## Massimo Stefani

List of Publications by Year in descending order

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	34493	23841
13,791	54	115
citations	h-index	g-index
143	143	13962
docs citations	times ranked	citing authors
	13,791 citations 143 docs citations	13,79154citationsh-index143143docs citationstimes ranked

#	Article	IF	CITATIONS
1	Xenohormesis underlyes the anti-aging and healthy properties of olive polyphenols. Mechanisms of Ageing and Development, 2022, 202, 111620.	2.2	10
2	The Transthyretin/Oleuropein Aglycone Complex: A New Tool against TTR Amyloidosis. Pharmaceuticals, 2022, 15, 277.	1.7	3
3	Natural Compound from Olive Oil Inhibits S100A9 Amyloid Formation and Cytotoxicity: Implications for Preventing Alzheimer's Disease. ACS Chemical Neuroscience, 2021, 12, 1905-1918.	1.7	18
4	Olive Polyphenols: Antioxidant and Anti-Inflammatory Properties. Antioxidants, 2021, 10, 1044.	2.2	92
5	Structural Features and Toxicity of α-Synuclein Oligomers Grown in the Presence of DOPAC. International Journal of Molecular Sciences, 2021, 22, 6008.	1.8	8
6	EVOO Polyphenols Relieve Synergistically Autophagy Dysregulation in a Cellular Model of Alzheimer's Disease. International Journal of Molecular Sciences, 2021, 22, 7225.	1.8	13
7	Insight into the molecular mechanism underlying the inhibition of α-synuclein aggregation by hydroxytyrosol. Biochemical Pharmacology, 2020, 173, 113722.	2.0	25
8	Allium roseum L. extract inhibits amyloid beta aggregation and toxicity involved in Alzheimer's disease. PLoS ONE, 2020, 15, e0223815.	1.1	11
9	The Amphipathic GM1 Molecule Stabilizes Amyloid Aggregates, Preventing their Cytotoxicity. Biophysical Journal, 2020, 119, 326-336.	0.2	7
10	Healthy Effects of Plant Polyphenols: Molecular Mechanisms. International Journal of Molecular Sciences, 2020, 21, 1250.	1.8	265
11	Healthspan Maintenance and Prevention of Parkinson's-like Phenotypes with Hydroxytyrosol and Oleuropein Aglycone in C. elegans. International Journal of Molecular Sciences, 2020, 21, 2588.	1.8	110
12	Oleuropein aglycone and hydroxytyrosol interfere differently with toxic Aβ1-42 aggregation. Food and Chemical Toxicology, 2019, 129, 1-12.	1.8	46
13	1,2,4-trihydroxynaphthalene-2- <i>O</i> -β-D-glucopyranoside: A new powerful antioxidant and inhibitor of Al² <sub>42</sub> aggregation isolated from the leaves of <i>Lawsonia inermis</i> . Natural Product Research, 2019, 33, 1406-1414.	1.0	11
14	1,2,4â€ŧrihydroxynaphthaleneâ€2â€Oâ€Î²â€Dâ€glucopyranoside delays amyloidâ€Î² <sub>42</sub> aggregation reduces amyloid cytotoxicity. BioFactors, 2018, 44, 272-280.	and 2.6	2
15	A new purified Lawsoniaside remodels amyloid-β42 fibrillation into a less toxic and non-amyloidogenic pathway. International Journal of Biological Macromolecules, 2018, 114, 830-835.	3.6	1
16	Diet Supplementation with Hydroxytyrosol Ameliorates Brain Pathology and Restores Cognitive Functions in a Mouse Model of Amyloid-β Deposition. Journal of Alzheimer's Disease, 2018, 63, 1161-1172.	1.2	39
17	Oleuropein aglycone: A polyphenol with different targets against amyloid toxicity. Biochimica Et Biophysica Acta - General Subjects, 2018, 1862, 1432-1442.	1.1	30
18	Toxic HypF-N Oligomers Selectively Bind the Plasma Membrane to Impair Cell Adhesion Capability. Biophysical Journal, 2018, 114, 1357-1367.	0.2	8

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19	Screening for amyloid-β aggregation inhibitor and neuronal toxicity of eight Tunisian medicinal plants. Industrial Crops and Products, 2018, 111, 823-833.	2.5	14
20	Oleuropein aglycone stabilizes the monomeric α-synuclein and favours the growth of non-toxic aggregates. Scientific Reports, 2018, 8, 8337.	1.6	54
