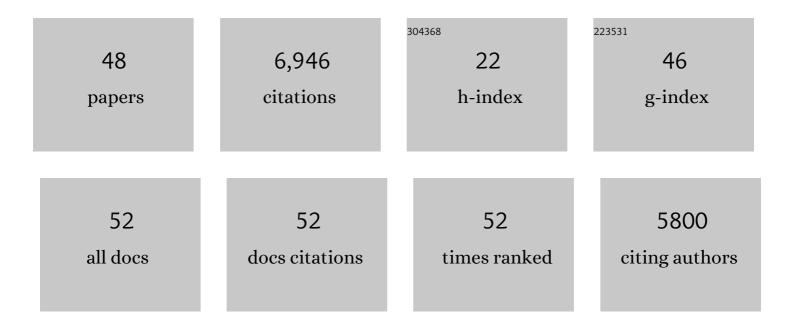
Victoria Campuzano

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Plasma sICAM-1 as a Biomarker of Carotid Plaque Inflammation in Patients with a Recent Ischemic Stroke. Translational Stroke Research, 2022, 13, 745-756.	2.3	6
2	The homeostatic role of hydrogen peroxide, superoxide anion and nitric oxide in the vasculature. Free Radical Biology and Medicine, 2021, 162, 615-635.	1.3	57
3	Co-Treatment With Verapamil and Curcumin Attenuates the Behavioral Alterations Observed in Williams–Beuren Syndrome Mice by Regulation of MAPK Pathway and Microglia Overexpression. Frontiers in Pharmacology, 2021, 12, 670785.	1.6	8
4	Anti-TGFβ (Transforming Growth Factor β) Therapy With Betaglycan-Derived P144 Peptide Gene Delivery Prevents the Formation of Aortic Aneurysm in a Mouse Model of Marfan Syndrome. Arteriosclerosis, Thrombosis, and Vascular Biology, 2021, 41, e440-e452.	1.1	12
5	Uric Acid Treatment After Stroke Prevents Long-Term Middle Cerebral Artery Remodelling and Attenuates Brain Damage in Spontaneously Hypertensive Rats. Translational Stroke Research, 2020, 11, 1332-1347.	2.3	16
6	Altered Neocortical Dynamics in a Mouse Model of Williams–Beuren Syndrome. Molecular Neurobiology, 2020, 57, 765-777.	1.9	11
7	Reactive Oxygen Species and Oxidative Stress in the Pathogenesis and Progression of Genetic Diseases of the Connective Tissue. Antioxidants, 2020, 9, 1013.	2.2	21
8	Stenosis coexists with compromised α1-adrenergic contractions in the ascending aorta of a mouse model of Williams-Beuren syndrome. Scientific Reports, 2020, 10, 889.	1.6	10
9	CD69 Plays a Beneficial Role in Ischemic Stroke by Dampening Endothelial Activation. Circulation Research, 2019, 124, 279-291.	2.0	21
10	Redox stress in Marfan syndrome: Dissecting the role of the NADPH oxidase NOX4 in aortic aneurysm. Free Radical Biology and Medicine, 2018, 118, 44-58.	1.3	57
11	Crosstalk between Peripheral Small Vessel Properties and Anxious-like Profiles: Sex, Genotype, and Interaction Effects in Mice with Normal Aging and 3×Tg-AD mice at Advanced Stages of Disease. Journal of Alzheimer's Disease, 2018, 62, 1531-1538.	1.2	6
12	Epigallocatechin-3-gallate improves cardiac hypertrophy and short-term memory deficits in a Williams-Beuren syndrome mouse model. PLoS ONE, 2018, 13, e0194476.	1.1	25
13	Treatment with Standard and Low Dose of Conjugated Equine Estrogen Differentially Modulates Estrogen Receptor Expression and Response to Angiotensin II in Mesenteric Venular Bed of Surgically Postmenopausal Hypertensive Rats. Journal of Pharmacology and Experimental Therapeutics, 2017, 362, 98-107.	1.3	6
14	Activation of α _{1A} â€adrenoceptors desensitizes the rat aorta response to phenylephrine through a neuronal NOS pathway, a mechanism lost with ageing. British Journal of Pharmacology, 2017, 174, 2015-2030.	2.7	12
15	Differences in the Thoracic Aorta by Region and Sex in a Murine Model of Marfan Syndrome. Frontiers in Physiology, 2017, 8, 933.	1.3	24
16	NADPH oxidase 4 attenuates cerebral artery changes during the progression of Marfan syndrome. American Journal of Physiology - Heart and Circulatory Physiology, 2016, 310, H1081-H1090.	1.5	13
17	Synaptic plasticity and spatial working memory are impaired in the CD mouse model of Williams-Beuren syndrome. Molecular Brain, 2016, 9, 76.	1.3	17
18	Metabolic abnormalities in Williams–Beuren syndrome. Journal of Medical Genetics, 2015, 52, 248-255.	1.5	29

