Cristine Alves da Costa

List of Publications by Year
in descending order

73 papers	4,835 citations	126907 33 h-index	98798 67 g-index
74 all docs	74 docs citations	74 times ranked	5935 citing authors

#	Article	IF	Citations
1	Guidelines for the use and interpretation of assays for monitoring autophagy (4th) Tj ETQq1 1 0.784314 rgBT /Overlock 10 Tf 50 742 Td	9.1	1,430
2	Presenilin-Dependent Transcriptional Control of the AÎ²-Degrading Enzyme Neprilysin by Intracellular Domains of Î²APP and APLP. Neuron, 2005, 46, 541-554.	8.1	317
3	Wild-type but Not Parkinson's Disease-related Ala-53 â†' Thr Mutant Î±-Synuclein Protects Neuronal Cells from Apoptotic Stimuli. Journal of Biological Chemistry, 2000, 275, 24065-24069.	3.4	198
4	New protease inhibitors prevent Î³-secretase-mediated production of AÎ²40/42 without affecting Notch cleavage. Nature Cell Biology, 2001, 3, 507-511.	10.3	181
5	Transcriptional repression of p53 by parkin and impairment by mutations associated with autosomal recessive juvenile Parkinson's disease. Nature Cell Biology, 2009, 11, 1370-1375.	10.3	173
6	Presenilin-Dependent Â-Secretase-Mediated Control of p53-Associated Cell Death in Alzheimer's Disease. Journal of Neuroscience, 2006, 26, 6377-6385.	3.6	164
7	Wild-type and mutated presenilins 2 trigger p53-dependent apoptosis and down-regulate presenilin 1 expression in HEK293 human cells and in murine neurons. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 4043-4048.	7.1	129
8	Î±-Synuclein and the Parkinson's disease-related mutant Ala53Thr-Î±-synuclein do not undergo proteasomal degradation in HEK293 and neuronal cells. Neuroscience Letters, 2000, 285, 79-82.	2.1	121
9	Î±-Synuclein Lowers p53-dependent Apoptotic Response of Neuronal Cells. Journal of Biological Chemistry, 2002, 277, 50980-50984.	3.4	119
10	Endogenous Î²-amyloid production in presenilin-deficient embryonic mouse fibroblasts. Nature Cell Biology, 2001, 3, 1030-1033.	10.3	94
11	Role of the proteasome in Alzheimerâ€™s disease. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2000, 1502, 133-138.	3.8	89
12	Nuclear p53-mediated repression of autophagy involves PINK1 transcriptional down-regulation. Cell Death and Differentiation, 2018, 25, 873-884.	11.2	87
13	p53 in neurodegenerative diseases and brain cancers. , 2014, 142, 99-113.		77
14	Î²-Synuclein Displays an Antiapoptotic p53-dependent Phenotype and Protects Neurons from 6-Hydroxydopamine-induced Caspase 3 Activation. Journal of Biological Chemistry, 2003, 278, 37330-37335.	3.4	70
15	Loss of function of DJ-1 triggered by Parkinson's disease-associated mutation is due to proteolytic resistance to caspase-6. Cell Death and Differentiation, 2010, 17, 158-169.	11.2	68
16	Overexpression of PrP^c triggers caspaseâ€ƒ3 activation: potentiation by proteasome inhibitors and blockade by antiâ€PrP antibodies. Journal of Neurochemistry, 2002, 83, 1208-1214.	3.9	65
17	The C-terminal Products of Cellular Prion Protein Processing, C1 and C2, Exert Distinct Influence on p53-dependent Staurosporine-induced Caspase-3 Activation. Journal of Biological Chemistry, 2007, 282, 1956-1963.	3.4	65
18	ER-stress-associated functional link between Parkin and DJ-1 via a transcriptional cascade involving the tumor suppressor p53 and the spliced X-box binding protein XBP-1. Journal of Cell Science, 2013, 126, 2124-33.	2.0	65

