Gary E Gibson

List of Publications by Year
in descending order

191	11,708	27035 58	38517 99
papers	citations	h-index	g-index

198	198	198	10762
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Altered succinylation of mitochondrial proteins, APP and tau in Alzheimerâ€™s disease. Nature Communications, 2022, 13, 159.	5.8	42
2	Selective linkage of mitochondrial enzymes to intracellular calcium stores differs between humanâ€induced pluripotent stem cells, neural stem cells, and neurons. Journal of Neurochemistry, 2021, 156, 867-879.	2.1	2
3	Regulation | Post Translational Modifications by Succinylation and Acetylation. , 2021, , 631-640.		0
4	The human brain acetylome reveals that decreased acetylation of mitochondrial proteins associates with Alzheimer's disease. Journal of Neurochemistry, 2021, 158, 282-296.	2.1	11
5	Serum Metabolomic and Lipidomic Profiling Reveals Novel Biomarkers of Efficacy for Benfotiamine in Alzheimerâ€™s Disease. International Journal of Molecular Sciences, 2021, 22, 13188.	1.8	13
6	Benfotiamine and Cognitive Decline in Alzheimerâ€™s Disease: Results of a Randomized Placebo-Controlled Phase IIa Clinical Trial. Journal of Alzheimer's Disease, 2020, 78, 989-1010.	1.2	52
7	Selective NADH communication from Î±-ketoglutarate dehydrogenase to mitochondrial transhydrogenase prevents reactive oxygen species formation under reducing conditions in the heart. Basic Research in Cardiology, 2020, 115, 53.	2.5	28
8	Succinylation Links Metabolism to Protein Functions. Neurochemical Research, 2019, 44, 2346-2359.	1.6	102
9	Mitochondria/metabolic reprogramming in the formation of neurons from peripheral cells: Cause or consequence and the implications to their utility. Neurochemistry International, 2018, 117, 65-76.	1.9	5
10	Benfotiamine treatment activates the Nrf2/ARE pathway and is neuroprotective in a transgenic mouse model of tauopathy. Human Molecular Genetics, 2018, 27, 2874-2892.	1.4	58
11	Interactions of Mitochondria/Metabolism and Calcium Regulation in Alzheimerâ€™s Disease: A Calcinist Point of View. Neurochemical Research, 2017, 42, 1636-1648.	1.6	29
12	Mild metabolic perturbations alter succinylation of mitochondrial proteins. Journal of Neuroscience Research, 2017, 95, 2244-2252.	1.3	32
13	[P2â€“019]: GLUCOSE METABOLISM AS A THERAPEUTIC TARGET IN ALZHEIMER's DISEASE. Alzheimer's and Dementia, 2017, 13, P611.	0.4	1
14	Reductions in the mitochondrial enzyme Î±â€ketoglutarate dehydrogenase complex in neurodegenerative disease â€“ beneficial or detrimental?. Journal of Neurochemistry, 2016, 139, 823-838.	2.1	26
15	Vitamin B1 (thiamine) and dementia. Annals of the New York Academy of Sciences, 2016, 1367, 21-30.	1.8	150
16	Mild mitochondrial metabolic deficits by Î±-ketoglutarate dehydrogenase inhibition cause prominent changes in intracellular autophagic signaling: Potential role in the pathobiology of Alzheimer's disease. Neurochemistry International, 2016, 96, 32-45.	1.9	27
17	Novel Metabolic Abnormalities in the Tricarboxylic Acid Cycle in Peripheral Cells From Huntingtonâ€™s Disease Patients. PLoS ONE, 2016, 11, e0160384.	1.1	21
18	Abnormalities in the Tricarboxylic Acid Cycle in Huntington Disease and in a Huntington Disease Mouse Model. Journal of Neuropathology and Experimental Neurology, 2015, 74, 527-537.	0.9	36

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19	Impaired mitochondrial energy metabolism as a novel risk factor for selective onset and progression of dementia in oldest-old subjects. Neuropsychiatric Disease and Treatment, 2015, 11, 565.	1.0	13
