Cristina Cecchi

List of Publications by Year
in descending order

82 papers	4,521 citations	116194 36 h-index	120465 65 g-index
84 all docs	84 docs citations	84 times ranked	6193 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	Effects of oligomer toxicity, fibril toxicity and fibril spreading in synucleinopathies. Cellular and Molecular Life Sciences, 2022, 79, 174.	2.4	45
3	AÎ² Oligomers Dysregulate Calcium Homeostasis by Mechanosensitive Activation of AMPA and NMDA Receptors. ACS Chemical Neuroscience, 2021, 12, 766-781.	1.7	35
4	The release of toxic oligomers from Î±-synuclein fibrils induces dysfunction in neuronal cells. Nature Communications, 2021, 12, 1814.	5.8	123
5	Calcium Dyshomeostasis in Alzheimerâ€™s Disease Pathogenesis. International Journal of Molecular Sciences, 2021, 22, 4914.	1.8	76
6	Exploring the Release of Toxic Oligomers from Î±-Synuclein Fibrils with Antibodies and STED Microscopy. Life, 2021, 11, 431.	1.1	17
7	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
8	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
9	Nanoscopic insights into the surface conformation of neurotoxic amyloid Î² oligomers. RSC Advances, 2020, 10, 21907-21913.	1.7	19
10	Trodusquemine displaces protein misfolded oligomers from cell membranes and abrogates their cytotoxicity through a generic mechanism. Communications Biology, 2020, 3, 435.	2.0	44
11	Targeting Pathological Amyloid Aggregates with Conformation-Sensitive Antibodies. Current Alzheimer Research, 2020, 17, 722-734.	0.7	12
12	Partial Failure of Proteostasis Systems Counteracting TDP-43 Aggregates in Neurodegenerative Diseases. International Journal of Molecular Sciences, 2019, 20, 3685.	1.8	18
13	Identification of Novel 1,3,5-Triphenylbenzene Derivative Compounds as Inhibitors of Hen Lysozyme Amyloid Fibril Formation. International Journal of Molecular Sciences, 2019, 20, 5558.	1.8	6
14	Capturing AÎ²42 aggregation in the cell. Journal of Biological Chemistry, 2019, 294, 1488-1489.	1.6	1
15	The Toxicity of Misfolded Protein Oligomers Is Independent of Their Secondary Structure. ACS Chemical Biology, 2019, 14, 1593-1600.	1.6	34
16	Probing the Origin of the Toxicity of Oligomeric Aggregates of Î±-Synuclein with Antibodies. ACS Chemical Biology, 2019, 14, 1352-1362.	1.6	33
17	The acute myeloid leukemiaâ€associated Nucleophosmin 1 gene mutations dictate amyloidogenicity of the Câ€terminal domain. FEBS Journal, 2019, 286, 2311-2328.	2.2	24
18	Trodusquemine enhances AÎ²42 aggregation but suppresses its toxicity by displacing oligomers from cell membranes. Nature Communications, 2019, 10, 225.	5.8	111

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19	Toxic HypF-N Oligomers Selectively Bind the Plasma Membrane to Impair Cell Adhesion Capability. Biophysical Journal, 2018, 114, 1357-1367.	0.2	8
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	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
22	Soluble Oligomers Require a Ganglioside to Trigger Neuronal Calcium Overload. Journal of Alzheimer's Disease, 2017, 60, 923-938.	1.2	41
23	Quantitative assessment of the degradation of aggregated TDPâ€43 mediated by the ubiquitin proteasome system and macroautophagy. FASEB Journal, 2017, 31, 5609-5624.	0.2	29
24	Structural basis of membrane disruption and cellular toxicity by Î±-synuclein oligomers. Science, 2017, 358, 1440-1443.	6.0	492
25	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
26	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
27	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
28	Single molecule experiments emphasize GM1 as a key player of the different cytotoxicity of structurally distinct AÎ²1â€“42 oligomers. Biochimica Et Biophysica Acta - Biomembranes, 2016, 1858, 386-392.	1.4	22
29	Selective Interaction between Toxic Amyloid Oligomers and the Cell Membrane Revealed by Innovative AFM Applications. Biophysical Journal, 2016, 110, 498a.	0.2	0
30	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
31	Destabilisation, aggregation, toxicity and cytosolic mislocalisation of nucleophosmin regions associated with acute myeloid leukemia. Oncotarget, 2016, 7, 59129-59143.	0.8	41
32	Nucleophosmin contains amyloidogenic regions that are able to form toxic aggregates under physiological conditions. FASEB Journal, 2015, 29, 3689-3701.	0.2	53
33	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
34	<scp>SIRT</scp>1 regulates <scp>MAPK</scp> pathways in vitiligo skin: insight into the molecular pathways of cell survival. Journal of Cellular and Molecular Medicine, 2014, 18, 514-529.	1.6	59
35	Toxicity of Protein Oligomers Is Rationalized by a Function Combining Size and Surface Hydrophobicity. ACS Chemical Biology, 2014, 9, 2309-2317.	1.6	166
36	A Complex Equilibrium among Partially Unfolded Conformations in Monomeric Transthyretin. Biochemistry, 2014, 53, 4381-4392.	1.2	12

