Robert E Burke

List of Publications by Year
in descending order

141 papers	10,973 citations	39113 52 h-index	37326 100 g-index
146 all docs	146 docs citations	146 times ranked	10535 citing authors

#	Article	IF	Citations
1	The drug adaptaquin blocks ATF4/CHOP-dependent pro-death Trib3 induction and protects in cellular and mouse models of Parkinson's disease. Neurobiology of Disease, 2020, 136, 104725.	2.1	37
2	Disease Modification for Parkinson's Disease: Axonal Regeneration and Trophic Factors. Movement Disorders, 2018, 33, 678-683.	2.2	24
3	Guanabenz promotes neuronal survival via enhancement of ATF4 and parkin expression in models of Parkinson disease. Experimental Neurology, 2018, 303, 95-107.	2.0	26
4	Protection of nigral dopaminergic neurons by AAV1 transduction with Rheb(S16H) against neurotoxic inflammation in vivo. Experimental and Molecular Medicine, 2018, 50, e440-e440.	3.2	14
5	Induction of axon growth in the adult brain: A new approach to restoration in Parkinson's disease. Movement Disorders, 2018, 33, 62-70.	2.2	9
6	Context-dependent expression of a conditionally-inducible form of active Akt. PLoS ONE, 2018, 13, e0197899.	1.1	3
7	A quantitative evaluation of a 2.5-kb rat tyrosine hydroxylase promoter to target expression in ventral mesencephalic dopamine neurons in vivo. Neuroscience, 2017, 346, 126-134.	1.1	11
8	Combining Constitutively Active Rheb Expression and Chondroitinase Promotes Functional Axonal Regeneration after Cervical Spinal Cord Injury. Molecular Therapy, 2017, 25, 2715-2726.	3.7	26
9	Autophagy linked FYVE (Alfy/WDFY3) is required for establishing neuronal connectivity in the mammalian brain. ELife, 2016, 5, .	2.8	78
10	Expressing Constitutively Active Rheb in Adult Dorsal Root Ganglion Neurons Enhances the Integration of Sensory Axons that Regenerate Across a Chondroitinase-Treated Dorsal Root Entry Zone Following Dorsal Root Crush. Frontiers in Molecular Neuroscience, 2016, 9, 49.	1.4	19
11	Expression mediated by three partial sequences of the human tyrosine hydroxylase promoter in vivo. Molecular Therapy - Methods and Clinical Development, 2016, 3, 16062.	1.8	1
12	Is Axonal Degeneration a Key Early Event in Parkinsonâ€™s Disease?. Journal of Parkinson's Disease, 2016, 6, 703-707.	1.5	36
13	Retrograde Axonal Degeneration in Parkinson Disease. Journal of Parkinson's Disease, 2016, 6, 1-15.	1.5	183
14	In Vivo AAV1 Transduction With hRheb(S16H) Protects Hippocampal Neurons by BDNF Production. Molecular Therapy, 2015, 23, 445-455.	3.7	34
15	An early axonopathy in a hLRRK2(R1441G) transgenic model of Parkinson disease. Neurobiology of Disease, 2015, 82, 359-371.	2.1	40
16	Expressing Constitutively Active Rheb in Adult Neurons after a Complete Spinal Cord Injury Enhances Axonal Regeneration beyond a Chondroitinase-Treated Glial Scar. Journal of Neuroscience, 2015, 35, 11068-11080.	1.7	54
17	Induction of GDNF and BDNF by hRheb(S16H) Transduction of SNpc Neurons: Neuroprotective Mechanisms of hRheb(S16H) in a Model of Parkinsonâ€™s Disease. Molecular Neurobiology, 2015, 51, 487-499.	1.9	63
18	Rheb GTPase Regulates Î²-Secretase Levels and Amyloid Î² Generation. Journal of Biological Chemistry, 2014, 289, 5799-5808.	1.6	49

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19	Quantitative morphological comparison of axon-targeting strategies for gene therapies directed to the nigro-striatal projection. Gene Therapy, 2014, 21, 115-122.	2.3	4
20	Axon degeneration in Parkinson's disease. Experimental Neurology, 2013, 246, 72-83.	2.0	367
21	AAV Transduction of Dopamine Neurons With Constitutively Active Rheb Protects From Neurodegeneration and Mediates Axon Regrowth. Molecular Therapy, 2012, 20, 275-286.	3.7	94
