Benoit I Giasson

List of Publications by Year
in descending order

153	15,775	17440 63	17592 121
papers	citations	h-index	g-index

156	156	156	12032
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Combinatorial model of amyloid Î² and tau reveals synergy between amyloid deposits and tangle formation. Neuropathology and Applied Neurobiology, 2022, 48, .	3.2	8
2	Unique seeding profiles and prion-like propagation of synucleinopathies are highly dependent on the host in human Î±-synuclein transgenic mice. Acta Neuropathologica, 2022, 143, 663-685.	7.7	12
3	Pathogenic tau recruits wild-type tau into brain inclusions and induces gut degeneration in transgenic SPAM mice. Communications Biology, 2022, 5, 446.	4.4	4
4	Soluble brain homogenates from diverse human and mouse sources preferentially seed diffuse AÎ² plaque pathology when injected into newborn mouse hosts.. Free Neuropathology, 2022, 3, .	3.0	2
5	Reassessment of Neuronal Tau Distribution in Adult Human Brain and Implications for Tau Pathobiology. Acta Neuropathologica Communications, 2022, 10, .	5.2	10
6	Anti-tau scFvs Targeted to the Cytoplasm or Secretory Pathway Variably Modify Pathology and Neurodegenerative Phenotypes. Molecular Therapy, 2021, 29, 859-872.	8.2	26
7	Novel SOD1 monoclonal antibodies against the electrostatic loop preferentially detect misfolded SOD1 aggregates. Neuroscience Letters, 2021, 742, 135553.	2.1	1
8	Il-10 signaling reduces survival in mouse models of synucleinopathy. Npj Parkinson's Disease, 2021, 7, 30.	5.3	8
9	Precision therapeutic targets for COVID-19. Virology Journal, 2021, 18, 66.	3.4	40
10	Pathogenic MAPT mutations Q336H and Q336R have isoformâ€dependent differences in aggregation propensity and microtubule dysfunction. Journal of Neurochemistry, 2021, 158, 455-466.	3.9	7
11	Prodromal neuroinvasion of pathological Î±-synuclein in brainstem reticular nuclei and white matter lesions in a model of Î±-synucleinopathy. Brain Communications, 2021, 3, fcab104.	3.3	7
12	Robust Î±-synuclein pathology in select brainstem neuronal populations is a potential instigator of multiple system atrophy. Acta Neuropathologica Communications, 2021, 9, 80.	5.2	11
13	Multiple system atrophy-associated oligodendroglial protein p25Î± stimulates formation of novel Î±-synuclein strain with enhanced neurodegenerative potential. Acta Neuropathologica, 2021, 142, 87-115.	7.7	55
14	â€œDonâ€™t Phos Over Tauâ€• recent developments in clinical biomarkers and therapies targeting tau phosphorylation in Alzheimerâ€™s disease and other tauopathies. Molecular Neurodegeneration, 2021, 16, 37.	10.8	89
15	Î±-Synuclein-induced dysregulation of neuronal activity contributes to murine dopamine neuron vulnerability. Npj Parkinson's Disease, 2021, 7, 76.	5.3	14
16	Disease-, region- and cell type specific diversity of Î±-synuclein carboxy terminal truncations in synucleinopathies. Acta Neuropathologica Communications, 2021, 9, 146.	5.2	10
17	Tau K321/K353 pseudoacetylation within KXGS motifs regulates tauâ€“microtubule interactions and inhibits aggregation. Scientific Reports, 2021, 11, 17069.	3.3	13
18	Collusion of Î±-Synuclein and AÎ² aggravating co-morbidities in a novel prion-type mouse model. Molecular Neurodegeneration, 2021, 16, 63.	10.8	12

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19	Photodynamic studies reveal rapid formation and appreciable turnover of tau inclusions. Acta Neuropathologica, 2021, 141, 359-381.	7.7	13
20	Targeted proteolytic products of Ï„ and Î±-synuclein in neurodegeneration. Essays in Biochemistry, 2021, 65, 905-912.	4.7	6
