James P O'callaghan

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	A role for neuroimmune signaling in a rat model of Gulf War Illness-related pain. Brain, Behavior, and Immunity, 2021, 91, 418-428.	2.0	14
2	Modeling Neuroimmune Interactions in Human Subjects and Animal Models to Predict Subtype-Specific Multidrug Treatments for Gulf War Illness. International Journal of Molecular Sciences, 2021, 22, 8546.	1.8	9
3	The β-adrenergic receptor blocker and anti-inflammatory drug propranolol mitigates brain cytokine expression in a long-term model of Gulf War Illness. Life Sciences, 2021, 285, 119962.	2.0	6
4	Alterations in high-order diffusion imaging in veterans with Gulf War Illness is associated with chemical weapons exposure and mild traumatic brain injury. Brain, Behavior, and Immunity, 2020, 89, 281-290.	2.0	17
5	Exploring the Role of Chemokine Receptor 6 (Ccr6) in the BXD Mouse Model of Gulf War Illness. Frontiers in Neuroscience, 2020, 14, 818.	1.4	4
6	Genome-wide transcriptome architecture in a mouse model of Gulf War Illness. Brain, Behavior, and Immunity, 2020, 89, 209-223.	2.0	13
7	Modeling the Genetic Basis of Individual Differences in Susceptibility to Gulf War Illness. Brain Sciences, 2020, 10, 143.	1.1	11
8	Microglial activation and responses to vasculature that result from an acute LPS exposure. NeuroToxicology, 2020, 77, 181-192.	1.4	30
9	Acetylcholinesterase inhibitor exposures as an initiating factor in the development of Gulf War Illness, a chronic neuroimmune disorder in deployed veterans. Neuropharmacology, 2020, 171, 108073.	2.0	34
10	Oligodendrocyte involvement in Gulf War Illness. Glia, 2019, 67, 2107-2124.	2.5	17
11	Neuroinflammation disorders exacerbated by environmental stressors. Metabolism: Clinical and Experimental, 2019, 100, 153951.	1.5	35
12	Astrocyteâ€specific transcriptome analysis using the ALDH1L1 bacTRAP mouse reveals novel biomarkers of astrogliosis in response to neurotoxicity. Journal of Neurochemistry, 2019, 150, 420-440.	2.1	18
13	Glial Reactivity in Response to Neurotoxins: Relevance and Methods. Neuromethods, 2019, , 51-67.	0.2	1
14	Corticosterone and pyridostigmine/DEET exposure attenuate peripheral cytokine expression: Supporting a dominant role for neuroinflammation in a mouse model of Gulf War Illness. NeuroToxicology, 2019, 70, 26-32.	1.4	35
15	Epigenetic impacts of stress priming of the neuroinflammatory response to sarin surrogate in mice: a model of Gulf War illness. Journal of Neuroinflammation, 2018, 15, 86.	3.1	47
16	Corticosterone potentiates DFP-induced neuroinflammation and affects high-order diffusion imaging in a rat model of Gulf War Illness. Brain, Behavior, and Immunity, 2018, 67, 42-46.	2.0	66
17	The Multiple Hit Hypothesis for Gulf War Illness: Self-Reported Chemical/Biological Weapons Exposure and Mild Traumatic Brain Injury. Brain Sciences, 2018, 8, 198.	1.1	34
18	The Neuroinflammatory Phenotype in a Mouse Model of Gulf War Illness is Unrelated to Brain Regional Levels of Acetylcholine as Measured by Quantitative HILIC-UPLC-MS/MS. Toxicological Sciences, 2018, 165, 302-313.	1.4	31

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19	A Logic Model of Neuronal-Glial Interaction Suggests Altered Homeostatic Regulation in the Perpetuation of Neuroinflammation. Frontiers in Cellular Neuroscience, 2018, 12, 336.	1.8	10
20	Prior exposure to corticosterone markedly enhances and prolongs the neuroinflammatory response to systemic challenge with LPS. PLoS ONE, 2018, 13, e0190546.	1.1	35
21	New horizons for focused ultrasound (FUS) – therapeutic applications in neurodegenerative diseases. Metabolism: Clinical and Experimental, 2017, 69, S3-S7.	1.5	31
22	The combined effects of 3,4-methylenedioxymethamphetamine (MDMA) and selected substituted methcathinones on measures of neurotoxicity. Neurotoxicology and Teratology, 2017, 61, 74-81.	1.2	24
23	Corticosterone primes the neuroinflammatory response to Gulf War Illnessâ€relevant organophosphates independently of acetylcholinesterase inhibition. Journal of Neurochemistry, 2017, 142, 444-455.	2.1	77
