Hardy J Rideout

List of Publications by Year
in descending order

41 papers	3,097 citations	236925 25 h-index	289244 40 g-index
43 all docs	43 docs citations	43 times ranked	3708 citing authors

#	Article	IF	Citations
1	Leucine rich repeat kinase 2 (<scp>LRRK2</scp>) peptide modulators: Recent advances and future directions. Peptide Science, 2022, 114, .	1.8	0
2	Distinct profiles of LRRK2 activation and Rab GTPase phosphorylation in clinical samples from different PD cohorts. Npj Parkinson's Disease, 2022, 8, .	5.3	12
3	Allosteric Inhibition of Parkinsonâ€™s-Linked LRRK2 by Constrained Peptides. ACS Chemical Biology, 2021, 16, 2326-2338.	3.4	15
4	Defining (and blocking) neuronal death in Parkinsonâ€™s disease: Does it matter what we call it?. Brain Research, 2021, 1771, 147639.	2.2	3
5	The Current State-of-the Art of LRRK2-Based Biomarker Assay Development in Parkinsonâ€™s Disease. Frontiers in Neuroscience, 2020, 14, 865.	2.8	30
6	The Future of Targeted Gene-Based Treatment Strategies and Biomarkers in Parkinsonâ€™s Disease. Biomolecules, 2020, 10, 912.	4.0	18
7	Elevated In Vitro Kinase Activity in Peripheral Blood Mononuclear Cells of <scp>Leucineâ€Rich</scp> Repeat Kinase 2 <scp>G2019S</scp> Carriers: A Novel <scp>Enzymeâ€Linked</scp> Immunosorbent Assayâ€“Based Method. Movement Disorders, 2020, 35, 2095-2100.	3.9	24
8	Kinase activity of mutant LRRK2 manifests differently in hetero-dimeric vs. homo-dimeric complexes. Biochemical Journal, 2019, 476, 559-579.	3.7	19
9	Vitamin B12 modulates Parkinsonâ€™s disease LRRK2 kinase activity through allosteric regulation and confers neuroprotection. Cell Research, 2019, 29, 313-329.	12.0	42
10	P62/SQSTM1 is a novel leucine-rich repeat kinase 2 (LRRK2) substrate that enhances neuronal toxicity. Biochemical Journal, 2018, 475, 1271-1293.	3.7	45
11	A motif within the armadillo repeat of Parkinsonâ€™s-linked LRRK2 interacts with FADD to hijack the extrinsic death pathway. Scientific Reports, 2018, 8, 3455.	3.3	24
12	Insights into the Influence of Specific Splicing Events on the Structural Organization of LRRK2. International Journal of Molecular Sciences, 2018, 19, 2784.	4.1	2
13	Neuronal death signaling pathways triggered by mutant LRRK2. Biochemical Society Transactions, 2017, 45, 123-129.	3.4	8
14	Nurr1:RXRÎ± heterodimer activation as monotherapy for Parkinsonâ€™s disease. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, 3999-4004.	7.1	61
15	LRRK2 and the â€œLRRKtosomeâ€•at the Crossroads of Programmed Cell Death: Clues from RIP Kinase Relatives. Advances in Neurobiology, 2017, 14, 193-208.	1.8	9
16	Is spinal muscular atrophy a disease of the motor neurons only: pathogenesis and therapeutic implications?. Cellular and Molecular Life Sciences, 2016, 73, 1003-1020.	5.4	49
17	Activation of FADD-Dependent Neuronal Death Pathways as a Predictor of Pathogenicity for LRRK2 Mutations. PLoS ONE, 2016, 11, e0166053.	2.5	16
18	The Neurobiology of LRRK2 and its Role in the Pathogenesis of Parkinsonâ€™s Disease. Neurochemical Research, 2014, 39, 576-592.	3.3	61

