Berta Puig

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Anchorless risk or released benefit? An updated view on the ADAM10-mediated shedding of the prion protein. Cell and Tissue Research, 2023, 392, 215-234.	1.5	4
2	Multiplexed mRNA analysis of brain-derived extracellular vesicles upon experimental stroke in mice reveals increased mRNA content with potential relevance to inflammation and recovery processes. Cellular and Molecular Life Sciences, 2022, 79, .	2.4	6
3	A multifactorial model of pathology for age of onset heterogeneity in familial Alzheimer's disease. Acta Neuropathologica, 2021, 141, 217-233.	3.9	33
4	Brain-Derived Extracellular Vesicles in Health and Disease: A Methodological Perspective. International Journal of Molecular Sciences, 2021, 22, 1365.	1.8	17
5	Prion protein oligomers cause neuronal cytoskeletal damage in rapidly progressive Alzheimer's disease. Molecular Neurodegeneration, 2021, 16, 11.	4.4	15
6	CD73-mediated adenosine production by CD8 T cell-derived extracellular vesicles constitutes an intrinsic mechanism of immune suppression. Nature Communications, 2021, 12, 5911.	5.8	66
7	Ligands binding to the prion protein induce its proteolytic release with therapeutic potential in neurodegenerative proteinopathies. Science Advances, 2021, 7, eabj1826.	4.7	18
8	Characterization of brainâ€derived extracellular vesicles reveals changes in cellular origin after stroke and enrichment of the prion protein with a potential role in cellular uptake. Journal of Extracellular Vesicles, 2020, 9, 1809065.	5.5	47
9	Show Me Your Friends and I Tell You Who You Are: The Many Facets of Prion Protein in Stroke. Cells, 2020, 9, 1609.	1.8	6
10	Transgenic Overexpression of the Disordered Prion Protein N1 Fragment in Mice Does Not Protect Against Neurodegenerative Diseases Due to Impaired ER Translocation. Molecular Neurobiology, 2020, 57, 2812-2829.	1.9	17
11	Generation and Function of Non-cell-bound CD73 in Inflammation. Frontiers in Immunology, 2019, 10, 1729.	2.2	43
12	GPI-anchor signal sequence influences PrPC sorting, shedding and signalling, and impacts on different pathomechanistic aspects of prion disease in mice. PLoS Pathogens, 2019, 15, e1007520.	2.1	34
13	Molecular Mechanisms of Prion Diseases. , 2019, , .		0
14	Structural and mechanistic aspects influencing the ADAM10-mediated shedding of the prion protein. Molecular Neurodegeneration, 2018, 13, 18.	4.4	45
15	Molecular Communication of a Dying Neuron in Stroke. International Journal of Molecular Sciences, 2018, 19, 2834.	1.8	109
16	Amyloid-β Precursor Protein Modulates the Sorting of Testican-1 and Contributes to Its Accumulation in Brain Tissue and Cerebrospinal Fluid from Patients with Alzheimer Disease. Journal of Neuropathology and Experimental Neurology, 2016, 75, 903-916.	0.9	18
17	Misfolding leads the way to unraveling signaling pathways in the pathophysiology of prion diseases. Prion, 2016, 10, 434-443.	0.9	2
18	Secretory pathway retention of mutant prion protein induces p38-MAPK activation and lethal disease in mice. Scientific Reports, 2016, 6, 24970.	1.6	22