20	The RNA-binding protein HuR is essential for the B cell antibody response. Nature Immunology, 2015, 16, 415-425.	7.0	125
21	Abnormal Glucose Metabolism in Alzheimerâ€™s Disease: Relation to Autophagy/Mitophagy and Therapeutic Approaches. Neurochemical Research, 2015, 40, 2557-2569.	1.6	30
22	Alphaâ€ketoglutarate dehydrogenase complexâ€dependent succinylation of proteins in neurons and neuronal cell lines. Journal of Neurochemistry, 2015, 134, 86-96.	2.1	96
23	Interactions of endoplasmic reticulum and mitochondria Ca2+ stores with capacitative calcium entry. Metabolic Brain Disease, 2014, 29, 1083-1093.	1.4	8
24	F5-02-01: Can abnormal glucose metabolism lead to the Alzheimer's disease pathologies?. , 2013, 9, P825-P825.		0
25	Abnormal thiamine-dependent processes in Alzheimer's Disease. Lessons from diabetes. Molecular and Cellular Neurosciences, 2013, 55, 17-25.	1.0	69
26	The negative impact ofÎ±â€ketoglutarate dehydrogenase complex deficiency on matrix substrateâ€level phosphorylation. FASEB Journal, 2013, 27, 2392-2406.	0.2	57
27	Deficits in the mitochondrial enzyme Î±-ketoglutarate dehydrogenase lead to Alzheimer's disease-like calcium dysregulation. Neurobiology of Aging, 2012, 33, 1121.e13-1121.e24.	1.5	49
28	_-Ketoglutarate Dehydrogenase Complex in Neurodegeneration. Oxidative Stress and Disease, 2012, , 433-454.	0.3	0
29	Abnormalities in the tricarboxylic acid (TCA) cycle in the brains of schizophrenia patients. European Neuropsychopharmacology, 2011, 21, 254-260.	0.3	75
30	Upâ€regulation of the mitochondrial malate dehydrogenase by oxidative stress is mediated by miRâ€743a. Journal of Neurochemistry, 2011, 118, 440-448.	2.1	68
31	Brain [Uâ€¹³C]glucose metabolism in mice with decreased Î±â€ketoglutarate dehydrogenase complex activity. Journal of Neuroscience Research, 2011, 89, 1997-2007.	1.3	18
32	Inactivation and Reactivation of the Mitochondrial Î±-Ketoglutarate Dehydrogenase Complex. Journal of Biological Chemistry, 2011, 286, 17640-17648.	1.6	61
33	A Mitocentric View of Alzheimer's Disease Suggests Multi-Faceted Treatments. Journal of Alzheimer's Disease, 2010, 20, S591-S607.	1.2	100
34	Thiamine and Oxidants Interact to Modify Cellular Calcium Stores. Neurochemical Research, 2010, 35, 2107-2116.	1.6	24
35	Cause and consequence: Mitochondrial dysfunction initiates and propagates neuronal dysfunction, neuronal death and behavioral abnormalities in age-associated neurodegenerative diseases. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2010, 1802, 122-134.	1.8	203
36	Mice deficient in dihydrolipoyl succinyl transferase show increased vulnerability to mitochondrial toxins. Neurobiology of Disease, 2009, 36, 320-330.	2.1	24

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37	Mitochondrial dihydrolipoyl succinyltransferase deficiency accelerates amyloid pathology and memory deficit in a transgenic mouse model of amyloid deposition. Free Radical Biology and Medicine, 2009, 47, 1019-1027.	1.3	58
38	Mild reduction in the activity of the Î±â€ketoglutarate dehydrogenase complex elevates GABA shunt and glycolysis. Journal of Neurochemistry, 2009, 109, 214-221.	2.1	46
39	Thiamine deficiency induces oxidative stress and exacerbates the plaque pathology in Alzheimer's mouse model. Neurobiology of Aging, 2009, 30, 1587-1600.	1.5	123
40	Dietary supplementation with resveratrol reduces plaque pathology in a transgenic model of Alzheimer's disease. Neurochemistry International, 2009, 54, 111-118.	1.9	438