22	Neurotrophic Effects of Serum- and Glucocorticoid-Inducible Kinase on Adult Murine Mesencephalic Dopamine Neurons. Journal of Neuroscience, 2012, 32, 11299-11308.	1.7	13
23	Regulation of Presynaptic Neurotransmission by Macroautophagy. Neuron, 2012, 74, 277-284.	3.8	286
24	Glial cell line-derived neurotrophic factor receptor-alpha 1 expressed in striatum in trans regulates development and injury response of dopamine neurons of the substantia nigra. Journal of Neurochemistry, 2011, 116, 486-498.	2.1	16
25	Age and Î±-synuclein expression interact to reveal a dependence of dopaminergic axons on endogenous Akt/PKB signaling. Neurobiology of Disease, 2011, 44, 215-222.	2.1	22
26	Dopaminergic pathway reconstruction by Akt/Rhebâ€induced axon regeneration. Annals of Neurology, 2011, 70, 110-120.	2.8	121
27	Akt Suppresses Retrograde Degeneration of Dopaminergic Axons by Inhibition of Macroautophagy. Journal of Neuroscience, 2011, 31, 2125-2135.	1.7	126
28	Clinical progression in Parkinson disease and the neurobiology of axons. Annals of Neurology, 2010, 67, 715-725.	2.8	778
29	Evaluation of the Braak staging scheme for Parkinson's disease: Introduction to a panel presentation. Movement Disorders, 2010, 25, S76-7.	2.2	14
30	The Wld^S mutation delays anterograde, but not retrograde, axonal degeneration of the dopaminergic nigroâ€striatal pathway <i>in vivo</i>. Journal of Neurochemistry, 2010, 113, 683-691.	2.1	31
31	Intracellular signalling pathways in dopamine cell death and axonal degeneration. Progress in Brain Research, 2010, 183, 79-97.	0.9	19
32	Mutant LRRK2R1441G BAC transgenic mice recapitulate cardinal features of Parkinson's disease. Nature Neuroscience, 2009, 12, 826-828.	7.1	475
33	Regulation of the postnatal development of dopamine neurons of the substantia nigra <i>in vivo</i> by Akt/protein kinase B. Journal of Neurochemistry, 2009, 110, 23-33.	2.1	38
34	Brain-derived neurotrophic factor regulates early postnatal developmental cell death of dopamine neurons of the substantia nigra in vivo. Molecular and Cellular Neurosciences, 2009, 41, 440-447.	1.0	25
35	A critical evaluation of the Braak staging scheme for Parkinson's disease. Annals of Neurology, 2008, 64, 485-491.	2.8	331
36	Programmed cell death and new discoveries in the genetics of parkinsonism. Journal of Neurochemistry, 2008, 104, 875-890.	2.1	33

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37	JNK2 and JNK3 combined are essential for apoptosis in dopamine neurons of the substantia nigra, but are not required for axon degeneration. Journal of Neurochemistry, 2008, 107, 1578-1588.	2.1	72
38	Antiapoptotic and Trophic Effects of Dominant-Negative Forms of Dual Leucine Zipper Kinase in Dopamine Neurons of the Substantia Nigra<i>In Vivo</i>. Journal of Neuroscience, 2008, 28, 672-680.	1.7	57
39	Rodent Toxin Models of PD. , 2008, , 133-146.		0
40	Kinase signaling pathways: potential therapeutic targets in Parkinsonâ€™s disease. Future Neurology, 2007, 2, 39-49.	0.9	3
41	Programmed cell death in Parkinson's disease. Handbook of Clinical Neurology / Edited By P J Vinken and G W Bruyn, 2007, 83, 591-605.	1.0	8
42	Histochemical Methods for the Detection of Apoptosis in the Nervous System. Current Protocols in Neuroscience, 2007, 39, Unit 1.15.	2.6	1
43	Inhibition of mitogen-activated protein kinase and stimulation of Akt kinase signaling pathways: Two approaches with therapeutic potential in the treatment of neurodegenerative disease. , 2007, 114, 261-277.		99
44	Oncoprotein Akt/PKB induces trophic effects in murine models of Parkinson's disease. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 18757-18762.	3.3	151
45	GDNF as a candidate striatal target-derived neurotrophic factor for the development of substantia nigra dopamine neurons. , 2006, , 41-45.		24