21	Oleuropein Aglycone Protects against MAO-A-Induced Autophagy Impairment and Cardiomyocyte Death through Activation of TFEB. Oxidative Medicine and Cellular Longevity, 2018, 2018, 1-13.	1.9	35
22	A FTIR microspectroscopy study of the structural and biochemical perturbations induced by natively folded and aggregated transthyretin in HL-1 cardiomyocytes. Scientific Reports, 2018, 8, 12508.	1.6	31
23	Oleuropein aglycone and polyphenols from olive mill waste water ameliorate cognitive deficits and neuropathology. British Journal of Clinical Pharmacology, 2017, 83, 54-62.	1.1	70
24	A specific nanobody prevents amyloidogenesis of D76N β2-microglobulin in vitro and modifies its tissue distribution in vivo. Scientific Reports, 2017, 7, 46711.	1.6	18
25	Soluble Oligomers Require a Ganglioside to Trigger Neuronal Calcium Overload. Journal of Alzheimer's Disease, 2017, 60, 923-938.	1.2	41
26	Olive polyphenols: new promising agents to combat aging-associated neurodegeneration. Expert Review of Neurotherapeutics, 2017, 17, 345-358.	1.4	99
27	Nutraceutical Properties of Olive Oil Polyphenols. An Itinerary from Cultured Cells through Animal Models to Humans. International Journal of Molecular Sciences, 2016, 17, 843.	1.8	222
28	The Polyphenol Oleuropein Aglycone Modulates the PARP1-SIRT1 Interplay: AnÂln Vitro and In Vivo Study. Journal of Alzheimer's Disease, 2016, 54, 737-750.	1.2	36
29	Biochemical and Electrophysiological Modification of Amyloid Transthyretin on Cardiomyocytes. Biophysical Journal, 2016, 111, 2024-2038.	0.2	19
30	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
31	Single molecule experiments emphasize GM1 as a key player of the different cytotoxicity of structurally distinct Al̂21–42 oligomers. Biochimica Et Biophysica Acta - Biomembranes, 2016, 1858, 386-392.	1.4	22
32	Molecular insights into cell toxicity of a novel familial amyloidogenic variant of β2â€microglobulin. Journal of Cellular and Molecular Medicine, 2016, 20, 1443-1456.	1.6	23
33	The polyphenol Oleuropein aglycone hinders the growth of toxic transthyretin amyloid assemblies. Journal of Nutritional Biochemistry, 2016, 30, 153-166.	1.9	39
34	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
35	Nutraceuticals and amyloid neurodegenerative diseases: a focus on natural phenols. Expert Review of Neurotherapeutics, 2015, 15, 41-52.	1.4	56
36	Oleuropein Aglycone: A Possible Drug against Degenerative Conditions. In Vivo Evidence of its Effectiveness against Alzheimer's Disease. Journal of Alzheimer's Disease, 2015, 45, 679-688.	1.2	59

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37	Oleuropein aglycone protects against pyroglutamylated-3 amyloid-ß toxicity: biochemical, epigenetic and functional correlates. Neurobiology of Aging, 2015, 36, 648-663.	1.5	91
38	Oleuropein aglycone induces autophagy <i>via</i> the AMPK/mTOR signalling pathway: a mechanistic insight. Oncotarget, 2015, 6, 35344-35357.	0.8	108
39	Employing Alzheimer Disease Animal Models for Translational Research: Focus on Dietary Components. Neurodegenerative Diseases, 2014, 13, 131-134.	0.8	25
40	Amyloid Aggregation: Role of Biological Membranes and the Aggregate–Membrane System. Journal of Physical Chemistry Letters, 2014, 5, 517-527.	2.1	88
41	Oleuropein aglycone counteracts Al²42 toxicity in the rat brain. Neuroscience Letters, 2014, 558, 67-72.	1.0	66
42	Beneficial properties of natural phenols: Highlight on protection against pathological conditions associated with amyloid aggregation. BioFactors, 2014, 40, 482-493.	2.6	83
43	Mild exposure of RIN-5F β-cells to human islet amyloid polypeptide aggregates upregulates antioxidant enzymes via NADPH oxidase-RACE: An hormetic stimulus. Redox Biology, 2014, 2, 114-122.	3.9	15
44	Protein Folding and Aggregation into Amyloid: The Interference by Natural Phenolic Compounds. International Journal of Molecular Sciences, 2013, 14, 12411-12457.	1.8	180