41	Presenilins Are Enriched in Endoplasmic Reticulum Membranes Associated with Mitochondria. American Journal of Pathology, 2009, 175, 1810-1816.	1.9	328
42	Thiamine Deficiency: A Model of Metabolic Encephalopathy and of Selective Neuronal Vulnerability. , 2009, , 235-260.		3
43	Translocation of Amyloid Precursor Protein C-terminal Fragment(s) to the Nucleus Precedes Neuronal Death due to Thiamine Deficiency-induced Mild Impairment of Oxidative Metabolism. Neurochemical Research, 2008, 33, 1365-1372.	1.6	16
44	Metabolic Impairment Induces Oxidative Stress, Compromises Inflammatory Responses, and Inactivates a Key Mitochondrial Enzyme in Microglia. Journal of Neurochemistry, 2008, 72, 1948-1958.	2.1	124
45	Oxidantâ€induced Changes in Mitochondria and Calcium Dynamics in the Pathophysiology of Alzheimer's Disease. Annals of the New York Academy of Sciences, 2008, 1147, 221-232.	1.8	42
46	Preface. Annals of the New York Academy of Sciences, 2008, 1147, xi-xii.	1.8	8
47	Novel functions of the Î±-ketoglutarate dehydrogenase complex may mediate diverse oxidant-induced changes in mitochondrial enzymes associated with Alzheimer's disease. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2008, 1782, 229-238.	1.8	55
48	Thiamine-Dependent Processes and Treatment Strategies in Neurodegeneration. Antioxidants and Redox Signaling, 2007, 9, 1605-1620.	2.5	91
49	Oxidative Stress and Transcriptional Regulation in Alzheimer Disease. Alzheimer Disease and Associated Disorders, 2007, 21, 276-291.	0.6	136
50	Responses of the mitochondrial alpha-ketoglutarate dehydrogenase complex to thiamine deficiency may contribute to regional selective vulnerability. Neurochemistry International, 2007, 50, 921-931.	1.9	39
51	Coordination and timing of spine and hip joints during full body reaching tasks. Human Movement Science, 2007, 26, 124-140.	0.6	37
52	Changes in inflammatory processes associated with selective vulnerability following mild impairment of oxidative metabolism. Neurobiology of Disease, 2007, 26, 353-362.	2.1	58
53	Enzyme-Catalyzed Side Reactions with Molecular Oxygen may Contribute to Cell Signaling and Neurodegenerative Diseases. Neurochemical Research, 2007, 32, 871-891.	1.6	44
54	Foreword. Neurochemical Research, 2007, 32, 533-534.	1.6	0

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55	Cisplatin-Induced Toxicity Is Associated with Platinum Deposition in Mouse Kidney Mitochondria in Vivo and with Selective Inactivation of the Î±-Ketoglutarate Dehydrogenase Complex in LLC-PK1 Cells. Biochemistry, 2006, 45, 8959-8971.	1.2	42
56	Phospholipid mass is increased in fibroblasts bearing the Swedish amyloid precursor mutation. Brain Research Bulletin, 2006, 69, 79-85.	1.4	4
57	Peripheral inflammatory mechanisms modulate microglial activation in response to mild impairment of oxidative metabolism. Neurochemistry International, 2006, 49, 548-556.	1.9	25
58	Correlations of disability and biologic alterations in Alzheimer brain and test of significance by a therapeutic trial in humans. Journal of Alzheimer's Disease, 2006, 9, 207-218.	1.2	16
59	Cellular mitochondrial heterogeneity in cultured astrocytes as demonstrated by immunogold labeling of Î±-ketoglutarate dehydrogenase. Glia, 2006, 53, 225-231.	2.5	47
60	Inhibitors of the Î±-ketoglutarate dehydrogenase complex alter [1-13C]glucose and [U-13C]glutamate metabolism in cerebellar granule neurons. Journal of Neuroscience Research, 2006, 83, 450-458.	1.3	50