21	Carboxyâ€terminal truncations of mouse Î±â€synuclein alter aggregation and prionâ€like seeding. FEBS Letters, 2020, 594, 1271-1283.	2.8	14
22	Novel monoclonal antibodies targeting the RRM2 domain of human TDP-43 protein. Neuroscience Letters, 2020, 738, 135353.	2.1	3
23	Differential cross-seeding properties of tau and Î±-synuclein in mouse models of tauopathy and synucleinopathy. Brain Communications, 2020, 2, fcaa090.	3.3	24
24	Î±-Synuclein Induces Progressive Changes in Brain Microstructure and Sensory-Evoked Brain Function That Precedes Locomotor Decline. Journal of Neuroscience, 2020, 40, 6649-6659.	3.6	10
25	Determinants of seeding and spreading of Î±-synuclein pathology in the brain. Science Advances, 2020, 6, .	10.3	61
26	Transgenic Mice Expressing Human Î±-Synuclein in Noradrenergic Neurons Develop Locus Ceruleus Pathology and Nonmotor Features of Parkinson's Disease. Journal of Neuroscience, 2020, 40, 7559-7576.	3.6	32
27	Carboxy-terminal truncation and phosphorylation of Î±-synuclein elongates survival in a prion-like seeding mouse model of synucleinopathy. Neuroscience Letters, 2020, 732, 135017.	2.1	11
28	Prominent amyloid plaque pathology and cerebral amyloid angiopathy in APP V717I (London) carrier â€“ phenotypic variability in autosomal dominant Alzheimerâ€™s disease. Acta Neuropathologica Communications, 2020, 8, 31.	5.2	14
29	Tau Ser208 phosphorylation promotes aggregation and reveals neuropathologic diversity in Alzheimerâ€™s disease and other tauopathies. Acta Neuropathologica Communications, 2020, 8, 88.	5.2	52
30	Generation and Characterization of Novel Monoclonal Antibodies TargetingÂp62/sequestosome-1 Across Human Neurodegenerative Diseases. Journal of Neuropathology and Experimental Neurology, 2020, 79, 407-418.	1.7	8
31	The emerging role of Î±-synuclein truncation in aggregation and disease. Journal of Biological Chemistry, 2020, 295, 10224-10244.	3.4	99
32	Combining P301L and S320F tau variants produces a novel accelerated model of tauopathy. Human Molecular Genetics, 2019, 28, 3255-3269.	2.9	24
33	Fragile X-associated tremor ataxia syndrome with co-occurrent progressive supranuclear palsy-like neuropathology. Acta Neuropathologica Communications, 2019, 7, 158.	5.2	8
34	Impaired tauâ€“microtubule interactions are prevalent among pathogenic tau variants arising from missense mutations. Journal of Biological Chemistry, 2019, 294, 18488-18503.	3.4	23
35	Exploring the Peripheral Initiation of Parkinsonâ€™s Disease in Animal Models. Neuron, 2019, 103, 547-549.	8.1	5
36	Unique Î±-synuclein pathology within the amygdala in Lewy body dementia: implications for disease initiation and progression. Acta Neuropathologica Communications, 2019, 7, 142.	5.2	49

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37	Comparative analyses of the in vivo induction and transmission of Î±-synuclein pathology in transgenic mice by MSA brain lysate and recombinant Î±-synuclein fibrils. Acta Neuropathologica Communications, 2019, 7, 80.	5.2	30
38	Endogenous oligodendroglial alpha-synuclein and TPPP/p25Î± orchestrate alpha-synuclein pathology in experimental multiple system atrophy models. Acta Neuropathologica, 2019, 138, 415-441.	7.7	45
39	Î±-Synuclein and astrocytes: tracing the pathways from homeostasis to neurodegeneration in Lewy body disease. Acta Neuropathologica, 2019, 138, 1-21.	7.7	109
40	Dissecting Î±-synuclein inclusion pathology diversity in multiple system atrophy: implications for the prion-like transmission hypothesis. Laboratory Investigation, 2019, 99, 982-992.	3.7	15