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19	Primary Cultured Neurons Devoid of Cellular Prion Display Lower Responsiveness to Staurosporine through the Control of p53 at Both Transcriptional and Post-transcriptional Levels. Journal of Biological Chemistry, 2004, 279, 612-618.	3.4	62
20	Apoptosis in Parkinson's disease: Is p53 the missing link between genetic and sporadic Parkinsonism?. Cellular Signalling, 2011, 23, 963-968.	3.6	60
21	The Endoplasmic Reticulum Stress/Unfolded Protein Response and Their Contributions to Parkinsonâ€™s Disease Physiopathology. Cells, 2020, 9, 2495.	4.1	54
22	DJ-1: A New Comer in Parkinsons Disease Pathology. Current Molecular Medicine, 2007, 7, 650-657.	1.3	52
23	The C-terminal Fragment of Presenilin 2 Triggers p53-mediated Staurosporine-induced Apoptosis, a Function Independent of the Presenilinase-derived N-terminal Counterpart. Journal of Biological Chemistry, 2003, 278, 12064-12069.	3.4	50
24	Glioma tumor grade correlates with parkin depletion in mutant p53-linked tumors and results from loss of function of p53 transcriptional activity. Oncogene, 2014, 33, 1764-1775.	5.9	49
25	6-Hydroxydopamine but Not 1-Methyl-4-phenylpyridinium Abolishes Î±-Synuclein Anti-apoptotic Phenotype by Inhibiting Its Proteasomal Degradation and by Promoting Its Aggregation. Journal of Biological Chemistry, 2006, 281, 9824-9831.	3.4	48
26	Î²-Amyloid Precursor Protein Intracellular Domain Controls Mitochondrial Function by Modulating Phosphatase and Tensin Homologâ€“Induced Kinase 1 Transcription in Cells and in Alzheimer Mice Models. Biological Psychiatry, 2018, 83, 416-427.	1.3	45
27	Synthesis of new 3-alkoxy-7-amino-4-chloro-isocoumarin derivatives as new Î²-amyloid peptide production inhibitors and their activities on various classes of protease. Bioorganic and Medicinal Chemistry, 2003, 11, 3141-3152.	3.0	44
28	JLK isocoumarin inhibitors: Selective ?-secretase inhibitors that do not interfere with notch pathway in vitro or in vivo. Journal of Neuroscience Research, 2003, 74, 370-377.	2.9	43
29	Alzheimer's Disease-Linked Mutation of Presenilin 2 (N141I-PS2) Drastically Lowers APPÎ± Secretion: Control by the Proteasome. Biochemical and Biophysical Research Communications, 1998, 252, 134-138.	2.1	42
30	Neprilysin activity and expression are controlled by nicastrin. Journal of Neurochemistry, 2006, 97, 1052-1056.	3.9	39
31	The γ /η-Secretase-Derived APP Intracellular Domain Fragments Regulate p53. Current Alzheimer Research, 2007, 4, 423-426.	1.4	38
32	p53 Is Regulated by and Regulates Members of the Î³-Secretase Complex. Neurodegenerative Diseases, 2010, 7, 50-55.	1.4	38
33	Amyloid-lowering isocoumarins are not direct inhibitors of Î³-secretase. Nature Cell Biology, 2002, 4, E110-E111.	10.3	37
34	Caspase-3-derived C-terminal Product of Synphilin-1 Displays Antiapoptotic Function via Modulation of the p53-dependent Cell Death Pathway. Journal of Biological Chemistry, 2006, 281, 11515-11522.	3.4	34
35	The caspase-derived C-terminal fragment of Î²APP induces caspase-independent toxicity and triggers selective increase of AÎ²42 in mammalian cells. Journal of Neurochemistry, 2001, 78, 1153-1161.	3.9	33
36	Direct Î±-synuclein promoter transactivation by the tumor suppressor p53. Molecular Neurodegeneration, 2016, 11, 13.	10.8	33

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37	Parkin differently regulates presenilin-1 and presenilin-2 functions by direct control of their promoter transcription. Journal of Molecular Cell Biology, 2013, 5, 132-142.	3.3	31
38	Presenilins at the crossroad of a functional interplay between PARK2/PARKIN and PINK1 to control mitophagy: Implication for neurodegenerative diseases. Autophagy, 2017, 13, 2004-2005.	9.1	30
39	The Transcription Factor Function of Parkin: Breaking the Dogma. Frontiers in Neuroscience, 2018, 12, 965.	2.8	27
40	C-Terminal Maturation Fragments of Presenilin 1 and 2 Control Secretion of APPÎ± and AÎ² by Human Cells and Are Degraded by Proteasome. Molecular Medicine, 1999, 5, 160-168.	4.4	26
41	Transcription- and phosphorylation-dependent control of a functional interplay between XBP1s and PINK1 governs mitophagy and potentially impacts Parkinson disease pathophysiology. Autophagy, 2021, 17, 4363-4385.	9.1	26
42	Catabolism of endogenous and overexpressed APH1a and PEN2: evidence for artifactual involvement of the proteasome in the degradation of overexpressed proteins. Biochemical Journal, 2006, 394, 501-509.	3.7	25
43	Effect of protein kinase A inhibitors on the production of AÎ²40 and AÎ²42 by human cells expressing normal and Alzheimer's disease-linked mutated Î²APP and presenilin 1. British Journal of Pharmacology, 1999, 126, 1186-1190.	5.4	24
44	p53-dependent Aph-1 and Pen-2 Anti-apoptotic Phenotype Requires the Integrity of the Î³-Secretase Complex but Is Independent of Its Activity. Journal of Biological Chemistry, 2007, 282, 10516-10525.	3.4	24
45	Activity of Pz-peptidase and endo-oligopeptidase are due to the same enzyme. Biochemical and Biophysical Research Communications, 1989, 162, 1460-1464.	2.1	22
46	Oxidative Tissue Response Promoted by 5â€"Aminolevulinic Acid Promptly Induces the Increase of Plasma Antioxidant Capacity. Free Radical Research, 1997, 26, 235-243.	3.3	21
47	Response to Correspondence: Pardossi-Piquard etÂal., â€œPresenilin-Dependent Transcriptional Control of the AÎ²-Degrading Enzyme Neprilysin by Intracellular Domains of Î²APP and APLP.â€•Neuron 46, 541â€"554. Neuron, 2007, 53, 483-486.	8.1	21
48	p53-dependent control of transactivation of the Pen2 promoter by presenilins. Journal of Cell Science, 2009, 122, 4003-4008.	2.0	21
49	Recent Advances on α-Synuclein Cell Biology: Functions and Dysfunctions. Current Molecular Medicine, 2003, 3, 17-24.	1.3	20
50	The caspase 6 derived N-terminal fragment of DJ-1 promotes apoptosis via increased ROS production. Cell Death and Differentiation, 2012, 19, 1769-1778.	11.2	19
51	Potential external source of AÎ² in biological samples. Nature Cell Biology, 2002, 4, E164-E165.	10.3	17
52	p53â€Dependent control of cell death by nicastrin: lack of requirement for presenilinâ€dependent Î³â€secretase complex. Journal of Neurochemistry, 2009, 109, 225-237.	3.9	17
53	Iron mobilization by succinylacetone methyl ester in rats. A model study for hereditary tyrosinemia and porphyrias characterized by 5-Aminolevulinic acid overload. Free Radical Research, 2000, 32, 343-353.	3.3	16
54	Presenilin-directed inhibitors of gamma-secretase trigger caspase3 activation in presenilin-expressing and presenilin-deficient cells. Journal of Neurochemistry, 2004, 90, 800-806.	3.9	14