61	The Î±-Ketoglutarateâ€“Dehydrogenase Complex: A Mediator Between Mitochondria and Oxidative Stress in Neurodegeneration. Molecular Neurobiology, 2005, 31, 043-064.	1.9	154
62	Inhibition of the alpha-ketoglutarate dehydrogenase complex by the myeloperoxidase products, hypochlorous acid and mono-N-chloramine. Journal of Neurochemistry, 2005, 92, 302-310.	2.1	51
63	Modification of endoplasmic reticulum Ca2+ stores by select oxidants produces changes reminiscent of those in cells from patients with Alzheimer disease. Free Radical Biology and Medicine, 2005, 39, 979-989.	1.3	18
64	CD40L deletion delays neuronal death in a model of neurodegeneration due to mild impairment of oxidative metabolism. Journal of Neuroimmunology, 2005, 164, 85-92.	1.1	16
65	Mitochondrial abnormalities in Alzheimer brain: Mechanistic implications. Annals of Neurology, 2005, 57, 695-703.	2.8	519
66	Mitochondrial Aconitase is a Transglutaminase 2 Substrate: Transglutamination is a Probable Mechanism Contributing to High-Molecular-Weight Aggregates of Aconitase and Loss of Aconitase Activity in Huntington Disease Brain. Neurochemical Research, 2005, 30, 1245-1255.	1.6	46
67	Reduction in the E2k Subunit of the Î±-Ketoglutarate Dehydrogenase Complex Has Effects Independent of Complex Activity. Journal of Biological Chemistry, 2005, 280, 10888-10896.	1.6	36
68	Phosphonate Analogues of Î±-Ketoglutarate Inhibit the Activity of the Î±-Ketoglutarate Dehydrogenase Complex Isolated from Brain and in Cultured Cellsâ€. Biochemistry, 2005, 44, 10552-10561.	1.2	80
69	Transglutaminase Activity Is Present in Highly Purified Nonsynaptosomal Mouse Brain and Liver Mitochondriaâ€. Biochemistry, 2005, 44, 7830-7843.	1.2	52
70	CD40â€“CD40L interactions promote neuronal death in a model of neurodegeneration due to mild impairment of oxidative metabolism. Neurochemistry International, 2005, 47, 204-215.	1.9	29
71	Mitochondrial function in fibroblasts with aging in culture and/or Alzheimer's disease. Neurobiology of Aging, 2005, 26, 839-848.	1.5	32
72	Oxidative stress in Alzheimer's disease. Neurobiology of Aging, 2005, 26, 575-578.	1.5	100

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73	Î±-KETO-ÃŸ-METHYL-n-VALERIC ACID DIMINISHES REACTIVE OXYGEN SPECIES AND ALTERS ENDOPLASMIC RETICULUM Ca2+ STORES. Free Radical Biology and Medicine, 2004, 37, 1779-1789.	1.3	25
74	Mitochondrial Enzymes in Schizophrenia. Journal of Molecular Neuroscience, 2004, 24, 315-322.	1.1	45
75	Mitochondrial Enzymes and Endoplasmic Reticulum Calcium Stores as Targets of Oxidative Stress in Neurodegenerative Diseases. Journal of Bioenergetics and Biomembranes, 2004, 36, 335-340.	1.0	53
76	Mitochondrial Heterogeneity Within and Between Different Cell Types. Neurochemical Research, 2004, 29, 651-658.	1.6	21
77	Mice deficient in dihydrolipoamide dehydrogenase show increased vulnerability to MPTP, malonate and 3â€nitropropionic acid neurotoxicity. Journal of Neurochemistry, 2004, 88, 1352-1360.	2.1	92
78	Selective response of various brain cell types during neurodegeneration induced by mild impairment of oxidative metabolism. Neurochemistry International, 2004, 45, 361-369.	1.9	76
79	Tricarboxylic acid cycle enzymes following thiamine deficiency. Neurochemistry International, 2004, 45, 1021-1028.	1.9	69
80	Inhibition of ?-ketoglutarate dehydrogenase complex promotes cytochromec release from mitochondria, caspase-3 activation, and necrotic cell death. Journal of Neuroscience Research, 2003, 74, 309-317.	1.3	59