41	MAPT mutations, tauopathy, and mechanisms of neurodegeneration. Laboratory Investigation, 2019, 99, 912-928.	3.7	190
42	rAAV-based brain slice culture models of Alzheimerâ€™s and Parkinsonâ€™s disease inclusion pathologies. Journal of Experimental Medicine, 2019, 216, 539-555.	8.5	48
43	An anti-CRF antibody suppresses the HPA axis and reverses stress-induced phenotypes. Journal of Experimental Medicine, 2019, 216, 2479-2491.	8.5	7
44	Phosphorylation of serine 305 in tau inhibits aggregation. Neuroscience Letters, 2019, 692, 187-192.	2.1	25
45	Locomotor differences in mice expressing wild-type human Î±-synuclein. Neurobiology of Aging, 2018, 65, 140-148.	3.1	15
46	Distinct differences in prion-like seeding and aggregation between Tau protein variants provide mechanistic insights into tauopathies. Journal of Biological Chemistry, 2018, 293, 2408-2421.	3.4	103
47	Adsorption and decontamination of Î±-synuclein from medically and environmentally-relevant surfaces. Colloids and Surfaces B: Biointerfaces, 2018, 166, 98-107.	5.0	7
48	Prion-like Spreading in Tauopathies. Biological Psychiatry, 2018, 83, 337-346.	1.3	70
49	Designing antibodies against LRRK2-targeted tau epitopes. PLoS ONE, 2018, 13, e0204367.	2.5	1
50	Physiological C-terminal truncation of Î±-synuclein potentiates the prion-like formation of pathological inclusions. Journal of Biological Chemistry, 2018, 293, 18914-18932.	3.4	64
51	Differential induction of mutant SOD1 misfolding and aggregation by tau and Î±-synuclein pathology. Molecular Neurodegeneration, 2018, 13, 23.	10.8	3
52	Motor neuron loss and neuroinflammation in a model of Î±-synuclein-induced neurodegeneration. Neurobiology of Disease, 2018, 120, 98-106.	4.4	32
53	TLR5 decoy receptor as a novel anti-amyloid therapeutic for Alzheimerâ€™s disease. Journal of Experimental Medicine, 2018, 215, 2247-2264.	8.5	50
54	Novel monoclonal antibodies targeting the microtubule-binding domain of human tau. PLoS ONE, 2018, 13, e0195211.	2.5	12

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55	Localized Induction of Wild-Type and Mutant Alpha-Synuclein Aggregation Reveals Propagation along Neuroanatomical Tracts. Journal of Virology, 2018, 92, .	3.4	28
56	Changes in proteome solubility indicate widespread proteostatic disruption in mouse models of neurodegenerative disease. Acta Neuropathologica, 2018, 136, 919-938.	7.7	27
57	Partial loss of ATP13A2 causes selective gliosis independent of robust lipofuscinosis. Molecular and Cellular Neurosciences, 2018, 92, 17-26.	2.2	11
58	Inflammatory pre-conditioning restricts the seeded induction of Î±-synuclein pathology in wild type mice. Molecular Neurodegeneration, 2017, 12, 1.	10.8	104
59	Prion-like transmission of Î±-synuclein pathology in the context of an NFL null background. Neuroscience Letters, 2017, 661, 114-120.	2.1	2
60	Comparison of the in vivo induction and transmission of Î±-synuclein pathology by mutant Î±-synuclein fibril seeds in transgenic mice. Human Molecular Genetics, 2017, 26, 4906-4915.	2.9	22
61	Glucocerebrosidase haploinsufficiency in A53T Î±-synuclein mice impacts disease onset and course. Molecular Genetics and Metabolism, 2017, 122, 198-208.	1.1	28
62	The major targets of acute norovirus infection are immune cells in the gut-associated lymphoid tissue. Nature Microbiology, 2017, 2, 1586-1591.	13.3	86
63	Intrastriatal injection of Î±-synuclein can lead to widespread synucleinopathy independent of neuroanatomic connectivity. Molecular Neurodegeneration, 2017, 12, 40.	10.8	51
64	Robust Central Nervous System Pathology in Transgenic Mice following Peripheral Injection of Î±-Synuclein Fibrils. Journal of Virology, 2017, 91, .	3.4	73
