-3202.	1.7	13
17	Toxic oligomers of the amyloidogenic HypF-N protein form pores in mitochondrial membranes. Scientific Reports, 2020, 10, 17733.	1.6	10
18	Soluble Prion Peptide 107â€“120 Protects Neuroblastoma SH-SY5Y Cells against Oligomers Associated with Alzheimerâ€™s Disease. International Journal of Molecular Sciences, 2020, 21, 7273.	1.8	2

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19	Nanoscopic insights into the surface conformation of neurotoxic amyloid Î² oligomers. RSC Advances, 2020, 10, 21907-21913.	1.7	19
20	Making biological membrane resistant to the toxicity of misfolded protein oligomers: a lesson from trodusquemine. Nanoscale, 2020, 12, 22596-22614.	2.8	16
21	Insight into the Folding and Dimerization Mechanisms of the N-Terminal Domain from Human TDP-43. International Journal of Molecular Sciences, 2020, 21, 6259.	1.8	13
22	Trodusquemine displaces protein misfolded oligomers from cell membranes and abrogates their cytotoxicity through a generic mechanism. Communications Biology, 2020, 3, 435.	2.0	44
23	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
24	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
25	Differential Interactome and Innate Immune Response Activation of Two Structurally Distinct Misfolded Protein Oligomers. ACS Chemical Neuroscience, 2019, 10, 3464-3478.	1.7	7
26	Probing conformational changes of monomeric transthyretin with second derivative fluorescence. Scientific Reports, 2019, 9, 10988.	1.6	14
27	Capturing AÎ²42 aggregation in the cell. Journal of Biological Chemistry, 2019, 294, 1488-1489.	1.6	1
28	The Toxicity of Misfolded Protein Oligomers Is Independent of Their Secondary Structure. ACS Chemical Biology, 2019, 14, 1593-1600.	1.6	34
29	Probing the Origin of the Toxicity of Oligomeric Aggregates of Î±-Synuclein with Antibodies. ACS Chemical Biology, 2019, 14, 1352-1362.	1.6	33
30	Trodusquemine enhances AÎ²42 aggregation but suppresses its toxicity by displacing oligomers from cell membranes. Nature Communications, 2019, 10, 225.	5.8	111
31	Toxic HypF-N Oligomers Selectively Bind the Plasma Membrane to Impair Cell Adhesion Capability. Biophysical Journal, 2018, 114, 1357-1367.	0.2	8
32	Insight into the aggregation of lipase from Pseudomonas sp. using mutagenesis: protection of aggregation prone region by adoption of Î±-helix structure. Protein Engineering, Design and Selection, 2018, 31, 419-426.	1.0	7
33	Backbone NMR assignments of HypF-N under conditions generating toxic and non-toxic oligomers. Biomolecular NMR Assignments, 2018, 12, 273-277.	0.4	1
34	Stability of an aggregation-prone partially folded state of human profilin-1 correlates with aggregation propensity. Journal of Biological Chemistry, 2018, 293, 10303-10313.	1.6	10
35	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
36	Structural differences between toxic and nontoxic HypF-N oligomers. Chemical Communications, 2018, 54, 8637-8640.	2.2	25

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37	Nanoscale Discrimination between Toxic and Nontoxic Protein Misfolded Oligomers with Tipâ€Enhanced Raman Spectroscopy. Small, 2018, 14, e1800890.	5.2	35
38	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
39	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
40	Very rapid amyloid fibril formation by a bacterial lipase in the absence of a detectable lag phase. Biochimica Et Biophysica Acta - Proteins and Proteomics, 2017, 1865, 652-663.	1.1	16
41	Soluble Oligomers Require a Ganglioside to Trigger Neuronal Calcium Overload. Journal of Alzheimer's Disease, 2017, 60, 923-938.	1.2	41
42	Structural basis of membrane disruption and cellular toxicity by Î±-synuclein oligomers. Science, 2017, 358, 1440-1443.	6.0	492
43	Chaperones as Suppressors of Protein Misfolded Oligomer Toxicity. Frontiers in Molecular Neuroscience, 2017, 10, 98.	1.4	44
44	FRET studies of various conformational states adopted by transthyretin. Cellular and Molecular Life Sciences, 2017, 74, 3577-3598.	2.4	7
45	Biophysical analysis of three novel profilin-1 variants associated with amyotrophic lateral sclerosis indicates a correlation between their aggregation propensity and the structural features of their globular state. Biological Chemistry, 2016, 397, 927-937.	1.2	17
46	Quantification of the Relative Contributions of Loss-of-function and Gain-of-function Mechanisms in TAR DNA-binding Protein 43 (TDP-43) Proteinopathies. Journal of Biological Chemistry, 2016, 291, 19437-19448.	1.6	75
47	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
48	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
49	Interaction of toxic and non-toxic HypF-N oligomers with lipid bilayers investigated at high resolution with atomic force microscopy. Oncotarget, 2016, 7, 44991-45004.	0.8	23
50	Destabilisation, aggregation, toxicity and cytosolic mislocalisation of nucleophosmin regions associated with acute myeloid leukemia. Oncotarget, 2016, 7, 59129-59143.	0.8	41
51	Nucleophosmin contains amyloidogenic regions that are able to form toxic aggregates under physiological conditions. FASEB Journal, 2015, 29, 3689-3701.	0.2	53
52	The Folding process of Human Profilin-1, a novel protein associated with familial amyotrophic lateral sclerosis. Scientific Reports, 2015, 5, 12332.	1.6	14
53	Mutations of Profilin-1 Associated with Amyotrophic Lateral Sclerosis Promote Aggregation Due to Structural Changes of Its Native State. ACS Chemical Biology, 2015, 10, 2553-2563.	1.6	23
54	SERS Detection of Amyloid Oligomers on Metallorganic-Decorated Plasmonic Beads. ACS Applied Materials & Interfaces, 2015, 7, 9420-9428.	4.0	89