81	Deficits in a tricarboxylic acid cycle enzyme in brains from patients with Parkinsonâ€™s disease. Neurochemistry International, 2003, 43, 129-135.	1.9	96
82	Inhibition of the Î±-ketoglutarate dehydrogenase complex alters mitochondrial function and cellular calcium regulation. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2003, 1637, 119-126.	1.8	53
83	Reversal of Thiamine Deficiency-Induced Neurodegeneration. Journal of Neuropathology and Experimental Neurology, 2003, 62, 195-207.	0.9	88
84	The role of the metabolic lesion in Alzheimer's disease. Journal of Alzheimer's Disease, 2002, 4, 225-232.	1.2	84
85	Oxidative stress increases internal calcium stores and reduces a key mitochondrial enzyme. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2002, 1586, 177-189.	1.8	45
86	Interactions of oxidative stress with thiamine homeostasis promote neurodegeneration. Neurochemistry International, 2002, 40, 493-504.	1.9	122
87	Oxidative processes in the brain and non-neuronal tissues as biomarkers of Alzheimer s disease. Frontiers in Bioscience - Landmark, 2002, 7, d1007-1015.	3.0	28
88	Interactions of oxidative stress with cellular calcium dynamics and glucose metabolism in Alzheimerâ€™s disease 1,2 1Guest Editors: Mark A. Smith and George Perry 2This article is a part of a series of reviews on â€œCauses and Consequences of Oxidative Stress in Alzheimerâ€™s Disease.â€•The full list of papers may be found on the homepage of the journal.. Free Radical Biology and Medicine, 2002, 32, 1061-1070.	1.3	60
89	Heterogeneous Expression of Transketolase in Rat Brain. Journal of Neurochemistry, 2002, 64, 1034-1044.	2.1	15
90	Regional Reductions of Transketolase in Thiamine-Deficient Rat Brain. Journal of Neurochemistry, 2002, 67, 684-691.	2.1	19

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91	Selective Changes in Cell Bodies and Growth Cones of Nerve Growth Factor-Differentiated PC12 Cells Induced by Chemical Hypoxia. Journal of Neurochemistry, 2002, 69, 603-611.	2.1	9
92	Immunochemical Characterization of the Deficiency of the Î±-Ketoglutarate Dehydrogenase Complex in Thiamine-Deficient Rat Brain. Journal of Neurochemistry, 2002, 70, 1143-1150.	2.1	40
93	Abnormalities in oxidative processes in non-neuronal tissues from patients with Alzheimer's disease*. Journal of Alzheimer's Disease, 2001, 3, 329-338.	1.2	36
94	Frontal Lobe Dysfunction in Progressive Supranuclear Palsy. Journal of Neurochemistry, 2001, 74, 878-881.	2.1	95
95	Dopaminergic cell death induced by MPP+, oxidant and specific neurotoxicants shares the common molecular mechanism. Journal of Neurochemistry, 2001, 76, 1010-1021.	2.1	238
96	Metabolic Impairment Elicits Brain Cell Type-Selective Changes in Oxidative Stress and Cell Death in Culture. Journal of Neurochemistry, 2001, 74, 114-124.	2.1	26
97	Mitochondrial impairment in the cerebellum of the patients with progressive supranuclear palsy. Journal of Neuroscience Research, 2001, 66, 1028-1034.	1.3	75
98	Dichloroacetate exerts therapeutic effects in transgenic mouse models of Huntington's disease. Annals of Neurology, 2001, 50, 112-116.	2.8	79
99	Co-culture with astrocytes or microglia protects metabolically impaired neurons. Mechanisms of Ageing and Development, 2001, 123, 21-27.	2.2	47
100	Quantitative Î±-Ketoglutarate Dehydrogenase Activity Staining in Brain Sections and in Cultured Cells. Analytical Biochemistry, 2000, 277, 86-93.	1.1	29
101	Inherent Abnormalities in Energy Metabolism in Alzheimer Disease: Interaction with Cerebrovascular Compromise. Annals of the New York Academy of Sciences, 2000, 903, 204-221.	1.8	182
