Benedetta Mannini

List of Publications by Year
in descending order

43	2,308	304368 22	276539 41
papers	citations	h-index	g-index

47	47	47	3301
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	The Pathological G51D Mutation in Alpha-Synuclein Oligomers Confers Distinct Structural Attributes and Cellular Toxicity. Molecules, 2022, 27, 1293.	1.7	6
2	AÎ² Oligomers Dysregulate Calcium Homeostasis by Mechanosensitive Activation of AMPA and NMDA Receptors. ACS Chemical Neuroscience, 2021, 12, 766-781.	1.7	35
3	Therapeutics Against Protein Misfolded Oligomers in Neurodegenerative Diseases. Biophysical Journal, 2021, 120, 286a.	0.2	0
4	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
5	Distinct responses of human peripheral blood cells to different misfolded protein oligomers. Immunology, 2021, 164, 358-371.	2.0	7
6	Two human metabolites rescue a C. elegans model of Alzheimerâ€™s disease via a cytosolic unfolded protein response. Communications Biology, 2021, 4, 843.	2.0	6
7	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
8	A dopamine metabolite stabilizes neurotoxic amyloid-Î² oligomers. Communications Biology, 2021, 4, 19.	2.0	25
9	Surface-Catalyzed Secondary Nucleation Dominates the Generation of Toxic IAPP Aggregates. Frontiers in Molecular Biosciences, 2021, 8, 757425.	1.6	24
10	Proteome-wide observation of the phenomenon of life on the edge of solubility. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 1015-1020.	3.3	115
11	Therapeutic Strategies to Reduce the Toxicity of Misfolded Protein Oligomers. International Journal of Molecular Sciences, 2020, 21, 8651.	1.8	23
12	A rationally designed bicyclic peptide remodels AÎ²42 aggregation in vitro and reduces its toxicity in a worm model of Alzheimerâ€™s disease. Scientific Reports, 2020, 10, 15280.	1.6	15
13	Trodusquemine displaces protein misfolded oligomers from cell membranes and abrogates their cytotoxicity through a generic mechanism. Communications Biology, 2020, 3, 435.	2.0	44
14	Small-molecule sequestration of amyloid-Î² as a drug discovery strategy for Alzheimerâ€™s disease. Science Advances, 2020, 6, .	4.7	95
15	Single molecule secondary structure determination of proteins through infrared absorption nanospectroscopy. Nature Communications, 2020, 11, 2945.	5.8	92
16	Rational design of a conformation-specific antibody for the quantification of AÎ² oligomers. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 13509-13518.	3.3	61
17	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
18	Differential Interactome and Innate Immune Response Activation of Two Structurally Distinct Misfolded Protein Oligomers. ACS Chemical Neuroscience, 2019, 10, 3464-3478.	1.7	7

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19	Trodusquemine enhances AÎ²42 aggregation but suppresses its toxicity by displacing oligomers from cell membranes. Nature Communications, 2019, 10, 225.	5.8	111
20	Systematic Development of Small Molecules to Inhibit Specific Microscopic Steps of Amyloid-Beta42 Aggregation in Alzheimer's Disease. Biophysical Journal, 2018, 114, 225a.	0.2	2
21	Modulating Amyloid-Beta Aggregation to Reduce the Toxicity of its Oligomeric Aggregates. Biophysical Journal, 2018, 114, 430a.	0.2	2
22	Toxic HypF-N Oligomers Selectively Bind the Plasma Membrane to Impair Cell Adhesion Capability. Biophysical Journal, 2018, 114, 1357-1367.	0.2	8
23	O2â€02â€02: TARGETING AMYLOID FORMATION USING RATIONALLY DESIGNED ANTIBODIES. Alzheimer's and Dementia, 2018, 14, P611.	0.4	0
24	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
25	Stabilization and Characterization of Cytotoxic AÎ²₄₀ Oligomers Isolated from an Aggregation Reaction in the Presence of Zinc Ions. ACS Chemical Neuroscience, 2018, 9, 2959-2971.	1.7	42
26	Attenuating the Toxicity of Amyloid-Beta Aggregation with Specific Species. Biophysical Journal, 2017, 112, 494a.	0.2	1
27	Systematic development of small molecules to inhibit specific microscopic steps of AÎ²42 aggregation in Alzheimerâ€™s disease. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, E200-E208.	3.3	180
28	Delivery of Native Proteins into C. elegans Using a Transduction Protocol Based on Lipid Vesicles. Scientific Reports, 2017, 7, 15045.	1.6	16
29	Chaperones as Suppressors of Protein Misfolded Oligomer Toxicity. Frontiers in Molecular Neuroscience, 2017, 10, 98.	1.4	44
30	Bis(indolyl)phenylmethane derivatives are effective small molecules for inhibition of amyloid fibril formation by hen lysozyme. European Journal of Medicinal Chemistry, 2016, 124, 361-371.	2.6	19
31	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
32	SERS Detection of Amyloid Oligomers on Metallorganic-Decorated Plasmonic Beads. ACS Applied Materials & Interfaces, 2015, 7, 9420-9428.	4.0	89
33	Toxicity of Protein Oligomers Is Rationalized by a Function Combining Size and Surface Hydrophobicity. ACS Chemical Biology, 2014, 9, 2309-2317.	1.6	166
34	Amyloid-Î² oligomer synaptotoxicity is mimicked by oligomers of the model protein HypF-N. Neurobiology of Aging, 2013, 34, 2100-2109.	1.5	31
35	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
36	Glycosaminoglycans (GAGs) Suppress the Toxicity of HypF-N Prefibrillar Aggregates. Journal of Molecular Biology, 2012, 421, 616-630.	2.0	17

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37	Salt Anions Promote the Conversion of HypF-N into Amyloid-Like Oligomers and Modulate the Structure of the Oligomers and the Monomeric Precursor State. Journal of Molecular Biology, 2012, 424, 132-149.	2.0	24
38	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
39	Large Proteins Have a Great Tendency to Aggregate but a Low Propensity to Form Amyloid Fibrils. PLoS ONE, 2011, 6, e16075.	1.1	51
40	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
41	The induction of Â-helical structure in partially unfolded HypF-N does not affect its aggregation propensity. Protein Engineering, Design and Selection, 2011, 24, 553-563.	1.0	9
42	A causative link between the structure of aberrant protein oligomers and their toxicity. Nature Chemical Biology, 2010, 6, 140-147.	3.9	499
43	Low-Level Expression of a Folding-Incompetent Protein in Escherichia coli: Search for the Molecular Determinants of Protein Aggregation In Vivo. Journal of Molecular Biology, 2010, 398, 600-613.	2.0	21