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19	Intracisternal Gtf2i Gene Therapy Ameliorates Deficits in Cognition and Synaptic Plasticity of a Mouse Model of Williams–Beuren Syndrome. Molecular Therapy, 2015, 23, 1691-1699.	3.7	36
20	Heterozygous deletion of the Williams–Beuren syndrome critical interval in mice recapitulates most features of the human disorder. Human Molecular Genetics, 2014, 23, 6481-6494.	1.4	69
21	Western-style diet modulates contractile responses to phenylephrine differently in mesenteric arteries from senescence-accelerated prone (SAMP8) and resistant (SAMR1) mice. Age, 2013, 35, 1219-1234.	3.0	15
22	Western-type diet induces senescence, modifies vascular function in non-senescence mice and triggers adaptive mechanisms in senescent ones. Experimental Gerontology, 2013, 48, 1410-1419.	1.2	12
23	TFII-I regulates target genes in the PI-3K and TGF-Î ² signaling pathways through a novel DNA binding motif. Gene, 2013, 527, 529-536.	1.0	18
24	Reduction of NADPH-Oxidase Activity Ameliorates the Cardiovascular Phenotype in a Mouse Model of Williams-Beuren Syndrome. PLoS Genetics, 2012, 8, e1002458.	1.5	29
25	Middle cerebral artery alterations in a rat chronic hypoperfusion model. Journal of Applied Physiology, 2012, 112, 511-518.	1.2	21
26	Essential role of the N-terminal region of TFII-I in viability and behavior. BMC Medical Genetics, 2010, 11, 61.	2.1	35
27	Copy number variation at the 7q11.23 segmental duplications is a susceptibility factor for the Williams-Beuren syndrome deletion. Genome Research, 2008, 18, 683-694.	2.4	64
28	Protein farnesyltransferase in embryogenesis, adult homeostasis, and tumor development. Cancer Cell, 2005, 7, 313-324.	7.7	106
29	Tumor induction by an endogenous K-ras oncogene is highly dependent on cellular context. Cancer Cell, 2003, 4, 111-120.	7.7	518
30	Frataxin point mutations in two patients with Friedreich's ataxia and unusual clinical features. Journal of Neurology, Neurosurgery and Psychiatry, 2000, 68, 661-664.	0.9	47
31	Maturation of wild-type and mutated frataxin by the mitochondrial processing peptidase. Human Molecular Genetics, 1998, 7, 1485-1489.	1.4	105
32	Frataxin is Reduced in Friedreich Ataxia Patients and is Associated with Mitochondrial Membranes. Human Molecular Genetics, 1997, 6, 1771-1780.	1.4	724
33	Evolution of the Friedreich's ataxia trinucleotide repeat expansion: Founder effect and premutations. Proceedings of the National Academy of Sciences of the United States of America, 1997, 94, 7452-7457.	3.3	320
34	Frataxin fracas. Nature Genetics, 1997, 15, 337-338.	9.4	78
35	Studies of human, mouse and yeast homologues indicate a mitochondrial function for frataxin. Nature Genetics, 1997, 16, 345-351.	9.4	489
36	Le gène de l'ataxie de Friedreich: des applications diagnostiques et une controverse sans fondement Medecine/Sciences, 1997, 13, 253.	0.0	0

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37	Bases moléculaires de l'ataxie de Friedreich et de l'ataxie par déficit en vitamine E. Annales De L'Institut Pasteur / Actualités, 1996, 7, 193-198.	0.1	0
38	Friedreich's Ataxia: Autosomal Recessive Disease Caused by an Intronic GAA Triplet Repeat Expansion. Science, 1996, 271, 1423-1427.	6.0	2,642
39	Blue-Light Receptor Requirement for Gravitropism, Autochemotropism and Ethylene Response in Phycomyces. Photochemistry and Photobiology, 1996, 63, 686-694.	1.3	53
40	Clinical and Genetic Abnormalities in Patients with Friedreich's Ataxia. New England Journal of Medicine, 1996, 335, 1169-1175.	13.9	1,015
41	Ataxie de Friedreich : les expansions de triplets frappent encore. Medecine/Sciences, 1996, 12, 431.	0.0	1
42	Isolation, characterization and transformation, by autonomous replication, ofMucor circinelloides OMPdecase-deficient mutants. Molecular Genetics and Genomics, 1995, 248, 126-135.	2.4	45
43	Genetic characterization of two phototropism mutants of Phycomyces with defects in the genes madl and madJ. Current Genetics, 1995, 27, 524-527.	0.8	17
44	The Friedreich ataxia critical region spans a 150-kb interval on chromosome 9q13. American Journal of Human Genetics, 1995, 57, 1061-7.	2.6	26
45	Isolation and characterization of phototropism mutants of Phycomyces insensitive to ultraviolet light. Current Genetics, 1994, 26, 49-53.	0.8	20
46	Isolation, characterization and mapping of pyrimidine auxotrophs of Phycomyces blakesleeanus. Current Genetics, 1993, 24, 515-519.	0.8	8
47	Cloning and sequence analysis of the Mucor circinelloides pyrG gene encoding orotidine-5′-monophosphate decarâ ylase: use of pyrG for homologous transformation. Gene, 1992, 116, 59-67.	1.0	64
48	A new gene (madl) involved in the phototropic response of Phycomyces. Molecular Genetics and Genomics, 1990, 223, 148-151.	2.4	16