102	Vascular Endothelium Is a Site of Free Radical Production and Inflammation in Areas of Neuronal Loss in Thiamine-deficient Brain. Annals of the New York Academy of Sciences, 2000, 903, 353-356.	1.8	42
103	Dietary restriction attenuates the neuronal loss, induction of heme oxygenase-1 and bloodâ€“brain barrier breakdown induced by impaired oxidative metabolism. Brain Research, 2000, 885, 62-69.	1.1	45
104	Vascular Factors Are Critical in Selective Neuronal Loss in an Animal Model of Impaired Oxidative Metabolism. Journal of Neuropathology and Experimental Neurology, 2000, 59, 207-217.	0.9	43
105	Phospholipid composition and levels are not altered in fibroblasts bearing presenilin-1 mutations. Brain Research Bulletin, 2000, 52, 207-212.	1.4	3
106	The Î±-ketoglutarate dehydrogenase complex in neurodegeneration. Neurochemistry International, 2000, 36, 97-112.	1.9	185
107	Protein-Bound Acrolein. Journal of Neurochemistry, 1999, 72, 751-756.	2.1	358
108	Oxidative Stress and a Key Metabolic Enzyme in Alzheimer Brains, Cultured Cells, and an Animal Model of Chronic Oxidative Deficits. Annals of the New York Academy of Sciences, 1999, 893, 79-94.	1.8	82

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109	Inhibition of select mitochondrial enzymes in PC12 cells exposed to S-(1,1,2,2-tetrafluoroethyl)-l-cysteine. Biochemical Pharmacology, 1999, 58, 1557-1565.	2.0	21
110	Cultures of astrocytes and microglia express interleukin 18. Molecular Brain Research, 1999, 67, 46-52.	2.5	166
111	Cerebrometabolic Aspects of Delirium in Relationship to Dementia. Dementia and Geriatric Cognitive Disorders, 1999, 10, 335-338.	0.7	58
112	Oxidative Stress Is Associated with Region-Specific Neuronal Death During Thiamine Deficiency. Journal of Neuropathology and Experimental Neurology, 1999, 58, 946-958.	0.9	120
113	Induction of Nitric Oxide Synthase and Microglial Responses Precede Selective Cell Death Induced by Chronic Impairment of Oxidative Metabolism. American Journal of Pathology, 1998, 153, 599-610.	1.9	85
114	Disturbances of the Blood-Brain Barrier without Expression of Amyloid Precursor Protein-Containing Neuritic Clusters or Neuronal Loss during Late Stages of Thiamine Deficiency in Guinea Pigs. Developmental Neuroscience, 1998, 20, 454-461.	1.0	5
115	Thiamine deficiency alters APP but not presenilin-1 immunoreactivity in vulnerable brain regions. NeuroReport, 1997, 8, 2631-2634.	0.6	10
116	Differential regulation of adenylyl cyclase in fibroblasts from sporadic and familial AlzheimerÊ¼s disease cases with PS1 and APP mutations. NeuroReport, 1997, 8, 2031-2035.	0.6	14
117	Abnormalities in Alzheimerâ€™s Disease Fibroblasts Bearing the APP670/671 Mutation. Neurobiology of Aging, 1997, 18, 573-580.	1.5	48
118	G protein subunit levels in fibroblasts from familial Alzheimer's disease patients: lower levels of the high molecular weight GsÎ± isoform in patients with decreased Î²-adrenergic receptor stimulated cAMP formation. Neuroscience Letters, 1997, 232, 33-36.	1.0	12
119	Calcium stores in cultured fibroblasts and their changes with Alzheimer's disease. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 1996, 1316, 71-77.	1.8	57
120	Altered oxidation and signal transduction systems in fibroblasts from Alzheimer patients. Life Sciences, 1996, 59, 477-489.	2.0	59
121	Regulation of bradykinin-induced Ins(1,4,5)P3 formation by protein kinase C in human fibroblasts. Life Sciences, 1996, 59, 1533-1543.	2.0	5
