

Owain W Howell

List of Publications by Year in descending order

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Version: 2024-02-01

47
papers

7,448
citations

159585

30
h-index

223800

46
g-index

50
all docs

50
docs citations

50
times ranked

7088
citing authors

#	ARTICLE	IF	CITATIONS
1	The association between neurodegeneration and local complement activation in the thalamus to progressive multiple sclerosis outcome. <i>Brain Pathology</i> , 2022, 32, e13054.	4.1	13
2	â€œEpendymalâ€â€ Gradient of Thalamic Damage in Progressive Multiple Sclerosis. <i>Annals of Neurology</i> , 2022, 92, 670-685.	5.3	15
3	Visualizing Cholesterol in the Brain by On-Tissue Derivatization and Quantitative Mass Spectrometry Imaging. <i>Analytical Chemistry</i> , 2021, 93, 4932-4943.	6.5	38
4	Metabolic profiling in serum, cerebrospinal fluid, and brain of patients with cerebrotendinous xanthomatosis. <i>Journal of Lipid Research</i> , 2021, 62, 100078.	4.2	14
5	CCN3 is dynamically regulated by treatment and disease state in multiple sclerosis. <i>Journal of Neuroinflammation</i> , 2020, 17, 349.	7.2	8
6	Unacylated-Chrelin Impairs Hippocampal Neurogenesis and Memory in Mice and Is Altered in Parkinsonâ€™s Dementia in Humans. <i>Cell Reports Medicine</i> , 2020, 1, 100120.	6.5	15
7	B cell rich meningeal inflammation associates with increased spinal cord pathology in multiple sclerosis. <i>Brain Pathology</i> , 2020, 30, 779-793.	4.1	76
8	Localization of sterols and oxysterols in mouse brain reveals distinct spatial cholesterol metabolism. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2020, 117, 5749-5760.	7.1	53
9	Neuroinflammation in the normal-appearing white matter (NAWM) of the multiple sclerosis brain causes abnormalities at the nodes of Ranvier. <i>PLoS Biology</i> , 2020, 18, e3001008.	5.6	28
10	B cell rich meningeal inflammation associates with increased spinal cord pathology in multiple sclerosis. <i>Brain Pathology</i> , 2020, 30, 779-793.	4.1	8
11	Substantial subpial cortical demyelination in progressive multiple sclerosis: have we underestimated the extent of cortical pathology?. <i>Neuroimmunology and Neuroinflammation</i> , 2020, , .	1.4	3
12	Using biomarkers to predict clinical outcomes in multiple sclerosis. <i>Practical Neurology</i> , 2019, 19, 342-349.	1.1	5
13	Calorie restriction activates new adult born olfactoryâ€bulb neurones in a ghrelinâ€dependent manner but acylâ€ghrelin does not enhance subventricular zone neurogenesis. <i>Journal of Neuroendocrinology</i> , 2019, 31, e12755.	2.6	14
14	Measurement of soluble CD59 in CSF in demyelinating disease: Evidence for an intrathecal source of soluble CD59. <i>Multiple Sclerosis Journal</i> , 2019, 25, 523-531.	3.0	9
15	Inflammatory intrathecal profiles and cortical damage in multiple sclerosis. <i>Annals of Neurology</i> , 2018, 83, 739-755.	5.3	219
16	Tissue microarray methodology identifies complement pathway activation and dysregulation in progressive multiple sclerosis. <i>Brain Pathology</i> , 2018, 28, 507-520.	4.1	31
17	Meningeal inflammation and cortical demyelination in acute multiple sclerosis. <i>Annals of Neurology</i> , 2018, 84, 829-842.	5.3	96
18	Complement is activated in progressive multiple sclerosis cortical grey matter lesions. <i>Journal of Neuroinflammation</i> , 2016, 13, 161.	7.2	101