24	Corticosterone and exogenous glucose alter blood glucose levels, neurotoxicity, and vascular toxicity produced by methamphetamine. Journal of Neurochemistry, 2017, 143, 198-213.	2.1	18
25	Advancing the Role of Neuroimmunity and Genetic Susceptibility in Gulf War Illness. EBioMedicine, 2017, 26, 11-12.	2.7	8
26	Supporting a Neuroimmune Basis of Gulf War Illness. EBioMedicine, 2016, 13, 5-6.	2.7	23
27	Vascular-directed responses of microglia produced by methamphetamine exposure: indirect evidence that microglia are involved in vascular repair?. Journal of Neuroinflammation, 2016, 13, 64.	3.1	21
28	Recent research on Gulf War illness and other health problems in veterans of the 1991 Gulf War: Effects of toxicant exposures during deployment. Cortex, 2016, 74, 449-475.	1.1	326
29	Corticosterone primes the neuroinflammatory response to <scp>DFP</scp> in mice: potential animal model of Gulf War Illness. Journal of Neurochemistry, 2015, 133, 708-721.	2.1	133
30	Translational Biomarkers of Neurotoxicity: A Health and Environmental Sciences Institute Perspective on the Way Forward. Toxicological Sciences, 2015, 148, 332-340.	1.4	43
31	Biomarkers of Parkinson's disease: Present and future. Metabolism: Clinical and Experimental, 2015, 64, S40-S46.	1.5	284
32	Early Activation of STAT3 Regulates Reactive Astrogliosis Induced by Diverse Forms of Neurotoxicity. PLoS ONE, 2014, 9, e102003.	1.1	114
33	Genetic correlational analysis reveals no association between MPP+ and the severity of striatal dopaminergic damage following MPTP treatment in BXD mouse strains. Neurotoxicology and Teratology, 2014, 45, 91-92.	1.2	3
34	SN79, a sigma receptor antagonist, attenuates methamphetamine-induced astrogliosis through a blockade of OSMR/gp130 signaling and STAT3 phosphorylation. Experimental Neurology, 2014, 254, 180-189.	2.0	47
35	Health assessment of gasoline and fuel oxygenate vapors: Neurotoxicity evaluation. Regulatory Toxicology and Pharmacology, 2014, 70, S35-S42.	1.3	24
36	Health assessment of gasoline and fuel oxygenate vapors: Reproductive toxicity assessment. Regulatory Toxicology and Pharmacology, 2014, 70, S48-S57.	1.3	20

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37	Systems analysis of genetic variation in MPTP neurotoxicity in mice. NeuroToxicology, 2013, 37, 26-34.	1.4	23
38	Chronic exposure to corticosterone enhances the neuroinflammatory and neurotoxic responses to methamphetamine. Journal of Neurochemistry, 2012, 122, 995-1009.	2.1	66
39	Early Alterations of Brain Cellular Energy Homeostasis in Huntington Disease Models. Journal of Biological Chemistry, 2012, 287, 1361-1370.	1.6	104
40	Organophosphates dysregulate dopamine signaling, glutamatergic neurotransmission, and induce neuronal injury markers in striatum. Journal of Neurochemistry, 2011, 119, 303-313.	2.1	82
41	Gestational lead exposure selectively decreases retinal dopamine amacrine cells and dopamine content in adult mice. Toxicology and Applied Pharmacology, 2011, 256, 258-267.	1.3	21
42	Effects of Repeated Treatment with Phosphodiesterase-4 Inhibitors on cAMP Signaling, Hippocampal Cell Proliferation, and Behavior in the Forced-Swim Test. Journal of Pharmacology and Experimental Therapeutics, 2011, 338, 641-647.	1.3	36
43	Astrogliosis in CNS Pathologies: Is There A Role for Microglia?. Molecular Neurobiology, 2010, 41, 232-241.	1.9	252
44	Spinal glia and chronic pain. Metabolism: Clinical and Experimental, 2010, 59, S21-S26.	1.5	43
45	Indirubins deplete striatal monoamines in the Intact and MPTP-treated mouse brain and block kainate-induced striatal astrogliosis. Neurotoxicology and Teratology, 2010, 32, 212-219.	1.2	16
46	Nerve agent exposure elicits site-specific changes in protein phosphorylation in mouse brain. Brain Research, 2010, 1342, 11-23.	1.1	22
47	Protracted exposure to supraphysiological levels of corticosterone does not cause neuronal loss or damage and protects against kainic acid-induced neurotoxicity in the hippocampus of C57BL/6J mice. NeuroToxicology, 2009, 30, 965-976.	1.4	5