65	Proteolysis of Î±â€synuclein fibrils in the lysosomal pathway limits induction of inclusion pathology. Journal of Neurochemistry, 2017, 140, 662-678.	3.9	59
66	The ER retention protein RER1 promotes alpha-synuclein degradation via the proteasome. PLoS ONE, 2017, 12, e0184262.	2.5	15
67	Generation and characterization of new monoclonal antibodies targeting the PHF1 and AT8 epitopes on human tau. Acta Neuropathologica Communications, 2017, 5, 58.	5.2	39
68	A novel panel of Î±-synuclein antibodies reveal distinctive staining profiles in synucleinopathies. PLoS ONE, 2017, 12, e0184731.	2.5	45
69	Novel antibodies to phosphorylated Î±-synuclein serine 129 and NFL serine 473 demonstrate the close molecular homology of these epitopes. Acta Neuropathologica Communications, 2016, 4, 80.	5.2	47
70	Propagation of AÎ², tau and Î±-synuclein pathology between experimental models and human reality: prions, propagons and propaganda. Acta Neuropathologica, 2016, 131, 1-3.	7.7	51
71	Propagation of alpha-synuclein pathology: hypotheses, discoveries, and yet unresolved questions from experimental and human brain studies. Acta Neuropathologica, 2016, 131, 49-73.	7.7	179
72	Non-prion-type transmission in A53T Î±-synuclein transgenic mice: a normal component of spinal homogenates from naÃ¯ve non-transgenic mice induces robust Î±-synuclein pathology. Acta Neuropathologica, 2016, 131, 151-154.	7.7	19

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73	S4-02-03: Cell-to-cell transmission of synucleinopathy. , 2015, 11, P258-P258.		0
74	Physiologically relevant factors influence tau phosphorylation by leucineâ€rich repeat kinase 2. Journal of Neuroscience Research, 2015, 93, 1567-1580.	2.9	18
75	Cp/Heph mutant mice have ironâ€induced neurodegeneration diminished by deferiprone. Journal of Neurochemistry, 2015, 135, 958-974.	3.9	35
76	Studies of lipopolysaccharide effects on the induction of Î±-synuclein pathology by exogenous fibrils in transgenic mice. Molecular Neurodegeneration, 2015, 10, 32.	10.8	29
77	Inefficient induction and spread of seeded tau pathology in P301L mouse model of tauopathy suggests inherent physiological barriers to transmission. Acta Neuropathologica, 2015, 130, 303-305.	7.7	9
78	The A53E Î±-synuclein pathological mutation demonstrates reduced aggregation propensity in vitro and in cell culture. Neuroscience Letters, 2015, 597, 43-48.	2.1	26
79	Divergent effects of the H50Q and G51D SNCA mutations on the aggregation of Î±â€synuclein. Journal of Neurochemistry, 2014, 131, 859-867.	3.9	104
80	Amyloidogenic Î±-synuclein seeds do not invariably induce rapid, widespread pathology in mice. Acta Neuropathologica, 2014, 127, 645-665.	7.7	103
81	Augmentation of phenotype in a transgenic Parkinson mouse heterozygous for a Gaucher mutation. Brain, 2014, 137, 3235-3247.	7.6	88
82	Brain Injection of Î±-Synuclein Induces Multiple Proteinopathies, Gliosis, and a Neuronal Injury Marker. Journal of Neuroscience, 2014, 34, 12368-12378.	3.6	115
83	Intramuscular injection of Î±-synuclein induces CNS Î±-synuclein pathology and a rapid-onset motor phenotype in transgenic mice. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, 10732-10737.	7.1	277
84	Transgenic mice expressing S129 phosphorylation mutations in Î±-synuclein. Neuroscience Letters, 2014, 563, 96-100.	2.1	12
85	A Cellular System that Degrades Misfolded Proteins and Protects against Neurodegeneration. Molecular Cell, 2014, 55, 15-30.	9.7	157
86	Conformational templating of Î±-synuclein aggregates in neuronal-glial cultures. Molecular Neurodegeneration, 2013, 8, 17.	10.8	61
