Niccolo Taddei

List of Publications by Year
in descending order

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99	9,318	57758 44	38395 95
papers	citations	h-index	g-index

102	102	102	8601
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Butyrate-Rich Diets Improve Redox Status and Fibrin Lysis in BehÃ§etâ€™s Syndrome. Circulation Research, 2021, 128, 278-280.	4.5	31
2	Neutrophil-mediated mechanisms of damage and in-vitro protective effect of colchicine in non-vascular BehÃ§et's syndrome. Clinical and Experimental Immunology, 2021, 206, 410-421.	2.6	24
3	The Impact of Oxidative Stress in Male Infertility. Frontiers in Molecular Biosciences, 2021, 8, 799294.	3.5	62
4	NADPH oxidase may be the key-player in skin response to the dietary factors: fibroblasts-keratinocytes co-culture studies. Free Radical Biology and Medicine, 2021, 177, S133.	2.9	0
5	Super-Resolution Microscopy Reveals an Altered Fibrin Network in Cirrhosis: The Key Role of Oxidative Stress in Fibrinogen Structural Modifications. Antioxidants, 2020, 9, 737.	5.1	9
6	Cadmium-Induced Cytotoxicity: Effects on Mitochondrial Electron Transport Chain. Frontiers in Cell and Developmental Biology, 2020, 8, 604377.	3.7	55
7	On the Suitability of Low-Cost Compact Instrumentation for Blood Impedance Measurements. IEEE Transactions on Instrumentation and Measurement, 2019, 68, 2412-2424.	4.7	8
8	Stem-Cell-Derived Circulating Progenitors Dysfunction in BehÃ§et's Syndrome Patients Correlates With Oxidative Stress. Frontiers in Immunology, 2019, 10, 2877.	4.8	11
9	Fibroblasts to Keratinocytes Redox Signaling: The Possible Role of ROS in Psoriatic Plaque Formation. Antioxidants, 2019, 8, 566.	5.1	18
10	ROSâ€challenged keratinocytes as a new model for oxidative stressâ€mediated skin diseases. Journal of Cellular Biochemistry, 2019, 120, 28-36.	2.6	21
11	Oxidative stress management during non-invasive ventilation in acute respiratory failure. Internal and Emergency Medicine, 2018, 13, 141-142.	2.0	0
12	Commentary to the review article: Subedi S, Yu Q, Chen Z, Shi Y. Management of pediatric psoriasis with acitretin: A review. Dermatol Ther. 2018 Jan;31(1). Dermatologic Therapy, 2018, 31, e12700.	1.7	3
13	Oxidative stress and inflammation: new molecular targets for cardiovascular diseases. Internal and Emergency Medicine, 2018, 13, 647-649.	2.0	8
14	A Biochemical Approach to Detect Oxidative Stress in Infertile Women Undergoing Assisted Reproductive Technology Procedures. International Journal of Molecular Sciences, 2018, 19, 592.	4.1	39
15	Sirt1 Protects against Oxidative Stress-Induced Apoptosis in Fibroblasts from Psoriatic Patients: A New Insight into the Pathogenetic Mechanisms of Psoriasis. International Journal of Molecular Sciences, 2018, 19, 1572.	4.1	49
16	Secukinumab reduces plasma oxidative stress in psoriasis: A case-based experience. Dermatologic Therapy, 2018, 31, e12675.	1.7	6
17	Food Allergen-IgE Impedance Measurements Evaluation in Allergic Children. Lecture Notes in Electrical Engineering, 2018, , 91-97.	0.4	0
18	Redox status alterations during the competitive season in Ã©lite soccer players: focus on peripheral leukocyte-derived ROS. Internal and Emergency Medicine, 2017, 12, 777-788.	2.0	31