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37	S-linolenoyl glutathione intake extends life-span and stress resistance via Sir-2.1 upregulation in Caenorhabditis elegans. Free Radical Biology and Medicine, 2014, 73, 127-135.	1.3	25
38	Plasma Membrane Injury Depends on Bilayer Lipid Composition in Alzheimer's Disease. Journal of Alzheimer's Disease, 2014, 41, 289-300.	1.2	23
39	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
40	Extracellular chaperones prevent AÎ²42-induced toxicity in rat brains. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2013, 1832, 1217-1226.	1.8	51
41	Light-responsive nanocomposite sponges for on demand chemical release with high spatial and dosage control. Journal of Materials Chemistry B, 2013, 1, 1096.	2.9	26
42	The amyloid-cell membrane system. The interplay between the biophysical features of oligomers/fibrils and cell membrane defines amyloid toxicity. Biophysical Chemistry, 2013, 182, 30-43.	1.5	96
43	Protective Properties of Novel Sâ€Acylâ€Glutathione Thioesters Against Ultravioletâ€induced Oxidative Stress. Photochemistry and Photobiology, 2013, 89, 442-452.	1.3	10
44	Lipid Rafts Mediate Amyloid-Induced Calcium Dyshomeostasis and Oxidative Stress in Alzheimerâ€™s Disease. Current Alzheimer Research, 2013, 10, 143-153.	0.7	44
45	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
46	Glycosaminoglycans (GAGs) Suppress the Toxicity of HypF-N Prefibrillar Aggregates. Journal of Molecular Biology, 2012, 421, 616-630.	2.0	17
47	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
48	SIRT1 modulates MAPK pathways in ischemicâ€“reperfused cardiomyocytes. Cellular and Molecular Life Sciences, 2012, 69, 2245-2260.	2.4	127
49	Novel S-acyl glutathione derivatives prevent amyloid oxidative stress and cholinergic dysfunction in Alzheimer disease models. Free Radical Biology and Medicine, 2012, 52, 1362-1371.	1.3	52
50	Neuronal Differentiation of Human Mesenchymal Stromal Cells Increases their Resistance to AÎ²42 Aggregate Toxicity. Journal of Alzheimer's Disease, 2011, 27, 651-664.	1.2	9
51	Membrane cholesterol enrichment prevents AÎ²-induced oxidative stress in Alzheimer's fibroblasts. Neurobiology of Aging, 2011, 32, 210-222.	1.5	41
52	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
53	Lipid rafts are primary mediators of amyloid oxidative attack on plasma membrane. Journal of Molecular Medicine, 2010, 88, 597-608.	1.7	41
54	Generation of reactive oxygen species by beta amyloid fibrils and oligomers involves different intra/extracellular pathways. Amino Acids, 2010, 38, 1101-1106.	1.2	37