122	Accumulation of amyloid precursor protein-like immunoreactivity in rat brain in response to thiamine deficiency. Brain Research, 1995, 677, 50-60.	1.1	57
123	Mitochondria, Aging, and Neurological Diseases. , 1995, , 95-107.		3
124	The role of signal transduction systems in mediating cell density dependent changes in tyrosine hydroxylase gene expression. Molecular Brain Research, 1995, 33, 254-260.	2.5	12
125	Blood-Brain Barrier Abnormalities in Vulnerable Brain Regions during Thiamine Deficiency. Experimental Neurology, 1995, 134, 64-72.	2.0	67
126	Distribution of the ?-ketoglutarate dehydrogenase complex in rat brain. Journal of Comparative Neurology, 1994, 346, 461-479.	0.9	46

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127	Selective enrichment of cholinergic neurons with the Î±-ketoglutarate dehydrogenase complex in rat brain. Neuroscience Letters, 1994, 168, 209-212.	1.0	14
128	Aging and the brain. Current Opinion in Neurology, 1994, 7, 287-293.	1.8	43
129	Use of Cultured Fibroblasts in Elucidating the Pathophysiology and Diagnosis of Alzheimer's Disease^a. Annals of the New York Academy of Sciences, 1994, 747, 225-244.	1.8	41
130	Molecular Mechanisms of Memory and the Pathophysiology of Alzheimer's Disease. Annals of the New York Academy of Sciences, 1994, 747, 245-255.	1.8	21
131	Synaptosomal plasma and mitochondrial membrane potentials during anoxia. Neuroscience Letters, 1992, 138, 133-136.	1.0	4
132	Cytosolic free calcium in lymphoblasts from young, aged and alzheimer subjects. Mechanisms of Ageing and Development, 1992, 63, 1-9.	2.2	21
133	Cytosolic Free Calcium and Gene Expression During Chemical Hypoxia. Journal of Neurochemistry, 1992, 59, 1836-1843.	2.1	25
134	Cytosolic free calcium and ATP in synaptosomes after ischemia. Life Sciences, 1991, 48, 1439-1445.	2.0	16
135	The Cellular Basis of Delirium and Its Relevance to Age-Related Disorders Including Alzheimer's Disease. International Psychogeriatrics, 1991, 3, 373-395.	0.6	68
136	Cytosolic free calcium concentrations in synaptosomes during histotoxic hypoxia. Neurochemical Research, 1991, 16, 461-467.	1.6	22
137	Acetylcholine formation from glucose following acute choline supplementation. Neurochemical Research, 1991, 16, 1009-1015.	1.6	2
138	Dopamine and serotonin in rat striatum duringin vivo hypoxic-hypoxia. Metabolic Brain Disease, 1989, 4, 143-153.	1.4	46
139	Cytosolic-free calcium and neurotransmitter release with decreased availability of glucose or oxygen. Neurochemical Research, 1989, 14, 437-443.	1.6	54
140	Regionally selective alterations in enzymatic activities and metabolic fluxes during thiamin deficiency. Neurochemical Research, 1989, 14, 17-24.	1.6	33
141	Phosphatidylinositol Metabolism During In Vitro Hypoxia. Journal of Neurochemistry, 1989, 52, 830-835.	2.1	24
142	Causes of cell damage in hypoxia/ischemia, aging and Alzheimer's disease. Neurobiology of Aging, 1989, 10, 608-609.	1.5	11
143	Effects of in vitro hypoxia on depolarization-stimulated accumulation of inositol phosphates in synaptosomes. Life Sciences, 1989, 45, 1443-1449.	2.0	5
144	An in vitro model of anoxic-induced damage in mouse brain. Neurochemical Research, 1988, 13, 9-20.	1.6	14

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145	Selective damage in striatum and hippocampus with in vitro anoxia. Neurochemical Research, 1988, 13, 329-335.	1.6	11
146	Changes in cytosolic free calcium with 1,2,3,4-tetrahydro-5-aminoacridine, 4-aminopyridine and 3,4-diaminopyridine. Biochemical Pharmacology, 1988, 37, 4191-4196.	2.0	25
