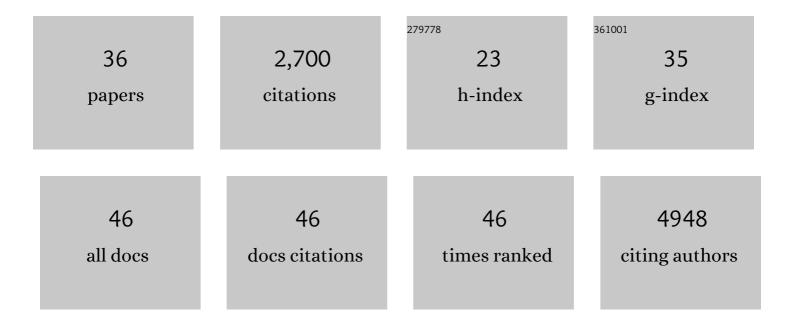
## Estibaliz Capetillo-Zarate

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Vesicular glutamate release from axons in white matter. Nature Neuroscience, 2007, 10, 311-320.	14.8	408
2	Intraneuronal β-amyloid accumulation and synapse pathology in Alzheimer's disease. Acta Neuropathologica, 2010, 119, 523-541.	7.7	341
3	Dysregulation of the mTOR Pathway Mediates Impairment of Synaptic Plasticity in a Mouse Model of Alzheimer's Disease. PLoS ONE, 2010, 5, e12845.	2.5	219
4	The Development of Amyloid beta Protein Deposits in the Aged Brain. Science of Aging Knowledge Environment: SAGE KE, 2006, 2006, re1-re1.	0.8	174
5	P2X4 receptor controls microglia activation and favors remyelination in autoimmune encephalitis. EMBO Molecular Medicine, 2018, 10, .	6.9	141
6	Effects of Synaptic Modulation on β-Amyloid, Synaptophysin, and Memory Performance in Alzheimer's Disease Transgenic Mice. Journal of Neuroscience, 2010, 30, 14299-14304.	3.6	125
7	Synaptic Activity Reduces Intraneuronal Aβ, Promotes APP Transport to Synapses, and Protects against Al²-Related Synaptic Alterations. Journal of Neuroscience, 2009, 29, 9704-9713.	3.6	119
8	Capillary cerebral amyloid angiopathy is associated with vessel occlusion and cerebral blood flow disturbances. Neurobiology of Aging, 2009, 30, 1936-1948.	3.1	116
9	Co-occurrence of Alzheimer's disease β-amyloid and tau pathologies at synapses. Neurobiology of Aging, 2010, 31, 1145-1152.	3.1	116
10	The endocytic pathway in microglia during health, aging and Alzheimer's disease. Ageing Research Reviews, 2016, 32, 89-103.	10.9	93
11	Inter-laboratory comparison of neuropathological assessments of β-amyloid protein: a study of the BrainNet Europe consortium. Acta Neuropathologica, 2008, 115, 533-546.	7.7	86
12	Degradation of Alzheimer's amyloid fibrils by microglia requires delivery of ClC-7 to lysosomes. Molecular Biology of the Cell, 2011, 22, 1664-1676.	2.1	86
13	Mangiferin and Morin Attenuate Oxidative Stress, Mitochondrial Dysfunction, and Neurocytotoxicity, Induced by Amyloid Beta Oligomers. Oxidative Medicine and Cellular Longevity, 2018, 2018, 1-13.	4.0	62
14	Contribution of Neurons and Glial Cells to Complement-Mediated Synapse Removal during Development, Aging and in Alzheimer's Disease. Mediators of Inflammation, 2018, 2018, 1-12.	3.0	54
15	Occurrence and co-localization of amyloid β-protein and apolipoprotein E in perivascular drainage channels of wild-type and APP-transgenic mice. Neurobiology of Aging, 2007, 28, 1221-1230.	3.1	53
16	Apolipoprotein E co-localizes with newly formed amyloid β-protein (Aβ) deposits lacking immunoreactivity against N-terminal epitopes of Aβ in a genotype-dependent manner. Acta Neuropathologica, 2005, 110, 459-471.	7.7	50
17	Dispersible amyloid β-protein oligomers, protofibrils, and fibrils represent diffusible but not soluble aggregates: their role in neurodegeneration in amyloid precursor protein (APP) transgenic mice. Neurobiology of Aging, 2012, 33, 2641-2660.	3.1	50
18	Accumulation of Intraneuronal β-Amyloid 42 Peptides Is Associated with Early Changes in Microtubule-Associated Protein 2 in Neurites and Synapses. PLoS ONE, 2013, 8, e51965.	2.5	48

