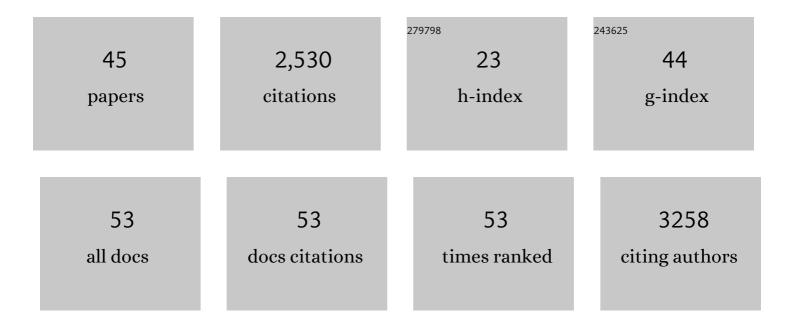
Nadia Soussi-Yanicostas

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Zebrafish as a Model for Neurological Disorders. International Journal of Molecular Sciences, 2022, 23, 4321.	4.1	6
2	Bixafen, a succinate dehydrogenase inhibitor fungicide, causes microcephaly and motor neuron axon defects during development. Chemosphere, 2021, 265, 128781.	8.2	18
3	Diisopropylfluorophosphate-induced status epilepticus drives complex glial cell phenotypes in adult male mice. Neurobiology of Disease, 2021, 152, 105276.	4.4	11
4	SDHI Fungicide Toxicity and Associated Adverse Outcome Pathways: What Can Zebrafish Tell Us?. International Journal of Molecular Sciences, 2021, 22, 12362.	4.1	26
5	Organophosphorus diisopropylfluorophosphate (DFP) intoxication in zebrafish larvae causes behavioral defects, neuronal hyperexcitation and neuronal death. Scientific Reports, 2020, 10, 19228.	3.3	11
6	A Fast, Simple, and Affordable Technique to Measure Oxygen Consumption in Living Zebrafish Embryos. Zebrafish, 2020, 17, 268-270.	1.1	3
7	Defective Excitatory/Inhibitory Synaptic Balance and Increased Neuron Apoptosis in a Zebrafish Model of Dravet Syndrome. Cells, 2019, 8, 1199.	4.1	28
8	Decreased microglial Wnt/β-catenin signalling drives microglial pro-inflammatory activation in the developing brain. Brain, 2019, 142, 3806-3833.	7.6	97
9	Neurons Expressing Pathological Tau Protein Trigger Dramatic Changes in Microglial Morphology and Dynamics. Frontiers in Neuroscience, 2019, 13, 1199.	2.8	15
10	Oxytocin receptor agonist reduces perinatal brain damage by targeting microglia. Glia, 2019, 67, 345-359.	4.9	65
11	Altered vaccineâ€induced immunity in children with Dravet syndrome. Epilepsia, 2018, 59, e45-e50.	5.1	15
12	Surfen and oxalyl surfen decrease tau hyperphosphorylation and mitigate neuron deficits in vivo in a zebrafish model of tauopathy. Translational Neurodegeneration, 2018, 7, 6.	8.0	26
13	Heparan Sulfate as a Therapeutic Target in Tauopathies: Insights From Zebrafish. Frontiers in Cell and Developmental Biology, 2018, 6, 163.	3.7	30
14	γâ€Aminobutyric acid receptor alpha 1 subunit loss of function causes genetic generalized epilepsy by impairing inhibitory network neurodevelopment. Epilepsia, 2018, 59, 2061-2074.	5.1	65
15	Non-canonical mTOR-Independent Role of DEPDC5 in Regulating GABAergic Network Development. Current Biology, 2018, 28, 1924-1937.e5.	3.9	47
16	A Rapid and Efficient Method of Identifying G0 Males with Mosaic Germ Line Cells. Zebrafish, 2016, 13, 535-536.	1.1	0
17	Tau Hyperphosphorylation and Oxidative Stress, a Critical Vicious Circle in Neurodegenerative Tauopathies?. Oxidative Medicine and Cellular Longevity, 2015, 2015, 1-17.	4.0	193
18	HS3ST2 expression is critical for the abnormal phosphorylation of tau in Alzheimer's disease-related tau pathology. Brain, 2015, 138, 1339-1354.	7.6	75