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19	Erythrocyte Membrane Fluidity Alterations in Sudden Sensorineural Hearing Loss Patients: The Role of Oxidative Stress. Thrombosis and Haemostasis, 2017, 117, 2334-2345.	3.4	24
20	Low dose cytokines reduce oxidative stress in primary lesional fibroblasts obtained from psoriatic patients. Journal of Dermatological Science, 2016, 83, 242-244.	1.9	23
21	Erythrocyte oxidative stress is associated with cell deformability in patients with retinal vein occlusion. Journal of Thrombosis and Haemostasis, 2016, 14, 2287-2297.	3.8	42
22	SIRT1 activity is decreased in lesional psoriatic skin. Internal and Emergency Medicine, 2016, 11, 891-893.	2.0	9
23	Neutrophil Activation Promotes Fibrinogen Oxidation and Thrombus Formation in BehÃ§et Disease. Circulation, 2016, 133, 302-311.	1.6	125
24	Treatment with low-dose cytokines reduces oxidative-mediated injury in perilesional keratinocytes from vitiligo skin. Journal of Dermatological Science, 2015, 79, 163-170.	1.9	49
25	Oxidative Modification of Fibrinogen Is Associated With Altered Function and Structure in the Subacute Phase of Myocardial Infarction. Arteriosclerosis, Thrombosis, and Vascular Biology, 2014, 34, 1355-1361.	2.4	77
26	<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.	3.6	59
27	Altered redox status in the blood of psoriatic patients: involvement of NADPH oxidase and role of anti-TNF-Î± therapy. Redox Report, 2013, 18, 100-106.	4.5	69
28	Protective Properties of Novel Sâ€Acylâ€Glutathione Thioesters Against Ultravioletâ€induced Oxidative Stress. Photochemistry and Photobiology, 2013, 89, 442-452.	2.5	10
29	Glycosaminoglycans (GAGs) Suppress the Toxicity of HypF-N Prefibrillar Aggregates. Journal of Molecular Biology, 2012, 421, 616-630.	4.2	17
30	SIRT1 modulates MAPK pathways in ischemicâ€“reperfused cardiomyocytes. Cellular and Molecular Life Sciences, 2012, 69, 2245-2260.	5.4	127
31	Circulating dendritic cell subsets in psoriatic patients before and after biologic therapy. Journal of Dermatology, 2012, 39, 274-274.	1.2	1
32	Antioxidant Capacity Evaluation In Different Extravirgin Olive Oils. Medicine and Science in Sports and Exercise, 2010, 42, 793.	0.4	0
33	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.	4.2	21
34	The Involvement of Smac/DIABLO, p53, NF-kB, and MAPK Pathways in Apoptosis of Keratinocytes from Perilesional Vitiligo Skin: Protective Effects of Curcumin and Capsaicin. Antioxidants and Redox Signaling, 2010, 13, 1309-1321.	5.4	58
35	A Computational Approach for Identifying the Chemical Factors Involved in the Glycosaminoglycans-Mediated Acceleration of Amyloid Fibril Formation. PLoS ONE, 2010, 5, e11363.	2.5	9
36	Ultrastructural and functional alterations of mitochondria in perilesional vitiligo skin. Journal of Dermatological Science, 2009, 54, 157-167.	1.9	61

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37	Agitation and High Ionic Strength Induce Amyloidogenesis of a Folded PDZ Domain in Native Conditions. Biophysical Journal, 2009, 96, 2289-2298.	0.5	32
38	Conformational Properties of Unfolded HypF-N. Journal of Physical Chemistry B, 2009, 113, 16209-16213.	2.6	17
39	Biological function in a non-native partially folded state of a protein. EMBO Journal, 2008, 27, 1525-35.	7.8	32
40	The Folding Process of Acylphosphatase from Escherichia coli is Remarkably Accelerated by the Presence of a Disulfide Bond. Journal of Molecular Biology, 2008, 379, 1107-1118.	4.2	14
41	Aggregation Propensity of the Human Proteome. PLoS Computational Biology, 2008, 4, e1000199.	3.2	81
42	The Distribution of Residues in a Polypeptide Sequence Is a Determinant of Aggregation Optimized by Evolution. Biophysical Journal, 2007, 93, 4382-4391.	0.5	55
43	Sequence and Structural Determinants of Amyloid Fibril Formation. Accounts of Chemical Research, 2006, 39, 620-627.	15.6	102
44	Stabilization of a Native Protein Mediated by Ligand Binding Inhibits Amyloid Formation Independently of the Aggregation Pathway. Journal of Medicinal Chemistry, 2006, 49, 6057-6064.	6.4	33
45	Assessing the role of aromatic residues in the amyloid aggregation of human muscle acylphosphatase. Protein Science, 2006, 15, 862-870.	7.6	107
46	Nature and Significance of the Interactions between Amyloid Fibrils and Biological Polyelectrolytesâ€. Biochemistry, 2006, 45, 12806-12815.	2.5	128
47	Prefibrillar Amyloid Aggregates Could Be Generic Toxins in Higher Organisms. Journal of Neuroscience, 2006, 26, 8160-8167.	3.6	222
48	NMR solution structure of the acylphosphatase from Escherichia coli. Journal of Biomolecular NMR, 2006, 36, 199-204.	2.8	15
49	Glycine Residues Appear to Be Evolutionarily Conserved for Their Ability to Inhibit Aggregation. Structure, 2005, 13, 1143-1151.	3.3	74
50	Amyloid Formation from HypF-N under Conditions in which the Protein is Initially in its Native State. Journal of Molecular Biology, 2005, 347, 323-335.	4.2	74
51	Evidence for a Mechanism of Amyloid Formation Involving Molecular Reorganisation within Native-like Precursor Aggregates. Journal of Molecular Biology, 2005, 351, 910-922.	4.2	129
52	Amyloid Formation of a Protein in the Absence of Initial Unfolding and Destabilization of the Native State. Biophysical Journal, 2005, 89, 4234-4244.	0.5	67
53	Aggregation of the Acylphosphatase from Sulfolobus solfataricus. Journal of Biological Chemistry, 2004, 279, 14111-14119.	3.4	99
54	Selection of antibody fragments specific for anÎ±-helix region of acylphosphatase. Journal of Molecular Recognition, 2004, 17, 62-66.	2.1	3