87	Induction of CNS Î±-synuclein pathology by fibrillar and non-amyloidogenic recombinant Î±-synuclein. Acta Neuropathologica Communications, 2013, 1, 38.	5.2	78
88	Robust cytoplasmic accumulation of phosphorylated TDP-43 in transgenic models of tauopathy. Acta Neuropathologica, 2013, 126, 39-50.	7.7	24
89	LRRK2 phosphorylates novel tau epitopes and promotes tauopathy. Acta Neuropathologica, 2013, 126, 809-827.	7.7	85
90	Unbiased screen reveals ubiquilin-1 and -2 highly associated with huntingtin inclusions. Brain Research, 2013, 1524, 62-73.	2.2	38

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91	Atp13a2-deficient mice exhibit neuronal ceroid lipofuscinosis, limited Î±-synuclein accumulation and age-dependent sensorimotor deficits. Human Molecular Genetics, 2013, 22, 2067-2082.	2.9	124
92	Thinking laterally about neurodegenerative proteinopathies. Journal of Clinical Investigation, 2013, 123, 1847-1855.	8.2	98
93	Does a prion-like mechanism play a major role in the apparent spread of Î±-synuclein pathology?. Alzheimer's Research and Therapy, 2012, 4, 48.	6.2	5
94	Characterization of cellular protective effects of ATP13A2/PARK9 expression and alterations resulting from pathogenic mutants. Journal of Neuroscience Research, 2012, 90, 2306-2316.	2.9	37
95	Characterization of kinases involved in the phosphorylation of aggregated Î±â€synuclein. Journal of Neuroscience Research, 2011, 89, 231-247.	2.9	73
96	E46K Human Î±-Synuclein Transgenic Mice Develop Lewy-like and Tau Pathology Associated with Age-dependent, Detrimental Motor Impairment. Journal of Biological Chemistry, 2011, 286, 35104-35118.	3.4	115
97	Induction of Intracellular Tau Aggregation Is Promoted by Î±-Synuclein Seeds and Provides Novel Insights into the Hyperphosphorylation of Tau. Journal of Neuroscience, 2011, 31, 7604-7618.	3.6	165
98	A novel, highâ€efficiency cellular model of fibrillar Î±â€synuclein inclusions and the examination of mutations that inhibit amyloid formation. Journal of Neurochemistry, 2010, 113, 374-388.	3.9	75
99	The G2019S pathogenic mutation disrupts sensitivity of leucineâ€rich repeat kinase 2 to manganese kinase inhibition. Journal of Neurochemistry, 2010, 115, 36-46.	3.9	19
100	Extensive enteric nervous system abnormalities in mice transgenic for artificial chromosomes containing Parkinson disease-associated Î±-synuclein gene mutations precede central nervous system changes. Human Molecular Genetics, 2010, 19, 1633-1650.	2.9	237
101	Residue Glu83 plays a major role in negatively regulating Î±-synuclein amyloid formation. Biochemical and Biophysical Research Communications, 2010, 391, 1415-1420.	2.1	17
102	Clinical and pathological characteristics of patients with Leucineâ€rich repeat kinaseâ€2 mutations. Movement Disorders, 2009, 24, 32-39.	3.9	44
103	Characterization of Hydrophobic Residue Requirements for Î±-Synuclein Fibrillization. Biochemistry, 2009, 48, 9427-9436.	2.5	82
104	Molecular mechanisms of Î±-synuclein neurodegeneration. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2009, 1792, 616-624.	3.8	174
105	Identification of compounds that inhibit the kinase activity of leucine-rich repeat kinase 2. Biochemical and Biophysical Research Communications, 2009, 378, 473-477.	2.1	84
106	Leucine-Rich Repeat Kinase 2 Expression Leads to Aggresome Formation That Is Not Associated With Î±-Synuclein Inclusions. Journal of Neuropathology and Experimental Neurology, 2009, 68, 785-796.	1.7	29
107	Characterization of antibodies that selectively detect Î±-synuclein in pathological inclusions. Acta Neuropathologica, 2008, 116, 37-46.	7.7	72
