Emiliano Biasini

List of Publications by Year in descending order

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EMILIANO RIASINI

#	Article	IF	CITATIONS
1	Pharmacological inactivation of the prion protein by targeting a folding intermediate. Communications Biology, 2021, 4, 62.	4.4	30
2	Defective cyclophilin A induces TDP-43 proteinopathy: implications for amyotrophic lateral sclerosis and frontotemporal dementia. Brain, 2021, 144, 3710-3726.	7.6	13
3	Doxycycline Inhibition of a Pseudotyped Virus Transduction Does Not Translate to Inhibition of SARS-CoV-2 Infectivity. Viruses, 2021, 13, 1745.	3.3	2
4	Astrocytic microdomains from mouse cortex gain molecular control over long-term information storage and memory retention. Communications Biology, 2021, 4, 1152.	4.4	9
5	Ligands binding to the prion protein induce its proteolytic release with therapeutic potential in neurodegenerative proteinopathies. Science Advances, 2021, 7, eabj1826.	10.3	18
6	Identification of compounds inhibiting prion replication and toxicity by removing PrP ^C from the cell surface. Journal of Neurochemistry, 2020, 152, 136-150.	3.9	11
7	The Compelling Demand for an Effective PrP ^C -Directed Therapy against Prion Diseases. ACS Medicinal Chemistry Letters, 2020, 11, 2063-2067.	2.8	10
8	Understanding prion structure and conversion. Progress in Molecular Biology and Translational Science, 2020, 175, 19-30.	1.7	10
9	All-atom simulation of the HET-s prion replication. PLoS Computational Biology, 2020, 16, e1007922.	3.2	10
10	Generation, optimization and characterization of novel anti-prion compounds. Bioorganic and Medicinal Chemistry, 2020, 28, 115717.	3.0	3
11	Modeling PrPSc Generation Through Deformed Templating. Frontiers in Bioengineering and Biotechnology, 2020, 8, 590501.	4.1	12
12	The cellular prion protein beyond prion diseases. Swiss Medical Weekly, 2020, 150, w20222.	1.6	13
13	Characterization of Physical, Mechanical, and Biological Properties of SilkBridge Nerve Conduit after Enzymatic Hydrolysis. ACS Applied Bio Materials, 2020, 3, 8361-8374.	4.6	10
14	All-atom simulation of the HET-s prion replication. , 2020, 16, e1007922.		0
15	All-atom simulation of the HET-s prion replication. , 2020, 16, e1007922.		0
16	All-atom simulation of the HET-s prion replication. , 2020, 16, e1007922.		0
17	All-atom simulation of the HET-s prion replication. , 2020, 16, e1007922.		0
18	All-atom simulation of the HET-s prion replication. , 2020, 16, e1007922.		0

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19	All-atom simulation of the HET-s prion replication. , 2020, 16, e1007922.		Ο
20	All-atom simulation of the HET-s prion replication. , 2020, 16, e1007922.		0
21	All-atom simulation of the HET-s prion replication. , 2020, 16, e1007922.		0
22	Full atomistic model of prion structure and conversion. PLoS Pathogens, 2019, 15, e1007864.	4.7	98
23	Ok Google, how could I design therapeutics against prion diseases?. Current Opinion in Pharmacology, 2019, 44, 39-45.	3.5	7
24	A designer chaperone against prion diseases. Nature Biomedical Engineering, 2019, 3, 167-168.	22.5	4
25	Acute Neurotoxicity Models of Prion Disease. ACS Chemical Neuroscience, 2018, 9, 431-445.	3.5	8
26	Interfering with HuR–RNA Interaction: Design, Synthesis and Biological Characterization of Tanshinone Mimics as Novel, Effective HuR Inhibitors. Journal of Medicinal Chemistry, 2018, 61, 1483-1498.	6.4	39
27	Pharmacological Agents Targeting the Cellular Prion Protein. Pathogens, 2018, 7, 27.	2.8	40
28	Regulation of HuR structure and function by dihydrotanshinone-I. Nucleic Acids Research, 2017, 45, 9514-9527.	14.5	64
29	A Smallâ€Molecule Inhibitor of Prion Replication and Mutant Prion Protein Toxicity. ChemMedChem, 2017, 12, 1286-1292.	3.2	5
30	An antipsychotic drug exerts anti-prion effects by altering the localization of the cellular prion protein. PLoS ONE, 2017, 12, e0182589.	2.5	19
31	The Anti-Prion Antibody 15B3 Detects Toxic Amyloid-β Oligomers. Journal of Alzheimer's Disease, 2016, 53, 1485-1497.	2.6	12
32	A cationic tetrapyrrole inhibits toxic activities of the cellular prion protein. Scientific Reports, 2016, 6, 23180.	3.3	34
33	Activation of zebrafish Src family kinases by the prion protein is an amyloid-β-sensitive signal that prevents the endocytosis and degradation of E-cadherin/β-catenin complexes in vivo. Molecular Neurodegeneration, 2016, 11, 18.	10.8	30
34	The prion protein family member Shadoo induces spontaneous ionic currents in cultured cells. Scientific Reports, 2016, 6, 36441.	3.3	2
35	Common therapeutic strategies for prion and Alzheimer's diseases. Biological Chemistry, 2016, 397, 1115-1124.	2.5	5
36	Exploring the role of MKK7 in excitotoxicity and cerebral ischemia: a novel pharmacological strategy against brain injury. Cell Death and Disease, 2015, 6, e1854-e1854.	6.3	29