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55	TDP-43 Inclusion Bodies Formed in Bacteria Are Structurally Amorphous, Non-Amyloid and Inherently Toxic to Neuroblastoma Cells. PLoS ONE, 2014, 9, e86720.	1.1	68
56	Toxicity of Protein Oligomers Is Rationalized by a Function Combining Size and Surface Hydrophobicity. ACS Chemical Biology, 2014, 9, 2309-2317.	1.6	166
57	A Complex Equilibrium among Partially Unfolded Conformations in Monomeric Transthyretin. Biochemistry, 2014, 53, 4381-4392.	1.2	12
58	Amyloid-Î² oligomer synaptotoxicity is mimicked by oligomers of the model protein HypF-N. Neurobiology of Aging, 2013, 34, 2100-2109.	1.5	31
59	Transthyretin suppresses the toxicity of oligomers formed by misfolded proteins in vitro. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2013, 1832, 2302-2314.	1.8	67
60	Extracellular chaperones prevent Aî²42-induced toxicity in rat brains. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2013, 1832, 1217-1226.	1.8	51
61	Membrane lipid composition and its physicochemical properties define cell vulnerability to aberrant protein oligomers. Journal of Cell Science, 2012, 125, 2416-27.	1.2	75
62	Glycosaminoglycans (GAGs) Suppress the Toxicity of HypF-N Prefibrillar Aggregates. Journal of Molecular Biology, 2012, 421, 616-630.	2.0	17
63	Molecular mechanisms used by chaperones to reduce the toxicity of aberrant protein oligomers. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 12479-12484.	3.3	137
64	Protein Misfolded Oligomers: Experimental Approaches, Mechanism of Formation, and Structure-Toxicity Relationships. Chemistry and Biology, 2012, 19, 315-327.	6.2	239
65	A comparison of the biochemical modifications caused by toxic and nonâ€toxic protein oligomers in cells. Journal of Cellular and Molecular Medicine, 2011, 15, 2106-2116.	1.6	53
66	Experimental free energy surfaces reveal the mechanisms of maintenance of protein solubility. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 21057-21062.	3.3	65
67	A causative link between the structure of aberrant protein oligomers and their toxicity. Nature Chemical Biology, 2010, 6, 140-147.	3.9	499
68	Conformational properties of the aggregation precursor state of HypF-N. Journal of Molecular Biology, 2008, 379, 554-567.	2.0	45
69	Amyloid Fibril Formation and Disaggregation of Fragment 1-29 of Apomyoglobin: Insights into the Effect of pH on Protein Fibrillogenesis. Journal of Molecular Biology, 2007, 367, 1237-1245.	2.0	62
70	Stabilization of a Native Protein Mediated by Ligand Binding Inhibits Amyloid Formation Independently of the Aggregation Pathway. Journal of Medicinal Chemistry, 2006, 49, 6057-6064.	2.9	33
71	Prefibrillar Amyloid Aggregates Could Be Generic Toxins in Higher Organisms. Journal of Neuroscience, 2006, 26, 8160-8167.	1.7	222
72	Protein Misfolding, Functional Amyloid, and Human Disease. Annual Review of Biochemistry, 2006, 75, 333-366.	5.0	5,737

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73	Amyloid Formation from HypF-N under Conditions in which the Protein is Initially in its Native State. Journal of Molecular Biology, 2005, 347, 323-335.	2.0	74
74	Prefibrillar Amyloid Protein Aggregates Share Common Features of Cytotoxicity. Journal of Biological Chemistry, 2004, 279, 31374-31382.	1.6	346
75	Monitoring the Process of HypF Fibrillization and Liposome Permeabilization by Protofibrils. Journal of Molecular Biology, 2004, 338, 943-957.	2.0	101
76	Inherent toxicity of aggregates implies a common mechanism for protein misfolding diseases. Nature, 2002, 416, 507-511.	13.7	2,322
77	Reduction of the amyloidogenicity of a protein by specific binding of ligands to the native conformation. Protein Science, 2001, 10, 879-886.	3.1	62
78	A Partially Structured Species of Î²2-Microglobulin Is Significantly Populated under Physiological Conditions and Involved in Fibrillogenesis. Journal of Biological Chemistry, 2001, 276, 46714-46721.	1.6	137
79	Solution conditions can promote formation of either amyloid protofilaments or mature fibrils from the HypF Nâ€terminal domain. Protein Science, 2001, 10, 2541-2547.	3.1	47
80	Stabilisation of Î±-helices by site-directed mutagenesis reveals the importance of secondary structure in the transition state for acylphosphatase folding. Journal of Molecular Biology, 2000, 300, 633-647.	2.0	53