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55	A causative link between the structure of aberrant protein oligomers and their toxicity. Nature Chemical Biology, 2010, 6, 140-147.	3.9	499
56	Biological Membranes as Protein Aggregation Matrices and Targets of Amyloid Toxicity. Methods in Molecular Biology, 2010, 648, 231-243.	0.4	19
57	A protective role for lipid raft cholesterol against amyloid-induced membrane damage in human neuroblastoma cells. Biochimica Et Biophysica Acta - Biomembranes, 2009, 1788, 2204-2216.	1.4	66
58	Differentiation Increases the Resistance of Neuronal Cells to Amyloid Toxicity. Neurochemical Research, 2008, 33, 2516-2531.	1.6	31
59	Replicating neuroblastoma cells in different cell cycle phases display different vulnerability to amyloid toxicity. Journal of Molecular Medicine, 2008, 86, 197-209.	1.7	23
60	Protective effect of new S-acylglutathione derivatives against amyloid-induced oxidative stress. Free Radical Biology and Medicine, 2008, 44, 1624-1636.	1.3	33
61	Curcumin protects cardiac cells against ischemia-reperfusion injury: effects on oxidative stress, NF-ÎºB, and JNK pathways. Free Radical Biology and Medicine, 2008, 45, 839-846.	1.3	71
62	Seladinâ€1/DHCR24 protects neuroblastoma cells against AÎ² toxicity by increasing membrane cholesterol content. Journal of Cellular and Molecular Medicine, 2008, 12, 1990-2002.	1.6	64
63	Increased susceptibility to amyloid toxicity in familial Alzheimer's fibroblasts. Neurobiology of Aging, 2007, 28, 863-876.	1.5	47
64	Overâ€expression of amyloid precursor protein in HEK cells alters p53 conformational state and protects against doxorubicin. Journal of Neurochemistry, 2007, 103, 322-333.	2.1	27
65	Differing molecular mechanisms appear to underlie early toxicity of prefibrillar HypF-N aggregates to different cell types. FEBS Journal, 2006, 273, 2206-2222.	2.2	15
66	Patterns of cell death triggered in two different cell lines by HypFâ€N prefibrillar aggregates. FASEB Journal, 2005, 19, 1-23.	0.2	42
67	Insights into the molecular basis of the differing susceptibility of varying cell types to the toxicity of amyloid aggregates. Journal of Cell Science, 2005, 118, 3459-3470.	1.2	85
68	Beneficial Effects of Poly (ADP-ribose) Polymerase Inhibition Against the Reperfusion Injury in Heart Transplantation. Free Radical Research, 2003, 37, 331-339.	1.5	24
69	Poly(ADP-ribose) Polymerase Activation and Cell Injury in the Course of Rat Heart Heterotopic Transplantation. Free Radical Research, 2002, 36, 79-87.	1.5	18
70	Oxidative stress and reduced antioxidant defenses in peripheral cells from familial Alzheimerâ€™s patients. Free Radical Biology and Medicine, 2002, 33, 1372-1379.	1.3	139
71	Biochemical changes and their relationship with morphological and functional findings in pig heart subjected to lasting volume overload: a possible role of acylphosphatase in the regulation of sarcoplasmic reticulum calcium pump. Basic Research in Cardiology, 2002, 97, 469-478.	2.5	9
72	Interaction between acylphosphatase and SERCA in SH-SY5Y cells. Molecular and Cellular Biochemistry, 2000, 211, 95-102.	1.4	4

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73	Early Changes Induced in the Left Ventricle by Pressure Overload. An Experimental Study on Swine Heart. Journal of Molecular and Cellular Cardiology, 2000, 32, 131-142.	0.9	21
74	Lack of SOD1 gene mutations and activity alterations in two Italian families with amyotrophic lateral sclerosis. Neuroscience Letters, 2000, 289, 157-160.	1.0	2
75	Gluthatione level is altered in lymphoblasts from patients with familial Alzheimer's disease. Neuroscience Letters, 1999, 275, 152-154.	1.0	107
76	A peptide fraction from factor VIII reduces PKC activity in cultured endothelial cells. Life Sciences, 1998, 62, 829-837.	2.0	0
77	Drosophila melanogasteracylphosphatase: A common ancestor for acylphosphatase isoenzymes of vertebrate species. FEBS Letters, 1998, 433, 205-210.	1.3	11
78	Alteration of Free Calcium Levels and Acylphosphatase Muscular Isoenzyme in Cultured Dystrophic Skin Fibroblasts. Biochemical and Biophysical Research Communications, 1997, 230, 327-330.	1.0	4
79	Alteration of acylphosphatase levels in familial Alzheimer's disease fibroblasts with presenilin gene mutations. Neuroscience Letters, 1996, 210, 153-156.	1.0	17
80	Setting Up and Statistical Evaluation of a New Haemoglobin Assay. Clinical Chemistry and Laboratory Medicine, 1995, 33, 519-24.	1.4	0
81	Crystallisation and preliminary X-ray analysis of the â€˜common-typeâ€™ acylphosphatase. FEBS Letters, 1995, 364, 243-244.	1.3	5
82	Cerebral soluble ubiquitin is increased in patients with Alzheimer's disease. Neuroscience Letters, 1993, 151, 158-161.	1.0	12