Nunilo Cremades

List of Publications by Year
in descending order

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49 papers	5,132 citations	109264 35 h-index	189801 50 g-index
55 all docs	55 docs citations	55 times ranked	6014 citing authors

#	Article	IF	Citations
1	Direct Observation of the Interconversion of Normal and Toxic Forms of Î±-Synuclein. Cell, 2012, 149, 1048-1059.	13.5	755
2	Structural basis of membrane disruption and cellular toxicity by Î±-synuclein oligomers. Science, 2017, 358, 1440-1443.	6.0	492
3	Structural characterization of toxic oligomers that are kinetically trapped during Î±-synuclein fibril formation. Proceedings of the National Academy of Sciences of the United States of America, 2015, 112, E1994-2003.	3.3	384
4	Alpha-Synuclein Oligomers Interact with Metal Ions to Induce Oxidative Stress and Neuronal Death in Parkinson's Disease. Antioxidants and Redox Signaling, 2016, 24, 376-391.	2.5	266
5	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
6	Antiparasitic Drug Nitazoxanide Inhibits the Pyruvate Oxidoreductases of Helicobacter pylori , Selected Anaerobic Bacteria and Parasites, and Campylobacter jejuni. Antimicrobial Agents and Chemotherapy, 2007, 51, 868-876.	1.4	207
7	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.	3.3	181
8	Structure and Properties of a Complex of Î±-Synuclein and a Single-Domain Camelid Antibody. Journal of Molecular Biology, 2010, 402, 326-343.	2.0	164
9	Best Practices for Generating and Using Alpha-Synuclein Pre-Formed Fibrils to Model Parkinsonâ€™s Disease in Rodents. Journal of Parkinson's Disease, 2018, 8, 303-322.	1.5	151
10	Identification of pharmacological chaperones as potential therapeutic agents to treat phenylketonuria. Journal of Clinical Investigation, 2008, 118, 2858-2867.	3.9	145
11	Calcium is a key factor in Î±-synuclein induced neurotoxicity. Journal of Cell Science, 2016, 129, 1792-801.	1.2	136
12	Targeting the Intrinsically Disordered Structural Ensemble of Î±-Synuclein by Small Molecules as a Potential Therapeutic Strategy for Parkinsonâ€™s Disease. PLoS ONE, 2014, 9, e87133.	1.1	126
13	The release of toxic oligomers from Î±-synuclein fibrils induces dysfunction in neuronal cells. Nature Communications, 2021, 12, 1814.	5.8	123
14	Single-Molecule Imaging of Individual Amyloid Protein Aggregates in Human Biofluids. ACS Chemical Neuroscience, 2016, 7, 399-406.	1.7	99
15	Defining Î±-synuclein species responsible for Parkinsonâ€™s disease phenotypes in mice. Journal of Biological Chemistry, 2019, 294, 10392-10406.	1.6	96
16	Structural Characteristics of Î±-Synuclein Oligomers. International Review of Cell and Molecular Biology, 2017, 329, 79-143.	1.6	95
17	Single-molecule FRET studies on alpha-synuclein oligomerization of Parkinsonâ€™s disease genetically related mutants. Scientific Reports, 2015, 5, 16696.	1.6	92
18	Nanobodies Raised against Monomeric Î±-Synuclein Distinguish between Fibrils at Different Maturation Stages. Journal of Molecular Biology, 2013, 425, 2397-2411.	2.0	90