108	Î±-Synuclein activates stress signaling protein kinases in THP-1 cells and microglia. Neurobiology of Aging, 2008, 29, 739-752.	3.1	202

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109	Mutations in <i>LRRK2</i> as a Cause of Parkinsonâ€™s Disease. NeuroSignals, 2008, 16, 99-105.	0.9	48
110	Neurofurans, Novel Indices of Oxidant Stress Derived from Docosahexaenoic Acid. Journal of Biological Chemistry, 2008, 283, 6-16.	3.4	73
111	Specificity and Regulation of Casein Kinase-Mediated Phosphorylation of Î±-Synuclein. Journal of Neuropathology and Experimental Neurology, 2008, 67, 402-416.	1.7	176
112	Specificity and Regulation of Casein Kinase-Mediated Phosphorylation of ??-Synuclein. Journal of Neuropathology and Experimental Neurology, 2008, PAP, 402-16.	1.7	116
113	Role of Mitochondrial Dysfunction in Parkinson???s Disease. Drugs and Aging, 2007, 24, 95-105.	2.7	18
114	Alphaâ€synuclein and its diseaseâ€causing mutants induce ICAMâ€1 and ILâ€6 in human astrocytes and astrocytoma cells. FASEB Journal, 2006, 20, 2000-2008.	0.5	126
115	Biochemical and pathological characterization of Lrrk2. Annals of Neurology, 2006, 59, 315-322.	5.3	229
116	Lack of evidence for Lrrk2 in Î±-synuclein pathological inclusions. Annals of Neurology, 2006, 60, 618-619.	5.3	23
117	The E46K Mutation in Î±-Synuclein Increases Amyloid Fibril Formation. Journal of Biological Chemistry, 2005, 280, 7800-7807.	3.4	327
118	Reversible Inhibition of Î±-Synuclein Fibrillization by Dopaminochrome-mediated Conformational Alterations*. Journal of Biological Chemistry, 2005, 280, 21212-21219.	3.4	248
119	A Precipitating Role for Truncated Î±-Synuclein and the Proteasome in Î±-Synuclein Aggregation. Journal of Biological Chemistry, 2005, 280, 22670-22678.	3.4	229
120	Cleavage of Î±-Synuclein by Calpain:Â Potential Role in Degradation of Fibrillized and Nitrated Species of Î±-Synucleinâ€. Biochemistry, 2005, 44, 7818-7829.	2.5	107
121	Snaring the Function of Î±-Synuclein. Cell, 2005, 123, 359-361.	28.9	143
122	Mouse Model of Multiple System Atrophy Î±-Synuclein Expression in Oligodendrocytes Causes Glial and Neuronal Degeneration. Neuron, 2005, 45, 847-859.	8.1	277
123	Role of Oxidative Damage in Protein Aggregation Associated with Parkinson's Disease and Related Disorders. Antioxidants and Redox Signaling, 2005, 7, 672-684.	5.4	52
124	Fibrillization of ?-synuclein and tau in familial Parkinson's disease caused by the A53T ?-synuclein mutation. Experimental Neurology, 2004, 187, 279-288.	4.1	151
125	More than just two peas in a pod: common amyloidogenic properties of tau and Î±-synuclein in neurodegenerative diseases. Trends in Neurosciences, 2004, 27, 129-134.	8.6	177
126	Parkinson's disease, dementia with Lewy bodies, multiple system atrophy and the spectrum of diseases with Î±-synuclein inclusions. , 2004, , 353-375.		5

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127	Mitochondrial Injury: A Hot Spot for Parkinsonism and Parkinson's Disease?. Science of Aging Knowledge Environment: SAGE KE, 2004, 2004, pe42-pe42.	0.8	4
128	Interactions of Amyloidogenic Proteins. NeuroMolecular Medicine, 2003, 4, 49-58.	3.4	91
129	Tau and 14-3-3 in glial cytoplasmic inclusions of multiple system atrophy. Acta Neuropathologica, 2003, 106, 243-250.	7.7	36
130	Distinct cleavage patterns of normal and pathologic forms of Î±-synuclein by calpain I in vitro. Journal of Neurochemistry, 2003, 86, 836-847.	3.9	147
131	A comparison of amyloid fibrillogenesis using the novel fluorescent compound K114. Journal of Neurochemistry, 2003, 86, 1359-1368.	3.9	124