45	Different ataxin-3 amyloid aggregates induce intracellular Ca 2+ deregulation by different mechanisms in cerebellar granule cells. Biochimica Et Biophysica Acta - Molecular Cell Research, 2013, 1833, 3155-3165.	1.9	22
46	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
47	Oleuropein Aglycone Protects Transgenic C. elegans Strains Expressing Aβ42 by Reducing Plaque Load and Motor Deficit. PLoS ONE, 2013, 8, e58893.	1.1	116
48	The Polyphenol Oleuropein Aglycone Protects TgCRND8 Mice against Aß Plaque Pathology. PLoS ONE, 2013, 8, e71702.	1.1	202
49	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
50	Toxic effects of amyloid fibrils on cell membranes: the importance of ganglioside GM1. FASEB Journal, 2012, 26, 818-831.	0.2	118
51	Structural features and cytotoxicity of amyloid oligomers: Implications in Alzheimer's disease and other diseases with amyloid deposits. Progress in Neurobiology, 2012, 99, 226-245.	2.8	154
52	Interactions of lysozyme with phospholipid vesicles: effects of vesicle biophysical features on protein misfolding and aggregation. Soft Matter, 2012, 8, 9115.	1.2	28
53	Lysozyme interaction with negatively charged lipid bilayers: protein aggregation and membrane fusion. Soft Matter, 2012, 8, 4524.	1.2	32
54	Neuronal Differentiation of Human Mesenchymal Stromal Cells Increases their Resistance to AÎ <sup>2</sup> 42 Aggregate Toxicity. Journal of Alzheimer's Disease, 2011, 27, 651-664.	1.2	9

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55	Does azurin bind to the transactivation domain of p53? A Trp phosphorescence study. Biophysical Chemistry, 2011, 159, 287-293.	1.5	9
56	Aβ(1-42) Aggregates into Non-Toxic Amyloid Assemblies in the Presence of the Natural Polyphenol Oleuropein Aglycon. Current Alzheimer Research, 2011, 8, 841-852.	0.7	113
57	Effect of Tetracyclines on the Dynamics of Formation and Destructuration of β2-Microglobulin Amyloid Fibrils. Journal of Biological Chemistry, 2011, 286, 2121-2131.	1.6	87
58	Structural Polymorphism of Amyloid Oligomers and Fibrils Underlies Different Fibrillization Pathways: Immunogenicity and Cytotoxicity. Current Protein and Peptide Science, 2010, 11, 343-354.	0.7	33
59	Oleuropein aglycon prevents cytotoxic amyloid aggregation of human amylinâ~†. Journal of Nutritional Biochemistry, 2010, 21, 726-735.	1.9	107
60	Biochemical and biophysical features of both oligomer/fibril and cell membrane in amyloid cytotoxicity. FEBS Journal, 2010, 277, 4602-4613.	2.2	164
61	A causative link between the structure of aberrant protein oligomers and their toxicity. Nature Chemical Biology, 2010, 6, 140-147.	3.9	499
62	Embryonic stem and haematopoietic progenitor cells resist to AÎ <sup>2</sup> oligomer toxicity and maintain the differentiation potency in culture. Amyloid: the International Journal of Experimental and Clinical Investigation: the Official Journal of the International Society of Amyloidosis, 2010, 17, 137-145.	1.4	3
63	Protein Aggregation Diseases: Toxicity of Soluble Prefibrillar Aggregates and Their Clinical Significance. Methods in Molecular Biology, 2010, 648, 25-41.	0.4	21
64	Cholesterol in Alzheimers Disease: Unresolved Questions. Current Alzheimer Research, 2009, 6, 15-29.	0.7	123
65	Proteomic analysis of cells exposed to prefibrillar aggregates of HypF-N. Biochimica Et Biophysica Acta - Proteins and Proteomics, 2009, 1794, 1243-1250.	1.1	3
66	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
67	Synthetic Lipid Vesicles Recruit Native-Like Aggregates and Affect the Aggregation Process of the Prion Ure2p: Insights on Vesicle Permeabilization and Charge Selectivity. Biophysical Journal, 2009, 96, 3319-3330.	0.2	16
68	Differentiation Increases the Resistance of Neuronal Cells to Amyloid Toxicity. Neurochemical Research, 2008, 33, 2516-2531.	1.6	31
