Paolo Paganetti

List of Publications by Year
in descending order

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77 papers	15,478 citations	81900 39 h-index	66911 78 g-index
87 all docs	87 docs citations	87 times ranked	27482 citing authors

#	Article	IF	Citations
1	The heat shock response, determined by QuantiGene multiplex, is impaired in HD mouse models and not caused by HSF1 reduction. Scientific Reports, 2021, 11, 9117.	3.3	7
2	Tau Seeds in Extracellular Vesicles Induce Tau Accumulation in Degradative Organelles of Cells. DNA and Cell Biology, 2021, 40, 1185-1199.	1.9	3
3	Emerging Evidences for an Implication of the Neurodegeneration-Associated Protein TAU in Cancer. Brain Sciences, 2020, 10, 862.	2.3	28
4	Tau affects P53 function and cell fate during the DNA damage response. Communications Biology, 2020, 3, 245.	4.4	36
5	Hijacking Endocytosis and Autophagy in Extracellular Vesicle Communication: Where the Inside Meets the Outside. Frontiers in Cell and Developmental Biology, 2020, 8, 595515.	3.7	22
6	Chloroquine, the Coronavirus Crisis, and Neurodegeneration: A Perspective. Frontiers in Neurology, 2020, 11, 596528.	2.4	2
7	Protoporphyrin IX tracer fluorescence modulation for improved brain tumor cell lines visualization. Journal of Photochemistry and Photobiology B: Biology, 2019, 201, 111640.	3.8	15
8	Phosphorylation of nuclear Tau is modulated by distinct cellular pathways. Scientific Reports, 2018, 8, 17702.	3.3	31
9	<scp>ER</scp> â€toâ€lysosomeâ€associated degradation of proteasomeâ€resistant <scp>ATZ</scp> polymers occurs via receptorâ€mediated vesicular transport. EMBO Journal, 2018, 37, .	7.8	144
10	Epithelial growth factor receptor expression influences 5-ALA induced glioblastoma fluorescence. Journal of Neuro-Oncology, 2017, 133, 497-507.	2.9	18
11	HSF1-dependent and -independent regulation of the mammalian in vivo heat shock response and its impairment in Huntington's disease mouse models. Scientific Reports, 2017, 7, 12556.	3.3	27
12	Split GFP technologies to structurally characterize and quantify functional biomolecular interactions of FTD-related proteins. Scientific Reports, 2017, 7, 14013.	3.3	31
13	Motifs in the tau protein that control binding to microtubules and aggregation determine pathological effects. Scientific Reports, 2017, 7, 13556.	3.3	35
14	Functional and dynamic polymerization of the ALS-linked protein TDP-43 antagonizes its pathologic aggregation. Nature Communications, 2017, 8, 45.	12.8	242
15	LBH589, A Hydroxamic Acid-Derived HDAC Inhibitor, is Neuroprotective in Mouse Models of Huntingtonâ€™s Disease. Journal of Huntington's Disease, 2016, 5, 347-355.	1.9	27
16	Enkephalin and dynorphin neuropeptides are differently correlated with locomotor hypersensitivity and levodopa-induced dyskinesia in parkinsonian rats. Experimental Neurology, 2016, 280, 80-88.	4.1	20
17	Synthesis and structureâ€“activity relationship of 2,6-disubstituted pyridine derivatives as inhibitors of Î²-amyloid-42 aggregation. Bioorganic and Medicinal Chemistry Letters, 2016, 26, 3330-3335.	2.2	14
18	Guidelines for the use and interpretation of assays for monitoring autophagy (3rd edition). Autophagy, 2016, 12, 1-222.	9.1	4,701