132	Role of Î±-Synuclein Carboxy-Terminus on Fibril Formation in Vitroâ€. Biochemistry, 2003, 42, 8530-8540.	2.5	314
133	Initiation and Synergistic Fibrillization of Tau and Alpha-Synuclein. Science, 2003, 300, 636-640.	12.6	791
134	Ubiquitination of Î±-Synuclein Is Not Required for Formation of Pathological Inclusions in Î±-Synucleinopathies. American Journal of Pathology, 2003, 163, 91-100.	3.8	129
135	Are Ubiquitination Pathways Central to Parkinson's Disease?. Cell, 2003, 114, 1-8.	28.9	216
136	Neuronal Î±-Synucleinopathy with Severe Movement Disorder in Mice Expressing A53T Human Î±-Synuclein. Neuron, 2002, 34, 521-533.	8.1	1,094
137	The Environmental Toxin Arsenite Induces Tau Hyperphosphorylationâ€. Biochemistry, 2002, 41, 15376-15387.	2.5	58
138	The relationship between oxidative/nitrative stress and pathological inclusions in Alzheimerâ€™s and Parkinsonâ€™s diseases1,2 11Guest Editors: Mark A. Smith and George Perry 22This article is 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, 1264-1275.	2.9	252
139	Novel antibodies to synuclein show abundant striatal pathology in Lewy body diseases. Annals of Neurology, 2002, 52, 205-210.	5.3	300
140	Concurrence of Î±-synuclein and tau brain pathology in the Contursi kindred. Acta Neuropathologica, 2002, 104, 7-11.	7.7	247
141	A Hydrophobic Stretch of 12 Amino Acid Residues in the Middle of Î±-Synuclein Is Essential for Filament Assembly. Journal of Biological Chemistry, 2001, 276, 2380-2386.	3.4	865
142	Prominent Perikaryal Expression of Î±- and Î²-Synuclein in Neurons of Dorsal Root Ganglion and in Medullary Neurons. Experimental Neurology, 2001, 172, 354-362.	4.1	49
143	Induction of Î±-Synuclein Aggregation by Intracellular Nitrative Insult. Journal of Neuroscience, 2001, 21, 8053-8061.	3.6	412
144	Oxidative post-translational modifications of Î±-synuclein in the 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) mouse model of Parkinson's disease. Journal of Neurochemistry, 2001, 76, 637-640.	3.9	184

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145	Immunohistochemical and Biochemical Studies Demonstrate a Distinct Profile of Î±-Synuclein Permutations in Multiple System Atrophy. Journal of Neuropathology and Experimental Neurology, 2000, 59, 830-841.	1.7	135
146	Synucleins are expressed in the majority of breast and ovarian carcinomas and in preneoplastic lesions of the ovary. Cancer, 2000, 88, 2154-2163.	4.1	145
147	A panel of epitope-specific antibodies detects protein domains distributed throughout human ?-synuclein in lewy bodies of Parkinson's disease. , 2000, 59, 528-533.		197
148	A new link between pesticides and Parkinson's disease. Nature Neuroscience, 2000, 3, 1227-1228.	14.8	67
149	Neurodegeneration with Brain Iron Accumulation, Type 1 Is Characterized by Î±-, Î²-, and Î³-Synuclein Neuropathology. American Journal of Pathology, 2000, 157, 361-368.	3.8	190
150	Chaperone-like activity of synucleins. FEBS Letters, 2000, 474, 116-119.	2.8	196
151	Mutant and Wild Type Human Î±-Synucleins Assemble into Elongated Filaments with Distinct Morphologies in Vitro. Journal of Biological Chemistry, 1999, 274, 7619-7622.	3.4	478
152	Glial cytoplasmic inclusions in white matter oligodendrocytes of multiple system atrophy brains contain insoluble ?-synuclein. Annals of Neurology, 1998, 44, 415-422.	5.3	633
153	Lewy Bodies Contain Altered Î±-Synuclein in Brains of Many Familial Alzheimer's Disease Patients with Mutations in Presenilin and Amyloid Precursor Protein Genes. American Journal of Pathology, 1998, 153, 1365-1370.	3.8	484