46	CHOP/GADD153 is a mediator of apoptotic death in substantia nigra dopamine neurons in an in vivo neurotoxin model of parkinsonism. Journal of Neurochemistry, 2005, 95, 974-986.	2.1	264
47	Anatomical basis of glial cell line-derived neurotrophic factor expression in the striatum and related basal ganglia during postnatal development of the rat. Journal of Comparative Neurology, 2005, 484, 57-67.	0.9	41
48	Mixed lineage kinase-c-jun N-terminal kinase signaling pathway: A new therapeutic target in Parkinson's disease. Movement Disorders, 2005, 20, 653-664.	2.2	83
49	The role of GDNF in patterning the excretory system. Developmental Biology, 2005, 283, 70-84.	0.9	71
50	CEP11004, a novel inhibitor of the mixed lineage kinases, suppresses apoptotic death in dopamine neurons of the substantia nigra induced by 6â€hydroxydopamine. Journal of Neurochemistry, 2004, 88, 469-480.	2.1	35
51	Lack of alpha-synuclein does not alter apoptosis of neonatal catecholaminergic neurons. European Journal of Neuroscience, 2004, 20, 1969-1972.	1.2	29
52	Patterns of developmental mRNA expression of neurturin and GFRÎ±2 in the rat striatum and substantia nigra do not suggest a role in the regulation of natural cell death in dopamine neurons. Developmental Brain Research, 2004, 148, 143-149.	2.1	11
53	Ontogenic cell death in the nigrostriatal system. Cell and Tissue Research, 2004, 318, 63-72.	1.5	50
54	Regulation of the Development of Mesencephalic Dopaminergic Systems by the Selective Expression of Glial Cell Line-Derived Neurotrophic Factor in Their Targets. Journal of Neuroscience, 2004, 24, 3136-3146.	1.7	80

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55	Glial cell line-derived neurotrophic factor receptor GFRÎ±1 is expressed in the rat striatum during postnatal development. Molecular Brain Research, 2004, 127, 96-104.	2.5	18
56	Recent Advances in Research on Parkinson Disease: Synuclein and Parkin. Neurologist, 2004, 10, 75-81.	0.4	27
57	Ectopic Expression of Cell Cycle Markers in Models of Induced Programmed Cell Death in Dopamine Neurons of the Rat Substantia Nigra Pars Compacta. Experimental Neurology, 2003, 179, 17-27.	2.0	54
58	Pitx3 is required for development of substantia nigra dopaminergic neurons. Proceedings of the National Academy of Sciences of the United States of America, 2003, 100, 4245-4250.	3.3	361
59	Regulation of Natural Cell Death in Dopaminergic Neurons of the Substantia Nigra by Striatal Glial Cell Line-Derived Neurotrophic FactorIn Vivo. Journal of Neuroscience, 2003, 23, 5141-5148.	1.7	109
60	Postnatal Developmental Programmed Cell Death in Dopamine Neurons. Annals of the New York Academy of Sciences, 2003, 991, 69-79.	1.8	52
61	The Developmental Time Course of Glial Cell Lineâ€Derived Neurotrophic Factor (GDNF) and GDNF Receptor Î±â€1 mRNA Expression in the Striatum and Substantia Nigra. Annals of the New York Academy of Sciences, 2003, 991, 284-287.	1.8	8
62	Distinct Nuclear and Cytoplasmic Localization of Caspase Cleavage Products in Two Models of Induced Apoptotic Death in Dopamine Neurons of the Substantia Nigra. Experimental Neurology, 2002, 175, 1-9.	2.0	41
63	Analysis of synphilin-1 and synuclein interactions by yeast two-hybrid Î²-galactosidase liquid assay. Neuroscience Letters, 2002, 325, 119-123.	1.0	34
64	Resistance of Â-synuclein null mice to the parkinsonian neurotoxin MPTP. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 14524-14529.	3.3	541
65	Striatal 6-hydroxydopamine induces apoptosis of nigral neurons in the adult rat. Brain Research, 2002, 958, 185-191.	1.1	60
66	Medial forebrain bundle axotomy during development induces apoptosis in dopamine neurons of the substantia nigra and activation of caspases in their degenerating axons. Journal of Comparative Neurology, 2002, 452, 65-79.	0.9	47