69	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
70	Biological function in a non-native partially folded state of a protein. EMBO Journal, 2008, 27, 1525-35.	3.5	32
71	Protein Folding and Misfolding on Surfaces. International Journal of Molecular Sciences, 2008, 9, 2515-2542.	1.8	67
72	The (1–63) Region of the p53 Transactivation Domain Aggregates In Vitro into Cytotoxic Amyloid Assemblies. Biophysical Journal, 2008, 94, 3635-3646.	0.2	50

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73	Nonspecific Interaction of Prefibrillar Amyloid Aggregates with Glutamatergic Receptors Results in Ca2+ Increase in Primary Neuronal Cells. Journal of Biological Chemistry, 2008, 283, 29950-29960.	1.6	46
74	Â2-Microglobulin is potentially neurotoxic, but the blood brain barrier is likely to protect the brain from its toxicity. Nephrology Dialysis Transplantation, 2008, 24, 1176-1181.	0.4	31
75	Generic Cell Dysfunction in Neurodegenerative Disorders: Role of Surfaces in Early Protein Misfolding, Aggregation, and Aggregate Cytotoxicity. Neuroscientist, 2007, 13, 519-531.	2.6	69
76	The intrachain disulfide bridge is responsible of the unusual stability properties of novel acylphosphatase fromEscherichia coli. FEBS Letters, 2006, 580, 6763-6768.	1.3	10
77	Natively Folded HypF-N and Its Early Amyloid Aggregates Interact with Phospholipid Monolayers and Destabilize Supported Phospholipid Bilayers. Biophysical Journal, 2006, 91, 4575-4588.	0.2	46
78	Assessing the role of aromatic residues in the amyloid aggregation of human muscle acylphosphatase. Protein Science, 2006, 15, 862-870.	3.1	107
79	Prefibrillar Amyloid Aggregates Could Be Generic Toxins in Higher Organisms. Journal of Neuroscience, 2006, 26, 8160-8167.	1.7	222
80	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
81	The Yeast Prion Ure2p Native-like Assemblies Are Toxic to Mammalian Cells Regardless of Their Aggregation State*. Journal of Biological Chemistry, 2006, 281, 15337-15344.	1.6	41
82	Preliminary characterization of two different crystal forms of acylphosphatase from the hyperthermophile archaeonSulfolobus solfataricus. Acta Crystallographica Section F: Structural Biology Communications, 2005, 61, 144-146.	0.7	3
83	Structure, conformational stability, and enzymatic properties of acylphosphatase from the hyperthermophile Sulfolobus solfataricus. Proteins: Structure, Function and Bioinformatics, 2005, 62, 64-79.	1.5	43
84	Patterns of cell death triggered in two different cell lines by HypFâ€N prefibrillar aggregates. FASEB Journal, 2005, 19, 1-23.	0.2	42
85	Investigating the Effects of Mutations on Protein Aggregation in the Cell. Journal of Biological Chemistry, 2005, 280, 10607-10613.	1.6	75
86	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
87	Reversal of Protein Aggregation Provides Evidence for Multiple Aggregated States. Journal of Molecular Biology, 2005, 346, 603-616.	2.0	86
88	Aggregation of the Acylphosphatase from Sulfolobus solfataricus. Journal of Biological Chemistry, 2004, 279, 14111-14119.	1.6	99
89	Three-dimensional structural characterization of a novelDrosophila melanogasteracylphosphatase. Acta Crystallographica Section D: Biological Crystallography, 2004, 60, 1177-1179.	2.5	18
90	Selection of antibody fragments specific for anα-helix region of acylphosphatase. Journal of Molecular Recognition, 2004, 17, 62-66.	1.1	3

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91	Studying the Folding Process of the Acylphosphatase fromSulfolobus solfataricus. A Comparative Analysis with Other Proteins from the Same Superfamilyâ€. Biochemistry, 2004, 43, 9116-9126.	1.2	19
92	Prefibrillar Amyloid Protein Aggregates Share Common Features of Cytotoxicity. Journal of Biological Chemistry, 2004, 279, 31374-31382.	1.6	346