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19	ZEB2 zinc-finger missense mutations lead to hypomorphic alleles and a mild Mowat–Wilson syndrome. Human Molecular Genetics, 2013, 22, 2652-2661.	2.9	51
20	Spatacsin and spastizin act in the same pathway required for proper spinal motor neuron axon outgrowth in zebrafish. Neurobiology of Disease, 2012, 48, 299-308.	4.4	42
21	Localization and characterization of kal 1.a and kal 1.b in the brain of adult zebrafish (Danio rerio). Brain Research Bulletin, 2012, 88, 345-353.	3.0	8
22	Requirement for Zebrafish Ataxin-7 in Differentiation of Photoreceptors and Cerebellar Neurons. PLoS ONE, 2012, 7, e50705.	2.5	32
23	Developmental aspects of respiratory chain from fetus to infancy. Seminars in Fetal and Neonatal Medicine, 2011, 16, 175-180.	2.3	8
24	Dynamic roles of FGF-2 and Anosmin-1 in the migration of neuronal precursors from the subventricular zone during pre- and postnatal development. Experimental Neurology, 2010, 222, 285-295.	4.1	47
25	Prokineticin 2 Expression Is Associated with Neural Repair of Injured Adult Zebrafish Telencephalon. Journal of Neurotrauma, 2010, 27, 959-972.	3.4	58
26	Prokineticin receptor 2 expression identifies migrating neuroblasts and their subventricular zone transientâ€amplifying progenitors in adult mice. Journal of Comparative Neurology, 2009, 512, 232-242.	1.6	41
27	Anosmin-1a is required for fasciculation and terminal targeting of olfactory sensory neuron axons in the zebrafish olfactory system. Molecular and Cellular Endocrinology, 2009, 312, 53-60.	3.2	39
28	A novel role for anosminâ€l in the adhesion and migration of oligodendrocyte precursors. Developmental Neurobiology, 2008, 68, 1503-1516.	3.0	45
29	Expression pattern of Anosminâ€1 during pre―and postnatal rat brain development. Developmental Dynamics, 2008, 237, 2518-2528.	1.8	22
30	Essential requirement for zebrafish anosmin-1a in the migration of the posterior lateral line primordium. Developmental Biology, 2008, 320, 469-479.	2.0	25
31	Localization of anosmin-1a and anosmin-1b in the inner ear and neuromasts of zebrafish. Gene Expression Patterns, 2007, 7, 274-281.	0.8	12
32	FGFR1 and anosmin-1 underlying genetically distinct forms of Kallmann syndrome are co-expressed and interact in olfactory bulbs. Development Genes and Evolution, 2007, 217, 169-175.	0.9	33
33	Anosmin-1 modulates the FGF-2-dependent migration of oligodendrocyte precursors in the developing optic nerve. Molecular and Cellular Neurosciences, 2006, 33, 2-14.	2.2	81
34	Anosmin-1 immunoreactivity during embryogenesis in a primitive eutherian mammal. Developmental Brain Research, 2003, 140, 157-167.	1.7	28
35	Loss-of-function mutations in FGFR1 cause autosomal dominant Kallmann syndrome. Nature Genetics, 2003, 33, 463-465.	21.4	764
36	Anosmin-1, Defective in the X-Linked Form of Kallmann Syndrome, Promotes Axonal Branch Formation from Olfactory Bulb Output Neurons. Cell, 2002, 109, 217-228.	28.9	201

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37	Anosmin-1 is a regionally restricted component of basement membranes and interstitial matrices during organogenesis: Implications for the developmental anomalies of X chromosome-linked Kallmann syndrome. Developmental Dynamics, 1999, 215, 26-44.	1.8	143
38	Biochemical and immunocytochemical analysis in chronic proximal spinal muscular atrophy. Muscle and Nerve, 1994, 17, 400-410.	2.2	8
39	Expression of myosin isoforms and of desmin, vimentin and titin in Tunisian Duchenne-like autosomal recessive muscular dystrophy. Journal of the Neurological Sciences, 1994, 123, 114-121.	0.6	4
40	Five skeletal myosin heavy chain genes are organized as a multigene complex in the human genome. Human Molecular Genetics, 1993, 2, 563-569.	2.9	22
41	Evolution of muscle specific proteins in Werdnig-Hoffman's disease. Journal of the Neurological Sciences, 1992, 109, 111-120.	0.6	19
42	Distinct contractile protein profile in congenital myotonic dystrophy and X-linked myotubular myopathy. Neuromuscular Disorders, 1991, 1, 103-111.	0.6	30
43	Biphasic expression of slow myosin light chains and slow tropomyosin isoforms during the development of the human quadriceps muscle. FEBS Letters, 1991, 280, 292-296.	2.8	12
44	Transcription of the embryonic myosin light chain gene is restricted to type II muscle fibers in human adult masseter. Developmental Biology, 1991, 147, 374-380.	2.0	7
45	Modification in the expression and localization of contractile and cytoskeletal proteins in Schwartz-Jampel syndrome. Journal of the Neurological Sciences, 1991, 104, 64-73.	0.6	12