67	Activation of Caspase-3 in Developmental Models of Programmed Cell Death in Neurons of the Substantia Nigra. Journal of Neurochemistry, 2002, 73, 322-333.	2.1	110
68	Increased Expression of Rat Synuclein in the Substantia Nigra Pars Compacta Identified by mRNA Differential Display in a Model of Developmental Target Injury. Journal of Neurochemistry, 2002, 73, 2586-2599.	2.1	119
69	Î±-Synuclein and parkin: coming together of pieces in puzzle of Parkinson's disease. Lancet, The, 2001, 358, 1567-1568.	6.3	36
70	Synuclein-1 is selectively up-regulated in response to nerve growth factor treatment in PC12 cells. Journal of Neurochemistry, 2001, 76, 1165-1176.	2.1	80
71	Expression of cyclin-dependent kinase 5 and its activator p35 in models of induced apoptotic death in neurons of the substantia nigra in vivo. Journal of Neurochemistry, 2001, 77, 1611-1625.	2.1	34
72	The expression of mRNAs for the proteasome complex is developmentally regulated in the rat mesencephalon. Developmental Brain Research, 2001, 129, 47-56.	2.1	38

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73	Apoptotic Morphology in Phenotypically Defined Dopaminergic Neurons of the Substantia Nigra. , 2001, 62, 101-112.		1
74	Animal Models of Induced Apoptotic Death in the Substantia Nigra. , 2001, 62, 89-99.		1
75	Developmental cell death in dopaminergic neurons of the substantia nigra of mice. Journal of Comparative Neurology, 2000, 424, 476-488.	0.9	127
76	Upregulation of cytosolic branched chain aminotransferase in substantia nigra following developmental striatal target injury. Molecular Brain Research, 2000, 75, 281-286.	2.5	16
77	Expression of c-fos, c-jun, and N-terminal kinase (JNK) in a Development Model of Induced Apoptotic Death in Neurons of the Substantia Nigra. Journal of Neurochemistry, 1999, 72, 557-564.	2.1	79
78	Synuclein expression is decreased in rat substantia nigra following induction of apoptosis by intrastriatal 6-hydroxydopamine. Neuroscience Letters, 1999, 275, 105-108.	1.0	64
79	Î±-Synuclein and Parkinsonâ€™s disease. Brain Research Bulletin, 1999, 50, 465-466.	1.4	10
80	?-Synuclein expression in substantia nigra and cortex in Parkinson's disease. Movement Disorders, 1999, 14, 417-422.	2.2	95
81	The Diagnosis and Treatment of Tardive Disorders. , 1998, 3, 119-125.		2
82	Apoptosis in Degenerative Diseases of the Basal Ganglia. Neuroscientist, 1998, 4, 301-311.	2.6	3
83	Programmed cell death: Does it play a role in parkinson's disease?. Annals of Neurology, 1998, 44, S126-S133.	2.8	92
84	Glial Cell Lineâ€Derived Neurotrophic Growth Factor Inhibits Apoptotic Death of Postnatal Substantia Nigra Dopamine Neurons in Primary Culture. Journal of Neurochemistry, 1998, 71, 517-525.	2.1	145
85	Apoptosis in neurodegenerative disorders. Current Opinion in Neurology, 1997, 10, 299-305.	1.8	141
86	Increased expression of cyclin-dependent kinase 5 in induced apoptotic neuron death in rat substantia nigra. Neuroscience Letters, 1997, 230, 41-44.	1.0	52
87	Early Developmental Destruction of Terminals in the Striatal Target Induces Apoptosis in Dopamine Neurons of the Substantia Nigra. Journal of Neuroscience, 1997, 17, 2030-2039.	1.7	104
88	The time course of developmental cell death in phenotypically defined dopaminergic neurons of the substantia nigra. Developmental Brain Research, 1997, 98, 191-196.	2.1	149
89	Apoptotic neuron death in rat substantia nigra induced by striatal excitotoxic injury is developmentally dependent. Neuroscience Letters, 1996, 220, 85-88.	1.0	50
90	Neuron Death in the Substantia Nigra of Weaver Mouse Occurs Late in Development and Is Not Apoptotic. Journal of Neuroscience, 1996, 16, 6134-6145.	1.7	57

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91	Augmented pharmacologic stimulation of striatal acetylcholine release following developmental hypoxic-ischemic injury. Brain Research, 1996, 706, 145-150.	1.1	7