#	Article	IF	CITATIONS
19	Transgenic Expression of Intraneuronal Aβ <sub>42</sub> But Not Aβ <sub>40</sub> Leads to Cellular Aβ Lesions, Degeneration, and Functional Impairment without Typical Alzheimer's Disease Pathology. Journal of Neuroscience, 2012, 32, 1273-1283.	3.6	44
20	Selective vulnerability of different types of commissural neurons for amyloid Â-protein-induced neurodegeneration in APP23 mice correlates with dendritic tree morphology. Brain, 2006, 129, 2992-3005.	7.6	43
21	Synaptic activity protects against AD and FTD-like pathology via autophagic-lysosomal degradation. Molecular Psychiatry, 2018, 23, 1530-1540.	7.9	39
22	Impaired β-Amyloid Secretion in Alzheimer's Disease Pathogenesis. Journal of Neuroscience, 2011, 31, 15384-15390.	3.6	35
23	Monocyte-Derived Dendritic Cells Upregulate Extracellular Catabolism of Aggregated Low-Density Lipoprotein on Maturation, Leading to Foam Cell Formation. Arteriosclerosis, Thrombosis, and Vascular Biology, 2015, 35, 2092-2103.	2.4	28
24	High-Resolution 3D Reconstruction Reveals Intra-Synaptic Amyloid Fibrils. American Journal of Pathology, 2011, 179, 2551-2558.	3.8	27
25	APP depletion alters selective pre- and post-synaptic proteins. Molecular and Cellular Neurosciences, 2019, 95, 86-95.	2.2	26
26	Intraneuronal Aß Accumulation, Amyloid Plaques, and Synapse Pathology in Alzheimer's Disease. Neurodegenerative Diseases, 2012, 10, 56-59.	1.4	21
27	Targeting Beta-Amyloid at the CSF: A New Therapeutic Strategy in Alzheimer's Disease. Frontiers in Aging Neuroscience, 2018, 10, 100.	3.4	20
28	A Neuron, Microglia, and Astrocyte Triple Co-culture Model to Study Alzheimer's Disease. Frontiers in Aging Neuroscience, 2022, 14, 844534.	3.4	18
29	Amyloid β / PKC-dependent alterations in NMDA receptor composition are detected in early stages of Alzheimer´s disease. Cell Death and Disease, 2022, 13, 253.	6.3	16
30	Tracing of temporo-entorhinal connections in the human brain: cognitively impaired argyrophilic grain disease cases show dendritic alterations but no axonal disconnection of temporo-entorhinal association neurons. Acta Neuropathologica, 2008, 115, 175-183.	7.7	13
31	Effects of Platelet-Rich Plasma on Cellular Populations of the Central Nervous System: The Influence of Donor Age. International Journal of Molecular Sciences, 2021, 22, 1725.	4.1	12
32	Whole Blood Expression Pattern of Inflammation and Redox Genes in Mild Alzheimer's Disease. Journal of Inflammation Research, 2021, Volume 14, 6085-6102.	3.5	9
33	Characterization of molecular biomarkers in cerebrospinal fluid and serum of E46K-SNCA mutation carriers. Parkinsonism and Related Disorders, 2022, 96, 29-35.	2.2	2
34	Recombinant Integrin β1 Signal Peptide Blocks Gliosis Induced by Aβ Oligomers. International Journal of Molecular Sciences, 2022, 23, 5747.	4.1	1
35	O3-05-03: SYNAPTIC ALTERATIONS IN APP KNOCKOUT NEURONS. , 2014, 10, P217-P217.		0

Polyphenols attenuate mitochondrial dysfunction induced by amyloid peptides., 2021, , 317-337.