93	Protein misfolding and aggregation: new examples in medicine and biology of the dark side of the protein world. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2004, 1739, 5-25.	1.8	366
94	Monitoring the Process of HypF Fibrillization and Liposome Permeabilization by Protofibrils. Journal of Molecular Biology, 2004, 338, 943-957.	2.0	101
95	Protein aggregation and aggregate toxicity: new insights into protein folding, misfolding diseases and biological evolution. Journal of Molecular Medicine, 2003, 81, 678-699.	1.7	1,444
96	Rationalization of the effects of mutations on peptide andprotein aggregation rates. Nature, 2003, 424, 805-808.	13.7	1,013
97	Relative Influence of Hydrophobicity and Net Charge in the Aggregation of Two Homologous Proteinsâ€. Biochemistry, 2003, 42, 15078-15083.	1.2	115
98	Comparison of the Folding Processes of Distantly Related Proteins. Importance of Hydrophobic Content in Folding. Journal of Molecular Biology, 2003, 330, 577-591.	2.0	47
99	Studies of the aggregation of mutant proteins in vitro provide insights into the genetics of amyloid diseases. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 16419-16426.	3.3	268
100	Crystal Structure and Anion Binding in the Prokaryotic Hydrogenase Maturation Factor HypF Acylphosphatase-like Domain. Journal of Molecular Biology, 2002, 321, 785-796.	2.0	63
101	Crystallization and preliminary X-ray characterization of the acylphosphatase-like domain from theEscherichia colihydrogenase maturation factor HypF. Acta Crystallographica Section D: Biological Crystallography, 2002, 58, 524-525.	2.5	6
102	Inherent toxicity of aggregates implies a common mechanism for protein misfolding diseases. Nature, 2002, 416, 507-511.	13.7	2,322
103	Kinetic partitioning of protein folding and aggregation. Nature Structural Biology, 2002, 9, 137-143.	9.7	373
104	Detection of two partially structured species in the folding process of the amyloidogenic protein β2-microglobulin. Journal of Molecular Biology, 2001, 307, 379-391.	2.0	115
105	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
106	Folding and Aggregation Are Selectively Influenced by the Conformational Preferences of the α-Helices of Muscle Acylphosphatase. Journal of Biological Chemistry, 2001, 276, 37149-37154.	1.6	45
107	Solution conditions can promote formation of either amyloid protofilaments or mature fibrils from the HypF Nâ€ŧerminal domain. Protein Science, 2001, 10, 2541-2547.	3.1	47
108	Initial denaturing conditions influence the slow folding phase of acylphosphatase associated with proline isomerization. Protein Science, 2000, 9, 1466-1473.	3.1	5

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109	Stabilisation of α-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
110	Mutational analysis of acylphosphatase suggests the importance of topology and contact order in protein folding. Nature Structural Biology, 1999, 6, 1005-1009.	9.7	257
111	The lowMrphosphotyrosine protein phosphatase behaves differently when phosphorylated at Tyr131or Tyr132by Src kinase. FEBS Letters, 1999, 456, 73-78.	1.3	63
112	Thermodynamics and Kinetics of Folding of Common-Type Acylphosphatase:Â Comparison to the Highly Homologous Muscle Isoenzymeâ€. Biochemistry, 1999, 38, 2135-2142.	1.2	51
113	Sequence-specific recognition of peptide substrates by the lowMrphosphotyrosine protein phosphatase isoforms. FEBS Letters, 1998, 422, 213-217.	1.3	13
114	Conformational Stability of Muscle Acylphosphatase:Â The Role of Temperature, Denaturant Concentration, and pHâ€. Biochemistry, 1998, 37, 1447-1455.	1.2	57
115	Structural characterization of the transition state for folding of muscle acylphosphatase 1 1Edited by P. E. Wright. Journal of Molecular Biology, 1998, 283, 893-903.	2.0	54