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19	Shedding light on prion disease. Prion, 2015, 9, 244-256.	0.9	17
20	The sheddase ADAM10 is a potent modulator of prion disease. ELife, 2015, 4, .	2.8	66
21	The GPI-anchoring of PrP. Prion, 2014, 8, 11-18.	0.9	49
22	Roles of endoproteolytic α•leavage and shedding of the prion protein in neurodegeneration. FEBS Journal, 2013, 280, 4338-4347.	2.2	48
23	Proteolytic processing of the prion protein in health and disease. American Journal of Neurodegenerative Disease, 2012, 1, 15-31.	0.1	58
24	Deposition of Hyperphosphorylated Tau in Cerebellum of PS1 E280A Alzheimer's Disease. Brain Pathology, 2011, 21, 452-463.	2.1	65
25	Lack of a-disintegrin-and-metalloproteinase ADAM10 leads to intracellular accumulation and loss of shedding of the cellular prion protein in vivo. Molecular Neurodegeneration, 2011, 6, 36.	4.4	93
26	N-Glycans and Glycosylphosphatidylinositol-Anchor Act on Polarized Sorting of Mouse PrPC in Madin-Darby Canine Kidney Cells. PLoS ONE, 2011, 6, e24624.	1.1	19
27	Lipid Alterations in Lipid Rafts from Alzheimer's Disease Human Brain Cortex. Journal of Alzheimer's Disease, 2010, 19, 489-502.	1.2	235
28	VDAC and ERα interaction in caveolae from human cortex is altered in Alzheimer's disease. Molecular and Cellular Neurosciences, 2009, 42, 172-183.	1.0	83
29	Brain Protein Preservation Largely Depends on the Postmortem Storage Temperature. Journal of Neuropathology and Experimental Neurology, 2007, 66, 35-46.	0.9	151
30	Abnormal Sp1 transcription factor expression in Alzheimer disease and tauopathies. Neuroscience Letters, 2006, 397, 30-34.	1.0	62
31	Low molecular weight species of tau in Alzheimer's disease are dependent on tau phosphorylation sites but not on delayed post-mortem delay in tissue processing. Neuroscience Letters, 2006, 399, 106-110.	1.0	18
32	Expression of transcription factors c-Fos, c-Jun, CREB-1 and ATF-2, and caspase-3 in relation with abnormal tau deposits in Pick's disease. Acta Neuropathologica, 2006, 111, 341-350.	3.9	4
33	Immediate Early Genes, Inducible Transcription Factors and Stress Kinases in Alzheimer's Disease. , 2006, , 243-260.		1
34	Individual and regional variations of phospho-tau species in progressive supranuclear palsy. Acta Neuropathologica, 2005, 110, 261-268.	3.9	15
35	Constitutive Dyrk1A is abnormally expressed in Alzheimer disease, Down syndrome, Pick disease, and related transgenic models. Neurobiology of Disease, 2005, 20, 392-400.	2.1	152
36	Accelerated amyloid deposition, neurofibrillary degeneration and neuronal loss in double mutant APP/tau transgenic mice. Neurobiology of Disease, 2005, 20, 814-822.	2.1	163

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37	Amyloid-β deposition in the cerebral cortex in Dementia with Lewy bodies is accompanied by a relative increase in AβPP mRNA isoforms containing the Kunitz protease inhibitor. Neurochemistry International, 2005, 46, 253-260.	1.9	22
38	Active stress kinase p38 enhances and perpetuates abnormal tau phosphorylation and deposition in Pick?s disease. Acta Neuropathologica, 2004, 107, 185-189.	3.9	30
39	Clusterin solubility and aggregation in Creutzfeldt-Jakob disease. Acta Neuropathologica, 2004, 108, 295-301.	3.9	49
40	Ubiquitin-negative mini-pick-like bodies in the dentate gyrus in p301l tauopathy. Journal of Alzheimer's Disease, 2004, 5, 445-454.	1.2	16
41	Phosphorylated Protein Kinases Associated with Neuronal and Glial <i>Tau</i> Deposits in Argyrophilic Grain Disease. Brain Pathology, 2003, 13, 62-78.	2.1	52
42	Caspase-3-associated apoptotic cell death in excitotoxic necrosis of the entorhinal cortex following intraperitoneal injection of kainic acid in the rat. Neuroscience Letters, 2002, 321, 182-186.	1.0	46
43	Abnormal synaptic protein expression and cell death in murine scrapie. Acta Neuropathologica, 2002, 103, 615-626.	3.9	79
44	Methylazoximethanol acetate-induced cell death in the granule cell layer of the developing mouse cerebellum is associated with caspase-3 activation, but does not depend on the tissue-type plasminogen activator. Neuroscience Letters, 2001, 299, 77-80.	1.0	10
45	Cleaved caspase-3, caspase-7 and poly (ADP-ribose) polymerase are complementarily but differentially expressed in human medulloblastomas. Neuroscience Letters, 2001, 306, 85-88.	1.0	24
46	Cell death signaling in the cerebellum in Creutzfeldt-Jakob disease. Acta Neuropathologica, 2001, 102, 207-215.	3.9	37
47	Prion protein deposition and abnormal synaptic protein expression in the cerebellum in Creutzfeldt–labob disease. Neuroscience, 2000, 97, 715-726	1.1	59