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19	BACE-1 is expressedÂin the bloodâ€“brain barrier endothelium and is upregulated in a murine model of Alzheimerâ€™s disease. Journal of Cerebral Blood Flow and Metabolism, 2016, 36, 1281-1294.	4.3	53
20	Deciphering the role of tau in neurodegeneration using Adeno-Associated Viral (AAV) vectors to express human tau in the mouse forebrain. Molecular Neurodegeneration, 2013, 8, .	10.8	0
21	Human genomeâ€“guided identification of memory-modulating drugs. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, E4369-74.	7.1	20
22	Transgenic expression of Î²1 antibody in brain neurons impairs age-dependent amyloid deposition in APP23 mice. Neurobiology of Aging, 2013, 34, 2866-2878.	3.1	4
23	Atg4b-Dependent Autophagic Flux Alleviates Huntingtonâ€™s Disease Progression. PLoS ONE, 2013, 8, e68357.	2.5	30
24	Increased Efflux of Amyloid-Î² Peptides through the Blood-Brain Barrier by Muscarinic Acetylcholine Receptor Inhibition Reduces Pathological Phenotypes in Mouse Models of Brain Amyloidosis. Journal of Alzheimer's Disease, 2013, 38, 767-786.	2.6	11
25	Discovery and Structure Activity Relationship of Small Molecule Inhibitors of Toxic Î²-Amyloid-42 Fibril Formation. Journal of Biological Chemistry, 2012, 287, 34786-34800.	3.4	53
26	Suppression of protein aggregation by chaperone modification of high molecular weight complexes. Brain, 2012, 135, 1180-1196.	7.6	103
27	Neuronal aggregates are associated with phenotypic onset in the R6/2 Huntington's disease transgenic mouse. Behavioural Brain Research, 2012, 229, 308-319.	2.2	11
28	A Screen for Enhancers of Clearance Identifies Huntingtin as a Heat Shock Protein 90 (Hsp90) Client Protein. Journal of Biological Chemistry, 2012, 287, 1406-1414.	3.4	84
29	Guidelines for the use and interpretation of assays for monitoring autophagy. Autophagy, 2012, 8, 445-544.	9.1	3,122
30	TR-FRET-Based Duplex Immunoassay Reveals an Inverse Correlation of Soluble and Aggregated Mutant huntingtin in Huntington's Disease. Chemistry and Biology, 2012, 19, 264-275.	6.0	70
31	Hdac6 Knock-Out Increases Tubulin Acetylation but Does Not Modify Disease Progression in the R6/2 Mouse Model of Huntington's Disease. PLoS ONE, 2011, 6, e20696.	2.5	91
32	Induction of autophagy with catalytic mTOR inhibitors reduces huntingtin aggregates in a neuronal cell model. Journal of Neurochemistry, 2011, 119, 398-407.	3.9	85
33	Microtiter plate quantification of mutant and wild-type huntingtin normalized to cell count. Analytical Biochemistry, 2011, 410, 304-306.	2.4	14
34	Altered chromatin architecture underlies progressive impairment of the heat shock response in mouse models of Huntington disease. Journal of Clinical Investigation, 2011, 121, 3306-3319.	8.2	151
35	Perturbation with Intrabodies Reveals That Calpain Cleavage Is Required for Degradation of Huntingtin Exon 1. PLoS ONE, 2011, 6, e16676.	2.5	27
36	Macrocyclic BACE-1 inhibitors acutely reduce AÎ² in brain after po application. Bioorganic and Medicinal Chemistry Letters, 2010, 20, 603-607.	2.2	55

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37	The mTOR kinase inhibitor Everolimus decreases S6 kinase phosphorylation but fails to reduce mutant huntingtin levels in brain and is not neuroprotective in the R6/2 mouse model of Huntington's disease. Molecular Neurodegeneration, 2010, 5, 26.	10.8	86
38	Huntingtin cleavage product A forms in neurons and is reduced by gamma-secretase inhibitors. Molecular Neurodegeneration, 2010, 5, 58.	10.8	16
39	Î²â€Secretase cleavage is not required for generation of the intracellular Câ€terminal domain of the amyloid precursor family of proteins. FEBS Journal, 2010, 277, 1503-1518.	4.7	22
40	Full-length huntingtin levels modulate body weight by influencing insulin-like growth factor 1 expression. Human Molecular Genetics, 2010, 19, 1528-1538.	2.9	100
41	Identical oligomeric and fibrillar structures captured from the brains of R6/2 and knock-in mouse models of Huntington's disease. Human Molecular Genetics, 2010, 19, 65-78.	2.9	185
42	Proteolysis of Mutant Huntingtin Produces an Exon 1 Fragment That Accumulates as an Aggregated Protein in Neuronal Nuclei in Huntington Disease. Journal of Biological Chemistry, 2010, 285, 8808-8823.	3.4	259
43	Rapid Cerebral Amyloid Binding by AÎ² Antibodies Infused into Î²-Amyloid Precursor Protein Transgenic Mice. Biological Psychiatry, 2010, 68, 971-974.	1.3	14
44	Optimization of an HTRF Assay for the Detection of Soluble Mutant Huntingtin in Human Buffy Coats: A Potential Biomarker in Blood for Huntington Disease. PLOS Currents, 2010, 2, RRN1205.	1.4	24
45	Development of a Method for the Highâ€Throughput Quantification of Cellular Proteins. ChemBioChem, 2009, 10, 1678-1688.	2.6	21
46	Inducible mutant huntingtin expression in HN10 cells reproduces Huntington's disease-like neuronal dysfunction. Molecular Neurodegeneration, 2009, 4, 11.	10.8	29
47	Structure-based design and synthesis of macrocyclic peptidomimetic Î²-secretase (BACE-1) inhibitors. Bioorganic and Medicinal Chemistry Letters, 2009, 19, 1361-1365.	2.2	35
48	Macrocyclic peptidomimetic Î²-secretase (BACE-1) inhibitors with activity in vivo. Bioorganic and Medicinal Chemistry Letters, 2009, 19, 1366-1370.	2.2	54
49	Single-step detection of mutant huntingtin in animal and human tissues: A bioassay for Huntingtonâ€™s disease. Analytical Biochemistry, 2009, 395, 8-15.	2.4	115
50	Acetylation Targets Mutant Huntingtin to Autophagosomes for Degradation. Cell, 2009, 137, 60-72.	28.9	367
51	Splicing of intron 3 of human BACE requires the flanking introns 2 and 4. Biochemical and Biophysical Research Communications, 2009, 388, 434-438.	2.1	2
52	Sensitive biochemical aggregate detection reveals aggregation onset before symptom development in cellular and murine models of Huntingtonâ€™s disease. Journal of Neurochemistry, 2008, 104, 846-858.	3.9	103
53	Consequences of Individual N-glycan Deletions and of Proteasomal Inhibition on Secretion of Active BACE. Molecular Biology of the Cell, 2008, 19, 4086-4098.	2.1	25
54	BACE1 and Mutated Presenilin-1 Differently Modulate AÎ²40 and AÎ²42 Levels and Cerebral Amyloidosis in APPDutch Transgenic Mice. Neurodegenerative Diseases, 2007, 4, 127-135.	1.4	19