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19	On the Mechanism of Nonspecific Inhibitors of Protein Aggregation: Dissecting the Interactions of Î±-Synuclein with Congo Red and Lacmoid. Biochemistry, 2009, 48, 8322-8334.	1.2	88
20	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
21	Fast Flow Microfluidics and Single-Molecule Fluorescence for the Rapid Characterization of Î±-Synuclein Oligomers. Analytical Chemistry, 2015, 87, 8818-8826.	3.2	81
22	Population of Nonnative States of Lysozyme Variants Drives Amyloid Fibril Formation. Journal of the American Chemical Society, 2011, 133, 7737-7743.	6.6	72
23	Amyloid-Î² and Î±-Synuclein Decrease the Level of Metal-Catalyzed Reactive Oxygen Species by Radical Scavenging and Redox Silencing. Journal of the American Chemical Society, 2016, 138, 3966-3969.	6.6	69
24	Hsp70 Oligomerization Is Mediated by an Interaction between the Interdomain Linker and the Substrate-Binding Domain. PLoS ONE, 2013, 8, e67961.	1.1	66
25	Flavodoxin:Quinone Reductase (FqrB): a Redox Partner of Pyruvate:Ferredoxin Oxidoreductase That Reversibly Couples Pyruvate Oxidation to NADPH Production in Helicobacter pylori and Campylobacter jejuni. Journal of Bacteriology, 2007, 189, 4764-4773.	1.0	63
26	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.	2.1	62
27	Local Cooperativity in an Amyloidogenic State of Human Lysozyme Observed at Atomic Resolution. Journal of the American Chemical Society, 2010, 132, 15580-15588.	6.6	55
28	Discovery of Specific Flavodoxin Inhibitors as Potential Therapeutic Agents against Helicobacter pylori Infection. ACS Chemical Biology, 2009, 4, 928-938.	1.6	48
29	Inhibition of Î±-Synuclein Fibril Elongation by Hsp70 Is Governed by a Kinetic Binding Competition between Î±-Synuclein Species. Biochemistry, 2017, 56, 1177-1180.	1.2	47
30	Effects of oligomer toxicity, fibril toxicity and fibril spreading in synucleinopathies. Cellular and Molecular Life Sciences, 2022, 79, 174.	2.4	45
31	Towards a new therapeutic target: Helicobacter pylori flavodoxin. Biophysical Chemistry, 2005, 115, 267-276.	1.5	44
32	Insights in the (un)structural organization of Bacillus pasteurii UreG, an intrinsically disordered GTPase enzyme. Molecular BioSystems, 2012, 8, 220-228.	2.9	44
33	Trodusquemine displaces protein misfolded oligomers from cell membranes and abrogates their cytotoxicity through a generic mechanism. Communications Biology, 2020, 3, 435.	2.0	44
34	Î±-Helical peptidic scaffolds to target Î±-synuclein toxic species with nanomolar affinity. Nature Communications, 2021, 12, 3752.	5.8	40
35	Cell surface localised Hsp70 is a cancer specific regulator of clathrinâ€independent endocytosis. FEBS Letters, 2015, 589, 2747-2753.	1.3	37
36	The role of water in the primary nucleation of protein amyloid aggregation. Biophysical Chemistry, 2021, 269, 106520.	1.5	36

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37	Multiplicity of Î±-Synuclein Aggregated Species and Their Possible Roles in Disease. International Journal of Molecular Sciences, 2020, 21, 8043.	1.8	33
38	The Flavodoxin from Helicobacter pylori:â€‰ Structural Determinants of Thermostability and FMN Cofactor Binding. Biochemistry, 2008, 47, 627-639.	1.2	32
39	The native-state ensemble of proteins provides clues for folding, misfolding and function. Trends in Biochemical Sciences, 2006, 31, 494-496.	3.7	30
40	The extent of protein hydration dictates the preference for heterogeneous or homogeneous nucleation generating either parallel or antiparallel Î²-sheet Î±-synuclein aggregates. Chemical Science, 2020, 11, 11902-11914.	3.7	30
41	Novel Small Molecules Targeting the Intrinsically Disordered Structural Ensemble of Î±-Synuclein Protect Against Diverse Î±-Synuclein Mediated Dysfunctions. Scientific Reports, 2019, 9, 16947.	1.6	25
42	Common conformational changes in flavodoxins induced by FMN and anion binding: The structure of Helicobacter pylori apoflavodoxin. Proteins: Structure, Function and Bioinformatics, 2007, 69, 581-594.	1.5	24
43	Filling Small, Empty Protein Cavities: Structural and Energetic Consequences. Journal of Molecular Biology, 2006, 358, 701-712.	2.0	23
44	Molten Globule and Native State Ensemble of Helicobacter pylori Flavodoxin: Can Crowding, Osmolytes or Cofactors Stabilize the Native Conformation Relative to the Molten Globule?. Biophysical Journal, 2008, 95, 1913-1927.	0.2	20
45	Preparation of Î±-Synuclein Amyloid Assemblies for Toxicity Experiments. Methods in Molecular Biology, 2018, 1779, 45-60.	0.4	15
46	All-or-none amyloid disassembly via chaperone-triggered fibril unzipping favors clearance of Î±-synuclein toxic species. Proceedings of the National Academy of Sciences of the United States of America, 2021, 118, .	3.3	15
47	Conformational Stability of Helicobacter pylori Flavodoxin. Journal of Biological Chemistry, 2008, 283, 2883-2895.	1.6	13
48	The Pathological G51D Mutation in Alpha-Synuclein Oligomers Confers Distinct Structural Attributes and Cellular Toxicity. Molecules, 2022, 27, 1293.	1.7	6
49	Ca2+ is a key factor in Î±-synuclein-induced neurotoxicity. Development (Cambridge), 2016, 143, e1.1-e1.1.	1.2	5