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55	Rationalization of the effects of mutations on peptide andprotein aggregation rates. Nature, 2003, 424, 805-808.	27.8	1,013
56	Relative Influence of Hydrophobicity and Net Charge in the Aggregation of Two Homologous Proteinsâ€. Biochemistry, 2003, 42, 15078-15083.	2.5	115
57	Protein Aggregation and Amyloid Fibril Formation by an SH3 Domain Probed by Limited Proteolysis. Journal of Molecular Biology, 2003, 334, 129-141.	4.2	102
58	Comparison of the Folding Processes of Distantly Related Proteins. Importance of Hydrophobic Content in Folding. Journal of Molecular Biology, 2003, 330, 577-591.	4.2	47
59	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.	7.1	268
60	Inherent toxicity of aggregates implies a common mechanism for protein misfolding diseases. Nature, 2002, 416, 507-511.	27.8	2,322
61	Kinetic partitioning of protein folding and aggregation. Nature Structural Biology, 2002, 9, 137-143.	9.7	373
62	Detection of two partially structured species in the folding process of the amyloidogenic protein Î²2-microglobulin. Journal of Molecular Biology, 2001, 307, 379-391.	4.2	115
63	Reduction of the amyloidogenicity of a protein by specific binding of ligands to the native conformation. Protein Science, 2001, 10, 879-886.	7.6	62
64	Folding and Aggregation Are Selectively Influenced by the Conformational Preferences of the Î±-Helices of Muscle Acylphosphatase. Journal of Biological Chemistry, 2001, 276, 37149-37154.	3.4	45
65	Solution conditions can promote formation of either amyloid protofilaments or mature fibrils from the HypF Nâ€terminal domain. Protein Science, 2001, 10, 2541-2547.	7.6	47
66	Solution conditions can promote formation of either amyloid protofilaments or mature fibrils from the HypF N-terminal domain. Protein Science, 2001, 10, 2541-2547.	7.6	103
67	Evidence concerning rate-limiting steps in protein folding from the effects of trifluoroethanol. Nature Structural Biology, 2000, 7, 58-61.	9.7	67
68	Initial denaturing conditions influence the slow folding phase of acylphosphatase associated with proline isomerization. Protein Science, 2000, 9, 1466-1473.	7.6	5
69	Stabilisation of Î±-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.	4.2	53
70	Designing conditions for in vitro formation of amyloid protofilaments and fibrils. Proceedings of the National Academy of Sciences of the United States of America, 1999, 96, 3590-3594.	7.1	1,021
71	Development of Enzymatic Activity during Protein Folding. Journal of Biological Chemistry, 1999, 274, 20151-20158.	3.4	26
72	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