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55	Exogenous Induction of Cerebral ÃŸ-Amyloidogenesis Is Governed by Agent and Host. Science, 2006, 313, 1781-1784.	12.6	875
56	EDEM1 regulates ER-associated degradation by accelerating de-mannosylation of folding-defective polypeptides and by inhibiting their covalent aggregation. Biochemical and Biophysical Research Communications, 2006, 349, 1278-1284.	2.1	154
57	Detection of a Soluble Form of BACE-1 in Human Cerebrospinal Fluid by a Sensitive Activity Assay. Clinical Chemistry, 2006, 52, 1168-1174.	3.2	66
58	Î²-site specific intrabodies to decrease and prevent generation of Alzheimer's AÎ² peptide. Journal of Cell Biology, 2005, 168, 863-868.	5.2	98
59	Structure-Based Design, Synthesis, and Memapsin 2 (BACE) Inhibitory Activity of Carbocyclic and Heterocyclic Peptidomimetics. Journal of Medicinal Chemistry, 2005, 48, 5175-5190.	6.4	74
60	EDEM Contributes to Maintenance of Protein Folding Efficiency and Secretory Capacity. Journal of Biological Chemistry, 2004, 279, 44600-44605.	3.4	40
61	Yeast growth selection system for the identification of cell-active inhibitors of Î²-secretase. Biochimica Et Biophysica Acta - General Subjects, 2004, 1674, 29-39.	2.4	20
62	BACE (Î²-secretase) modulates the processing of APLP2 in vivo. Molecular and Cellular Neurosciences, 2004, 25, 642-649.	2.2	107
63	Role of EDEM in the Release of Misfolded Glycoproteins from the Calnexin Cycle. Science, 2003, 299, 1397-1400.	12.6	431
64	Targeting Presenilin-type Aspartic Protease Signal Peptide Peptidase with Î³-Secretase Inhibitors. Journal of Biological Chemistry, 2003, 278, 16528-16533.	3.4	114
65	Sequential assistance of molecular chaperones and transient formation of covalent complexes during protein degradation from the ER. Journal of Cell Biology, 2002, 158, 247-257.	5.2	204
66	Expression of human Î²â€secretase in the mouse brain increases the steadyâ€state level of Î²â€amyloid. Journal of Neurochemistry, 2002, 80, 799-806.	3.9	116
67	The disulphide bonds in the catalytic domain of BACE are critical but not essential for amyloid precursor protein processing activity. Journal of Neurochemistry, 2002, 80, 1079-1088.	3.9	31
68	A Splice Variant of Î²-Secretase Deficient in the Amyloidogenic Processing of the Amyloid Precursor Protein. Journal of Biological Chemistry, 2001, 276, 12019-12023.	3.4	74
69	Amyloid Î² interacts with the amyloid precursor protein: a potential toxic mechanism in Alzheimer's disease. Nature Neuroscience, 2000, 3, 460-464.	14.8	252
70	Membrane-type 1 Matrix Metalloprotease (MT1-MMP) Enables Invasive Migration of Glioma Cells in Central Nervous System White Matter. Journal of Cell Biology, 1999, 144, 373-384.	5.2	232
71	Two amyloid precursor protein transgenic mouse models with Alzheimerâ€‰disease-likeâ€‰pathology. Proceedings of the National Academy of Sciences of the United States of America, 1997, 94, 13287-13292.	7.1	1,355
72	Distinct Processing of Endogenous and Overexpressed Recombinant Presenilin 1. Neurobiology of Aging, 1997, 18, 181-189.	3.1	18

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73	Effect of alkalizing agents on the processing of the Î²-amyloid precursor protein. Brain Research, 1996, 716, 91-100.	2.2	77
74	The Carboxyl Termini of Î²-Amyloid Peptides 1-40 and 1-42 Are Generated by Distinct Î³-Secretase Activities. Journal of Biological Chemistry, 1996, 271, 28655-28659.	3.4	151
75	Intracellular Accumulation of Î²-Amyloid in Cells Expressing the Swedish Mutant Amyloid Precursor Protein. Journal of Biological Chemistry, 1995, 270, 26727-26730.	3.4	88
76	Proteolytic processing of theAplysia A peptide precursor in AtT-20 cells. Brain Research, 1994, 633, 53-62.	2.2	13
77	Heterogeneous distribution of calmodulin-and cAMP-dependent regulation of Ca2+ uptake in cardiac sarcoplasmic reticulum subfracitons. FEBS Journal, 1988, 176, 535-541.	0.2	15