92	Delayed-onset dyskinesias. Neurology, 1996, 47, 1358-1359.	1.5	7
93	Time course and morphology of dopaminergic neuronal death caused by the neurotoxin 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine. Experimental Neurology, 1995, 4, 257-269.	1.7	555
94	6-Hydroxydopamine Lesion of the Rat Substantia Nigra: Time Course and Morphology of Cell Death. Experimental Neurology, 1995, 4, 131-137.	1.7	228
95	Apoptosis in substantia nigra following developmental hypoxic-ischemic injury. Neuroscience, 1995, 69, 893-901.	1.1	66
96	Exclusion of the DYT1 locus in a non-Jewish family with early-onset dystonia. Movement Disorders, 1994, 9, 626-632.	2.2	40
97	Localization of c-fos, c-jun, and hsp70 mRNA expression in brain after neonatal hypoxia-ischemia. Developmental Brain Research, 1994, 77, 111-121.	2.1	53
98	Apoptosis in substantia nigra following developmental striatal excitotoxic injury.. Proceedings of the National Academy of Sciences of the United States of America, 1994, 91, 8117-8121.	3.3	127
99	Acute and Persistent Suppression of Preproenkephalin mRNA Expression in the Striatum Following Developmental Hypoxicâ€Ischemic Injury. Journal of Neurochemistry, 1994, 62, 1878-1886.	2.1	25
100	Quantitation of the levels of tyrosine hydroxylase and preproenkephalin mRNAs in nigrostriatal sites after 6-hydroxydopamine lesions. Life Sciences, 1993, 52, 1577-1584.	2.0	10
101	Immediate early gene induction after neonatal hypoxia-ischemia. Molecular Brain Research, 1993, 18, 228-238.	2.5	117
102	Relative loss of the striatal striosome compartment, defined by calbindin-D28k immunostaining, following developmental hypoxic-ischemic injury. Neuroscience, 1993, 56, 305-315.	1.1	62
103	Naturally Occurring Cell Death during Postnatal Development of the Substantia Nigra Pars Compacta of Rat. Molecular and Cellular Neurosciences, 1993, 4, 30-35.	1.0	126
104	Disorders of Movement in Leigh Syndrome. Neuropediatrics, 1993, 24, 60-67.	0.3	58
105	Neonatal hypoxic-ischemic or excitotoxic striatal injury results in a decreased adult number of substantia nigra neurons. Neuroscience, 1992, 50, 559-569.	1.1	85
106	Effect of Striatal Lesion with Quinolinate on the Development of Substantia Nigra Dopaminergic Neurons: A Quantitative Morphological Analysis. Developmental Neuroscience, 1992, 14, 362-368.	1.0	16
107	Letters to the Editor. Movement Disorders, 1992, 7, 387-391.	2.2	11
108	Risk Factors for the tardive dyskinesias. Movement Disorders, 1992, 7, 8-8.	2.2	2

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109	Effect of unilateral perinatal hypoxic-ischemic brain injury on striatal dopamine uptake sites and D1 and D2 receptors in adult rats. Neuroscience Letters, 1991, 129, 197-200.	1.0	37
110	The effect of neonatal hypoxia-ischemia on striatal cholinergic neuropil: A quantitative morphologic analysis. Experimental Neurology, 1991, 113, 63-73.	2.0	16
111	Unilateral hypoxic-ischemic injury in neonatal rat results in a persistent increase in the density of striatal tyrosine hydroxylase immunoperoxidase staining. Developmental Brain Research, 1991, 58, 171-179.	2.1	40
112	Preserved striatal tyrosine hydroxylase activity, assessed in vivo, following neonatal hypoxia-ischemia. Developmental Brain Research, 1991, 61, 277-280.	2.1	10
113	Effect of Unilateral Perinatal Hypoxic-Ischemic Brain Injury in the Rat on Dopamine D1and D2Receptors and Uptake Sites: A Quantitative Autoradiographic Study. Journal of Neurochemistry, 1991, 57, 1951-1961.	2.1	64
114	Effect of Unilateral Perinatal Hypoxic-Ischemic Brain Injury in the Rat on Striatal Muscarinic Cholinergic Receptors and High-Affinity Choline Uptake Sites: A Quantitative Autoradiographic Study. Journal of Neurochemistry, 1991, 57, 1962-1970.	2.1	18