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37	Decoding the function of the N-terminal tail of the cellular prion protein to inspire novel therapeutic avenues for neurodegenerative diseases. Virus Research, 2015, 207, 62-68.	2.2	9
38	Role of Lipid Rafts and GM1 in the Segregation and Processing of Prion Protein. PLoS ONE, 2014, 9, e98344.	2.5	37
39	Epitope scanning indicates structural differences in brain-derived monomeric and aggregated mutant prion proteins related to genetic prion diseases. Biochemical Journal, 2013, 454, 417-425.	3.7	12
40	A Mutant Prion Protein Sensitizes Neurons to Glutamate-Induced Excitotoxicity. Journal of Neuroscience, 2013, 33, 2408-2418.	3.6	43
41	An N-terminal Fragment of the Prion Protein Binds to Amyloid-β Oligomers and Inhibits Their Neurotoxicity in Vivo. Journal of Biological Chemistry, 2013, 288, 7857-7866.	3.4	162
42	Infectious and Pathogenic Forms of PrP. , 2013, , 135-146.		0
43	Targeting the cellular prion protein to treat neurodegeneration. Future Medicinal Chemistry, 2012, 4, 1655-1658.	2.3	10
44	Ion channels induced by the prion protein. Prion, 2012, 6, 40-45.	1.8	33
45	Prion protein at the crossroads of physiology and disease. Trends in Neurosciences, 2012, 35, 92-103.	8.6	150
46	The N-Terminal, Polybasic Region of PrPC Dictates the Efficiency of Prion Propagation by Binding to PrPSc. Journal of Neuroscience, 2012, 32, 8817-8830.	3.6	66
47	The Toxicity of a Mutant Prion Protein Is Cell-Autonomous, and Can Be Suppressed by Wild-Type Prion Protein on Adjacent Cells. PLoS ONE, 2012, 7, e33472.	2.5	13
48	A Drug-Based Cellular Assay (DBCA) for studying cytotoxic and cytoprotective activities of the prion protein: A practical guide. Methods, 2011, 53, 214-219.	3.8	12
49	An N-terminal Polybasic Domain and Cell Surface Localization Are Required for Mutant Prion Protein Toxicity. Journal of Biological Chemistry, 2011, 286, 14724-14736.	3.4	55
50	The N-Terminal, Polybasic Region Is Critical for Prion Protein Neuroprotective Activity. PLoS ONE, 2011, 6, e25675.	2.5	38
51	The hydrophobic core region governs mutant prion protein aggregation and intracellular retention. Biochemical Journal, 2010, 430, 477-486.	3.7	22
52	Mutant Prion Protein Expression Is Associated with an Alteration of the Rab GDP Dissociation Inhibitor α (GDI)/Rab11 Pathway. Molecular and Cellular Proteomics, 2010, 9, 611-622.	3.8	35
53	A Novel, Drug-based, Cellular Assay for the Activity of Neurotoxic Mutants of the Prion Protein. Journal of Biological Chemistry, 2010, 285, 7752-7765.	3.4	34
54	Synthetic amyloid-β oligomers impair long-term memory independently of cellular prion protein. Proceedings of the National Academy of Sciences of the United States of America, 2010, 107, 2295-2300.	7.1	435

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55	Immunopurification of Pathological Prion Protein Aggregates. PLoS ONE, 2009, 4, e7816.	2.5	17
56	Multiple biochemical similarities between infectious and nonâ€infectious aggregates of a prion protein carrying an octapeptide insertion. Journal of Neurochemistry, 2008, 104, 1293-1308.	3.9	34
57	Nonâ€infectious aggregates of the prion protein react with several PrP ^{Sc} â€directed antibodies. Journal of Neurochemistry, 2008, 105, 2190-2204.	3.9	44
58	GFP-tagged mutant prion protein forms intra-axonal aggregates in transgenic mice. Neurobiology of Disease, 2008, 31, 20-32.	4.4	28
59	Aggregated, Wild-Type Prion Protein Causes Neurological Dysfunction and Synaptic Abnormalities. Journal of Neuroscience, 2008, 28, 13258-13267.	3.6	60
60	Human prion proteins with pathogenic mutations share common conformational changes resulting in enhanced binding to glycosaminoglycans. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 7546-7551.	7.1	55
61	Proteomic analysis of spinal cord of presymptomatic amyotrophic lateral sclerosis G93A SOD1 mouse. Biochemical and Biophysical Research Communications, 2007, 353, 719-725.	2.1	72
62	Redox regulation of cyclophilin A by glutathionylation. Proteomics, 2006, 6, 817-825.	2.2	43
63	Analysis of the cerebellar proteome in a transgenic mouse model of inherited prion disease reveals preclinical alteration of calcineurin activity. Proteomics, 2006, 6, 2823-2834.	2.2	19
64	Proteasome inhibition and aggregation in Parkinson's disease: a comparative study in untransfected and transfected cells. Journal of Neurochemistry, 2004, 88, 545-553.	3.9	67
65	Mutant PrP Is Delayed in Its Exit from the Endoplasmic Reticulum, but Neither Wild-type nor Mutant PrP Undergoes Retrotranslocation Prior to Proteasomal Degradation. Journal of Biological Chemistry, 2003, 278, 21732-21743.	3.4	205