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55	Interplay between Parkin and p53 Governs a Physiological Homeostasis That Is Disrupted in Parkinson's Disease and Cerebral Cancer. Neurodegenerative Diseases, 2014, 13, 118-121.	1.4	14
56	Determination of 5-aminolevulinic acid in blood plasma, tissues and cell cultures by high-performance liquid chromatography with electrochemical detection. Biomedical Applications, 1997, 695, 245-250.	1.7	13
57	A novel parkin-mediated transcriptional function links p53 to familial Parkinsonâ€™s disease. Cell Cycle, 2010, 9, 16-17.	2.6	13
58	Nuclear TP53: An unraveled function as transcriptional repressor of PINK1. Autophagy, 2018, 14, 1-3.	9.1	11
59	JLK Inhibitors: Isocoumarin Compounds as Putative Probes to Selectively Target the γ-Secretase Pathway. Current Alzheimer Research, 2005, 2, 327-334.	1.4	10
60	Parkin: Much More than a Simple Ubiquitin Ligase. Neurodegenerative Diseases, 2012, 10, 49-51.	1.4	9
61	Upregulation of the Sarco-Endoplasmic Reticulum Calcium ATPase 1 Truncated Isoform Plays a Pathogenic Role in Alzheimerâ€™s Disease. Cells, 2019, 8, 1539.	4.1	9
62	Î±-synuclein and p53 functional interplay in physiopathological contexts. Oncotarget, 2017, 8, 9001-9002.	1.8	8
63	MÃ©tabolisme du prÃ©curseur du peptide amyloÃ¯de et prÃ©sÃ©nilines. Medecine/Sciences, 2002, 18, 717-724	0.2	7
64	Therapeutic potential of parkin as a tumor suppressor via transcriptional control of cyclins in glioblastoma cell and animal models. Theranostics, 2021, 11, 10047-10063.	10.0	7
65	Amyloid-lowering isocoumarins are not direct inhibitors of Î³-secretase - Reponse. Nature Cell Biology, 2002, 4, E111-E112.	10.3	5
66	Recent Insights on the Pro-Apoptotic Phenotype Elicited by Presenilin 2 and its Caspase and Presenilinase-Derived Fragments. Current Alzheimer Research, 2005, 2, 507-514.	1.4	5
67	Study on the Putative Contribution of Caspases and the Proteasome to the Degradation of Aph-1a and Pen-2. Neurodegenerative Diseases, 2007, 4, 156-163.	1.4	4
68	Parkin as a Molecular Bridge Linking Alzheimerâ€™s and Parkinsonâ€™s Diseases?. Biomolecules, 2022, 12, 559.	4.0	3
69	Periphilin is a novel interactor of synphilin-1, a protein implicated in Parkinson's disease. Neurogenetics, 2010, 11, 203-215.	1.4	2
70	Reply: Potential external source of AÎ² in biological samples. Nature Cell Biology, 2002, 4, E165-E166.	10.3	1
71	The transcription factor XBP-1 in neurodegenerative diseases. Molecular Neurodegeneration, 2013, 8, .	10.8	0
72	6-Hydroxydopamine but not 1-methyl-4-phenylpyridinium abolishes Î±-synuclein anti-apoptotic phenotype by inhibiting its proteasomal degradation and by promoting its aggregation.. Journal of Biological Chemistry, 2013, 288, 21208.	3.4	0

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73	p53, a Molecular Bridge Between Alzheimerâ€™s Disease Pathology and Cancers?. Research and Perspectives in Alzheimer's Disease, 2011, , 95-101.	0.1	0