115	Intraventricular infusion of epidermal growth factor restores dopaminergic pathway in hemiparkinsonian rats. Movement Disorders, 1991, 6, 281-287.	2.2	46
116	Dopamine beta-hydroxylase gene excluded in four subtypes of hereditary dystonia. Human Genetics, 1991, 87, 311-316.	1.8	6
117	Chapter 40 Antimuscarinic drugs in the treatment of movement disorders. Progress in Brain Research, 1990, 84, 389-397.	0.9	32
118	Quantitative morphological analysis of striatal cholinergic neurons in perinatal asphyxia. Annals of Neurology, 1990, 27, 81-88.	2.8	45
119	Dystonia gene in Ashkenazi Jewish population is located on chromosome 9q32-34. Annals of Neurology, 1990, 27, 114-120.	2.8	141
120	Letters to the editor. Movement Disorders, 1990, 5, 178-183.	2.2	16
121	An assessment of the validity of densitometric measures of striatal tyrosine hydroxylase-positive fibers: relationship to apomorphine-induced rotations in 6-hydroxydopamine lesioned rats. Journal of Neuroscience Methods, 1990, 35, 63-73.	1.3	60
122	Demonstration of a medial to lateral gradient in the density of cholinergic neuropil in the rat striatum. Neuroscience Letters, 1990, 108, 58-64.	1.0	18
123	Tardive akathisia: An analysis of clinical features and response to open therapeutic trials. Movement Disorders, 1989, 4, 157-175.	2.2	139
124	Idiopathic dystonia among ashkenazi jews: Evidence for autosomal dominant inheritance. Annals of Neurology, 1989, 26, 612-620.	2.8	232
125	Asymmetrical perfusion fixation in a rodent model of perinatal hypoxia-ischemia may lead to artifactual morphologic asymmetries. Experimental Neurology, 1989, 103, 293-296.	2.0	9
126	Human gene for torsion dystonia located on chromosome 9q32-q34. Neuron, 1989, 2, 1427-1434.	3.8	246

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127	Effect of Postmortem Factors on Muscarinic Receptor Subtypes in Rat Brain. Journal of Neurochemistry, 1987, 49, 592-596.	2.1	20
128	The relative selectivity of anticholinergic drugs for the M1 and M2 muscarinic receptor subtypes. Movement Disorders, 1986, 1, 135-144.	2.2	32
129	Analysis of the clinical course of non-Jewish, autosomal dominant torsion dystonia. Movement Disorders, 1986, 1, 163-178.	2.2	26
130	Natural history and treatment of tardive dystonia. Movement Disorders, 1986, 1, 193-208.	2.2	268
131	A case of parkinsonism following striatal lacunar infarction.. Journal of Neurology, Neurosurgery and Psychiatry, 1986, 49, 1087-1088.	0.9	41
132	Tetrabenazine induces acute dystonic reactions. Annals of Neurology, 1985, 17, 200-202.	2.8	83
133	Pharmacokinetics of trihexyphenidyl after short-term and long-term administration to dystonic patients. Annals of Neurology, 1985, 18, 35-40.	2.8	32
134	The effect of selective lesions on vestibular nuclear complex choline acetyltransferase activity in the rat. Brain Research, 1985, 360, 172-182.	1.1	5
135	Choline acetyltransferase activity of the principal vestibular nuclei of rat, studied by micropunch technique. Brain Research, 1985, 328, 196-199.	1.1	20
136	Studies of somatostatin-induced barrel rotation in rats. Regulatory Peptides, 1983, 7, 207-220.	1.9	29
137	Neurotensin interacts with dopaminergic neurons in rat brain. Peptides, 1983, 4, 43-48.	1.2	31
138	Chlorpromazine methiodide acts at the vestibular nuclear complex to induce barrel rotation in the rat. Brain Research, 1983, 288, 273-281.	1.1	17
139	Electroencephalographic studies of chlorpromazine methiodide and somatostatin-induced barrel rotation in rats. Experimental Neurology, 1983, 79, 704-713.	2.0	20
140	Chlorpromazine methiodide-induced barrel rotation: an antimuscarinic effect. Brain Research, 1982, 250, 133-142.	1.1	18
141	THE EFFECT OF NEUROTENSIN ON DOPAMINERGIC NEURONS IN RAT BRAIN. Annals of the New York Academy of Sciences, 1982, 400, 420-421.	1.8	18