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73	Acceleration of the folding of acylphosphatase by stabilization of local secondary structure. Nature Structural Biology, 1999, 6, 380-387.	9.7	87
74	Thermodynamics and Kinetics of Folding of Common-Type Acylphosphatase:Â Comparison to the Highly Homologous Muscle Isoenzymeâ€. Biochemistry, 1999, 38, 2135-2142.	2.5	51
75	The Contribution of Acidic Residues to the Conformational Stability of Common-Type Acylphosphatase. Archives of Biochemistry and Biophysics, 1999, 363, 349-355.	3.0	6
76	Sequenceâ€specific recognition of peptide substrates by the low M_r phosphotyrosine protein phosphatase isoforms. FEBS Letters, 1998, 422, 213-217.	2.8	13
77	Expression, purification and preliminary crystal analysis of the human lowMrphosphotyrosine protein phosphatase isoform 1. FEBS Letters, 1998, 426, 52-56.	2.8	16
78	Drosophila melanogasteracylphosphatase: A common ancestor for acylphosphatase isoenzymes of vertebrate species. FEBS Letters, 1998, 433, 205-210.	2.8	11
79	Conformational Stability of Muscle Acylphosphatase:Â The Role of Temperature, Denaturant Concentration, and pHâ€. Biochemistry, 1998, 37, 1447-1455.	2.5	57
80	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.	2.5	14
81	Looking for Residues Involved in the Muscle Acylphosphatase Catalytic Mechanism and Structural Stabilization:â€‰ Role of Asn41, Thr42, and Thr46. Biochemistry, 1996, 35, 7077-7083.	2.5	48
82	C-terminal region contributes to muscle acylphosphatase three-dimensional structure stabilisation. FEBS Letters, 1996, 384, 172-176.	2.8	12
83	Properties of Cys21-mutated muscle acylphosphatases. The Protein Journal, 1996, 15, 27-34.	1.1	8
84	Expression, Purification, and Characterization of Acylphosphatase Muscular Isoenzyme as Fusion Protein with GlutathioneS-Transferase. Protein Expression and Purification, 1995, 6, 799-805.	1.3	28
85	Properties of N-terminus truncated and C-terminus mutated muscle acylphosphatases. FEBS Letters, 1995, 362, 175-179.	2.8	11
86	Crystallisation and preliminary X-ray analysis of the â€˜common-typeâ€™ acylphosphatase. FEBS Letters, 1995, 364, 243-244.	2.8	5
87	Arginine-23 is involved in the catalytic site of muscle acylphosphatase. BBA - Proteins and Proteomics, 1994, 1208, 75-80.	2.1	31
88	The crystal structure of a low-molecular-weight phosphotyrosine protein phosphatase. Nature, 1994, 370, 575-578.	27.8	224
89	Equilibrium Unfolding Studies of Horse Muscle Acylphosphatase. FEBS Journal, 1994, 225, 811-817.	0.2	20
90	Aspartic-129 is an essential residue in the catalytic mechanism of the lowMrphosphotyrosine protein phosphatase. FEBS Letters, 1994, 350, 328-332.	2.8	47

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91	Cerebral soluble ubiquitin is increased in patients with Alzheimer's disease. Neuroscience Letters, 1993, 151, 158-161.	2.1	12
92	Investigating interdomain region mutants Phe194 Leu and Phe194 Trp of yeast phosphoglycerate kinase by 1H-NMR spectroscopy. FEBS Journal, 1992, 205, 93-104.	0.2	8
93	Preparation and properties of desâ€Tyr⁹⁸ and desâ€Arg⁹⁷â€Tyr⁹⁸ acylphosphatase (muscular isoenzyme). International Journal of Peptide and Protein Research, 1991, 38, 278-284.	0.1	3
94	Increased Acylphosphatase Levels in Erythrocytes, Muscle and Liver of Tri-Iodothyronine Treated Rabbits. Hormone and Metabolic Research, 1990, 22, 33-37.	1.5	9
95	Changes in Na+,K+-ATPase, Ca2+-ATPase and some soluble enzymes related to energy metabolism in brains of patients with Alzheimer's disease. Neuroscience Letters, 1990, 112, 338-342.	2.1	86
96	Isolation and quantitation of ubiquitin from rat brain. Protein Expression and Purification, 1990, 1, 93-96.	1.3	1
97	Effect of acylphosphatase on human erythrocyte membrane Ca2+-ATPase. Biochemical and Biophysical Research Communications, 1990, 168, 651-658.	2.1	10
98	Increased acylphosphatase levels in erythrocytes from hyperthyroid patients. Clinica Chimica Acta, 1989, 183, 351-358.	1.1	6
99	Post-mortem modifications of the specific activity of some brain enzymes. Neuroscience Letters, 1988, 85, 244-248.	2.1	13