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19	Extensive grey matter pathology in the cerebellum in multiple sclerosis is linked to inflammation in the subarachnoid space. <i>Neuropathology and Applied Neurobiology</i> , 2015, 41, 798-813.	3.2	82
20	Complement activation in multiple sclerosis plaques: an immunohistochemical analysis. <i>Acta Neuropathologica Communications</i> , 2014, 2, 53.	5.2	124
21	Cortical grey matter demyelination can be induced by elevated pro-inflammatory cytokines in the subarachnoid space of MOG-immunized rats. <i>Brain</i> , 2013, 136, 3596-3608.	7.6	125
22	GLRB is the third major gene of effect in hyperekplexia. <i>Human Molecular Genetics</i> , 2013, 22, 927-940.	2.9	50
23	New Hyperekplexia Mutations Provide Insight into Glycine Receptor Assembly, Trafficking, and Activation Mechanisms. <i>Journal of Biological Chemistry</i> , 2013, 288, 33745-33759.	3.4	35
24	Polymorphisms in Neuropsychiatric and Neuroinflammatory Disorders and the Role of Next Generation Sequencing in Early Diagnosis and Treatment. <i>Advances in Protein Chemistry and Structural Biology</i> , 2012, 89, 85-116.	2.3	2
25	Meningeal inflammation plays a role in the pathology of primary progressive multiple sclerosis. <i>Brain</i> , 2012, 135, 2925-2937.	7.6	310
26	The neuropathological basis of clinical progression in multiple sclerosis. <i>Acta Neuropathologica</i> , 2011, 122, 155-170.	7.7	188
27	Related B cell clones populate the meninges and parenchyma of patients with multiple sclerosis. <i>Brain</i> , 2011, 134, 534-541.	7.6	186
28	Meningeal inflammation is widespread and linked to cortical pathology in multiple sclerosis. <i>Brain</i> , 2011, 134, 2755-2771.	7.6	685
29	Activated Microglia Mediate Axoglial Disruption That Contributes to Axonal Injury in Multiple Sclerosis. <i>Journal of Neuropathology and Experimental Neurology</i> , 2010, 69, 1017-1033.	1.7	190
30	A Gradient of neuronal loss and meningeal inflammation in multiple sclerosis. <i>Annals of Neurology</i> , 2010, 68, 477-493.	5.3	588
31	NPY augments the proliferative effect of FGF2 and increases the expression of FGFR1 on nestin positive postnatal hippocampal precursor cells, via the Y1 receptor. <i>Journal of Neurochemistry</i> , 2010, 113, 615-627.	3.9	20
32	Two Binding Sites for [³ H]PBR28 in Human Brain: Implications for TSPO PET Imaging of Neuroinflammation. <i>Journal of Cerebral Blood Flow and Metabolism</i> , 2010, 30, 1608-1618.	4.3	187
33	HDAC1 nuclear export induced by pathological conditions is essential for the onset of axonal damage. <i>Nature Neuroscience</i> , 2010, 13, 180-189.	14.8	188
34	Detection of Epstein-Barr virus and B-cell follicles in the multiple sclerosis brain: what you find depends on how and where you look. <i>Brain</i> , 2010, 133, e157-e157.	7.6	66
35	Expression and Function of Junctional Adhesion Molecule-C in Myelinated Peripheral Nerves. <i>Science</i> , 2007, 318, 1472-1475.	12.6	55
36	NPY mediates basal and seizure-induced proliferation in the subcallosal zone. <i>NeuroReport</i> , 2007, 18, 1005-1008.	1.2	13

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37	Neuropeptide Y is important for basal and seizure-induced precursor cell proliferation in the hippocampus. <i>Neurobiology of Disease</i> , 2007, 26, 174-188.	4.4	96
38	The junctional adhesion molecule (JAM) α is required for maintaining the integrity and function of myelinated peripheral nerves. <i>FASEB Journal</i> , 2007, 21, A65.	0.5	0
39	Disruption of neurofascin localization reveals early changes preceding demyelination and remyelination in multiple sclerosis. <i>Brain</i> , 2006, 129, 3173-3185.	7.6	167
40	Meningeal B-cell follicles in secondary progressive multiple sclerosis associate with early onset of disease and severe cortical pathology. <i>Brain</i> , 2006, 130, 1089-1104.	7.6	1,142
41	Neuropeptide α Y stimulates neuronal precursor proliferation in the postnatal and adult dentate gyrus. <i>Journal of Neurochemistry</i> , 2005, 93, 560-570.	3.9	174
42	Neuropeptide Y is neuroproliferative for post-natal hippocampal precursor cells. <i>Journal of Neurochemistry</i> , 2003, 86, 646-659.	3.9	166
43	Enhanced Learning and Memory and Altered GABAergic Synaptic Transmission in Mice Lacking the α 5 Subunit of the GABA _A Receptor. <i>Journal of Neuroscience</i> , 2002, 22, 5572-5580.	3.6	591
44	Loss of the Major GABA _A Receptor Subtype in the Brain Is Not Lethal in Mice. <i>Journal of Neuroscience</i> , 2001, 21, 3409-3418.	3.6	215
45	Sedative but not anxiolytic properties of benzodiazepines are mediated by the GABA _A receptor α 1 subtype. <i>Nature Neuroscience</i> , 2000, 3, 587-592.	14.8	898
46	Changes in [3H]zolpidem and [3H]Ro 15-1788 binding in rat globus pallidus and substantia nigra pars reticulata following a nigrostriatal tract lesion. <i>Brain Research</i> , 2000, 862, 280-283.	2.2	13
47	Autoradiographic localization of α 5 subunit-containing GABA _A receptors in rat brain. <i>Brain Research</i> , 1999, 822, 265-270.	2.2	145