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19	LRRK2 Parkinson disease mutations enhance its microtubule association. Human Molecular Genetics, 2012, 21, 890-899.	2.9	177
20	Targeted disruption of neuronal 19S proteasome subunits induces the formation of ubiquitinated inclusions in the absence of cell death. Journal of Neurochemistry, 2011, 119, 630-643.	3.9	7
21	Pathological roles of Î±-synuclein in neurological disorders. Lancet Neurology, The, 2011, 10, 1015-1025.	10.2	328
22	The WD40 Domain Is Required for LRRK2 Neurotoxicity. PLoS ONE, 2009, 4, e8463.	2.5	100
23	The Parkinson Disease Protein Leucine-Rich Repeat Kinase 2 Transduces Death Signals via Fas-Associated Protein with Death Domain and Caspase-8 in a Cellular Model of Neurodegeneration. Journal of Neuroscience, 2009, 29, 1011-1016.	3.6	147
24	A novel cell death pathway that is partially caspase dependent, but morphologically non-apoptotic, elicited by proteasomal inhibition of rat sympathetic neurons. Journal of Neurochemistry, 2008, 105, 653-665.	3.9	7
25	Differential effects of Parkin and its mutants on protein aggregation, the ubiquitin-proteasome system, and neuronal cell death in human neuroblastoma cells. Journal of Neurochemistry, 2007, 102, 1292-1303.	3.9	21
26	Dopaminergic neurons in rat ventral midbrain cultures undergo selective apoptosis and form inclusions, but do not upâ€regulate iHSP70, following proteasomal inhibition. Journal of Neurochemistry, 2005, 93, 1304-1313.	3.9	74
27	Î±-Synuclein Is Required for the Fibrillar Nature of Ubiquitinated Inclusions Induced by Proteasomal Inhibition in Primary Neurons. Journal of Biological Chemistry, 2004, 279, 46915-46920.	3.4	45
28	Application of proteasomal inhibitors to mouse sympathetic neurons activates the intrinsic apoptotic pathway. Journal of Neurochemistry, 2004, 90, 1511-1520.	3.9	50
29	Neurobiology of Î±-Synuclein. Molecular Neurobiology, 2004, 30, 001-022.	4.0	95
30	Involvement of macroautophagy in the dissolution of neuronal inclusions. International Journal of Biochemistry and Cell Biology, 2004, 36, 2551-2562.	2.8	154
31	Regulation of Î±-synuclein by bFGF in cultured ventral midbrain dopaminergic neurons. Journal of Neurochemistry, 2003, 84, 803-813.	3.9	39
32	Lack of p53 delays apoptosis, but increases ubiquitinated inclusions, in proteasomal inhibitor-treated cultured cortical neurons. Molecular and Cellular Neurosciences, 2003, 24, 430-441.	2.2	43
33	Mechanisms of Caspase-Independent Neuronal Death: Energy Depletion and Free Radical Generation. Journal of Neuroscience, 2003, 23, 11015-11025.	3.6	89
34	Cyclin-Dependent Kinase Activity Is Required for Apoptotic Death But Not Inclusion Formation in Cortical Neurons after Proteasomal Inhibition. Journal of Neuroscience, 2003, 23, 1237-1245.	3.6	107
35	Proteasomal Inhibition-Induced Inclusion Formation and Death in Cortical Neurons Require Transcription and Ubiquitination. Molecular and Cellular Neurosciences, 2002, 21, 223-238.	2.2	118
36	Cyclin-Dependent Kinases and P53 Pathways Are Activated Independently and Mediate Bax Activation in Neurons after DNA Damage. Journal of Neuroscience, 2001, 21, 5017-5026.	3.6	100

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37	Expression of A53T Mutant But Not Wild-Type Î±-Synuclein in PC12 Cells Induces Alterations of the Ubiquitin-Dependent Degradation System, Loss of Dopamine Release, and Autophagic Cell Death. Journal of Neuroscience, 2001, 21, 9549-9560.	3.6	540
38	Synuclein-1 is selectively up-regulated in response to nerve growth factor treatment in PC12 cells. Journal of Neurochemistry, 2001, 76, 1165-1176.	3.9	80
39	Proteasomal inhibition leads to formation of ubiquitin/Î±â€synucleinâ€immunoreactive inclusions in PC12 cells. Journal of Neurochemistry, 2001, 78, 899-908.	3.9	253
40	Reduced Mitochondrial Membrane Potential and Altered Responsiveness of a Mitochondrial Membrane Megachannel in p53-Induced Senescence. Biochemical and Biophysical Research Communications, 1999, 261, 123-130.	2.1	34
41	Morphine enhancement of sucrose palatability: Analysis by the taste reactivity test. Pharmacology Biochemistry and Behavior, 1996, 53, 731-734.	2.9	49