116	Structural and Kinetic Investigations on the 15â^'21 and 42â^'45 Loops of Muscle Acylphosphatase:Â Evidence for Their Involvement in Enzyme Catalysis and Conformational Stabilizationâ€. Biochemistry, 1997, 36, 7217-7224.	1.2	14
117	Structural, catalytic, and functional properties of low Mr phosphotyrosine protein phosphatases. Evidence of a long evolutionary history. International Journal of Biochemistry and Cell Biology, 1997, 29, 279-292.	1.2	51
118	Structure and function of the low Mr phosphotyrosine protein phosphatases. BBA - Proteins and Proteomics, 1997, 1341, 137-156.	2.1	95
119	Looking for Residues Involved in the Muscle Acylphosphatase Catalytic Mechanism and Structural Stabilization:  Role of Asn41, Thr42, and Thr46. Biochemistry, 1996, 35, 7077-7083.	1.2	48
120	C-terminal region contributes to muscle acylphosphatase three-dimensional structure stabilisation. FEBS Letters, 1996, 384, 172-176.	1.3	12
121	Expression, Purification, and Characterization of Acylphosphatase Muscular Isoenzyme as Fusion Protein with GlutathioneS-Transferase. Protein Expression and Purification, 1995, 6, 799-805.	0.6	28
122	Properties of N-terminus truncated and C-terminus mutated muscle acylphosphatases. FEBS Letters, 1995, 362, 175-179.	1.3	11
123	Arginine-23 is involved in the catalytic site of muscle acylphosphatase. BBA - Proteins and Proteomics, 1994, 1208, 75-80.	2.1	31
124	The crystal structure of a low-molecular-weight phosphotyrosine protein phosphatase. Nature, 1994, 370, 575-578.	13.7	224
125	Equilibrium Unfolding Studies of Horse Muscle Acylphosphatase. FEBS Journal, 1994, 225, 811-817.	0.2	20
126	Aspartic-129 is an essential residue in the catalytic mechanism of the lowMrphosphotyrosine protein phosphatase. FEBS Letters, 1994, 350, 328-332.	1.3	47

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127	Crystallisation of a low molecular weight phosphotyrosine protein phosphatase from bovine liver. FEBS Letters, 1994, 343, 107-108.	1.3	3
128	Dephosphorylation of tyrosine phosphorylated synthetic peptides by rat liver phosphotyrosine protein phosphatase isoenzymes. FEBS Letters, 1993, 326, 131-134.	1.3	61
129	Expression and turnover of acylphosphatase (muscular isoenzyme) in L6 myoblasts during myogenesis. Archives of Biochemistry and Biophysics, 1992, 294, 261-264.	1.4	23
130	Preparation and properties of <i>des</i> â€Tyr <sup>98</sup> and <i>des</i> â€Arg <sup>97</sup> â€Tyr <sup>98</sup> acylphosphatase (muscular isoenzyme). International Journal of Peptide and Protein Research, 1991, 38, 278-284.	0.1	3
131	The sequence-specific assignment of the 1H-NMR spectrum of an enzyme, horse-muscle acylphosphatase. FEBS Journal, 1989, 182, 85-93.	0.2	19
132	Mobility of secondary structure units of horse-muscle acylphosphatase. Relation to antigenicity. FEBS Journal, 1989, 185, 99-103.	0.2	10
133	Guinea pig acylphosphatase: The amino acid sequence. The Protein Journal, 1988, 7, 417-426.	1.1	10
134	Duck skeletal muscle acylphosphatase: Primary structure. The Protein Journal, 1986, 5, 307-321.	1.1	12
135	Rabbit skeletal muscle acylphosphatase: The amino acid sequence of form Ra1. Archives of Biochemistry and Biophysics, 1985, 241, 418-424.	1.4	28
136	The primary structure of turkey muscle acylphosphatase. FEBS Journal, 1983, 137, 269-277.	0.2	21
137	Hydrolysis by horse muscle acylphosphatase of (Ca2+ + Mg2+)-ATPase phosphorylated intermediate. Archives of Biochemistry and Biophysics, 1981, 208, 37-41.	1.4	34
138	Effect of acylphosphates on Ca2+ uptake by sarcoplasmic reticulum vesicles. Archives of Biochemistry and Biophysics, 1980, 200, 357-363.	1.4	12
139	PREPARATION AND SOME PROPERTIES OF A DIMERIC FORM (Sâ€S) OF HORSE MUSCLE ACYLPHOSPHATASE. International Journal of Peptide and Protein Research, 1979, 14, 227-233.	0.1	5