147	Dopamine, Acetylcholine, and Glutamate Interactions in Aging Behavioral and Neurochemical Correlates. Annals of the New York Academy of Sciences, 1988, 515, 191-202.	1.8	30
148	Selective alteration of mouse brain neurotransmitter release with age. Neurobiology of Aging, 1987, 8, 147-152.	1.5	77
149	Authors' response to commentaries. Neurobiology of Aging, 1987, 8, 372-375.	1.5	0
150	Diminished mitogen-induced calcium uptake by lymphocytes from alzheimer patients. Biological Psychiatry, 1987, 22, 1079-1086.	0.7	61
151	Calcium and the aging nervous system. Neurobiology of Aging, 1987, 8, 329-343.	1.5	378
152	Effect of age on behavioral and enzymatic changes during thiamin deficiency. Neurobiology of Aging, 1987, 8, 429-434.	1.5	23
153	Differential alteration of dopamine, acetylcholine, and glutamate release during anoxia and/or 3,4-diaminopyridine treatment. Neurochemical Research, 1987, 12, 1019-1027.	1.6	36
154	Locomotor activity as a predictor of times and dosages for studies of nicotine's neurochemical actions. Pharmacology Biochemistry and Behavior, 1987, 26, 305-312.	1.3	24
155	Human red blood cell choline uptake with age and Alzheimer's disease. Neurobiology of Aging, 1986, 7, 205-209.	1.5	21
156	In vivo brain calcium homeostasis during aging. Mechanisms of Ageing and Development, 1986, 37, 1-12.	2.2	24
157	Automated method to estimate catecholamine and indoleamine content and turnover rates. Biomedical Applications, 1986, 374, 239-249.	1.7	6
158	Monoamine Neurotransmitter Metabolism and Locomotor Activity During Chemical Hypoxia. Journal of Neurochemistry, 1986, 46, 733-738.	2.1	28
159	Effect of Decreased Oxygen on In Vitro Release of Endogenous 3,4-Dihydroxyphenylethylamine from Mouse Striatum. Journal of Neurochemistry, 1986, 47, 1924-1931.	2.1	23
160	Behavioral and neurochemical correlates of morphine and hypoxia interactions. Pharmacology Biochemistry and Behavior, 1986, 24, 1687-1693.	1.3	3
161	Oxidative metabolism and acetylcholine synthesis during acetylpyridine treatment. Neurochemical Research, 1985, 10, 453-467.	1.6	11
162	Subsynaptosomal Calcium Distribution During Hypoxia and 3,4-Diaminopyridine Treatment. Journal of Neurochemistry, 1985, 45, 1779-1790.	2.1	27

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163	Oral health in a long-term care institution equipped with a dental service. Community Dentistry and Oral Epidemiology, 1985, 13, 260-263.	0.9	58
164	Subsynaptosomal distribution of calcium during aging and 3,4-diaminopyridine treatment. Neurobiology of Aging, 1985, 6, 297-304.	1.5	52
165	Synaptosomal Calcium Metabolism During Hypoxia and 3,4-Diaminopyridine Treatment. Journal of Neurochemistry, 1984, 42, 248-253.	2.1	43
166	Selective alteration of neurotransmitter release by low oxygen in vitro. Neurochemical Research, 1984, 9, 1039-1049.	1.6	61
167	Correlation of enzymatic, metabolic, and behavioral deficits in thiamin deficiency and its reversal. Neurochemical Research, 1984, 9, 803-814.	1.6	136
168	Thiamin antagonists and the release of acetylcholine and norepinephrine from brain slices. Biochemical Pharmacology, 1984, 33, 2325-2327.	2.0	11
169	The pyruvate dehydrogenase complex during aging. Mechanisms of Ageing and Development, 1984, 26, 67-73.	2.2	11
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171	Amelioration of age-related neurochemical and behavioral deficits by 3,4-diaminopyridine. Neurobiology of Aging, 1983, 4, 25-30.	1.5	58
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