48	Mild steel welding fume causes manganese accumulation and subtle neuroinflammatory changes but not overt neuronal damage in discrete brain regions of rats after short-term inhalation exposure. NeuroToxicology, 2009, 30, 915-925.	1.4	51
49	Defining "Neuroinflammation― Annals of the New York Academy of Sciences, 2008, 1139, 318-330.	1.8	122
50	AMP-activated protein kinase phosphorylation in brain is dependent on method of killing and tissue preparation. Journal of Neurochemistry, 2008, 105, 833-841.	2.1	31
51	Autoantibodies to neurotypic and gliotypic proteins as biomarkers of neurotoxicity: Assessment of trimethyltin (TMT). NeuroToxicology, 2008, 29, 109-115.	1.4	21
52	Distinct Roles of PDE4 and PDE10A in the Regulation of cAMP/PKA Signaling in the Striatum. Journal of Neuroscience, 2008, 28, 10460-10471.	1.7	257
53	Low-Level Human Equivalent Gestational Lead Exposure Produces Supernormal Scotopic Electroretinograms, Increased Retinal Neurogenesis, and Decreased Retinal Dopamine Utilization in Rats. Environmental Health Perspectives, 2008, 116, 618-625.	2.8	33
54	Recapitulation of cell signaling events associated with astrogliosis using the brain slice preparation. Journal of Neurochemistry, 2007, 100, 720-726.	2.1	20

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55	Divergent Roles for Tumor Necrosis Factor-α in the Brain. Journal of NeuroImmune Pharmacology, 2007, 2, 140-153.	2.1	196
56	Mechanism of age-dependent susceptibility and novel treatment strategy in glutaric acidemia type I. Journal of Clinical Investigation, 2007, 117, 3258-3270.	3.9	92
57	Deficiency of TNF receptors suppresses microglial activation and alters the susceptibility of brain regions to MPTPâ€induced neurotoxicity: role of TNFâ€Î± 1. FASEB Journal, 2006, 20, 670-682.	0.2	213
58	Development of an animal model to study the potential neurotoxic effects associated with welding fume inhalation. NeuroToxicology, 2006, 27, 745-751.	1.4	11
59	Minocycline attenuates microglial activation but fails to mitigate striatal dopaminergic neurotoxicity: role of tumor necrosis factor-alpha. Journal of Neurochemistry, 2006, 96, 706-718.	2.1	238
60	Calcium/calmodulin-dependent protein kinase II activity and expression are altered in the hippocampus of Pb2+-exposed rats. Brain Research, 2005, 1044, 51-58.	1.1	38
61	Microscale sample deposition onto hydrophobic target plates for trace level detection of neuropeptides in brain tissue by MALDI-MS. Journal of Mass Spectrometry, 2005, 40, 1338-1346.	0.7	28
62	Associations of cortical astrogliosis with cognitive performance and dementia status. Journal of Alzheimer's Disease, 2005, 6, 595-604.	1.2	90
63	Glial fibrillary acidic protein and related glial proteins as biomarkers of neurotoxicity. Expert Opinion on Drug Safety, 2005, 4, 433-442.	1.0	216
64	Depression, cytokines, and glial function. Metabolism: Clinical and Experimental, 2005, 54, 33-38.	1.5	64
65	Induction of gp130-related Cytokines and Activation of JAK2/STAT3 Pathway in Astrocytes Precedes Up-regulation of Glial Fibrillary Acidic Protein in the 1-Methyl-4-phenyl-1,2,3,6-tetrahydropyridine Model of Neurodegeneration. Journal of Biological Chemistry, 2004, 279, 19936-19947.	1.6	229
66	Brain concentrations of d-MDMA are increased after stress. Psychopharmacology, 2004, 173, 278-286.	1.5	46
67	Focused microwave irradiation of the brain preserves in vivo protein phosphorylation: comparison with other methods of sacrifice and analysis of multiple phosphoproteins. Journal of Neuroscience Methods, 2004, 135, 159-168.	1.3	99
68	Neurotoxic esterase: not so toxic?. Nature Genetics, 2003, 33, 437-438.	9.4	12
69	Mice deficient in TNF receptors are protected against dopaminergic neurotoxicity: Implications for Parkinson's disease. FASEB Journal, 2002, 16, 1474-1476.	0.2	340
70	Measurement of Glial Fibrillary Acidic Protein. Current Protocols in Toxicology / Editorial Board, Mahin D Maines (editor-in-chief) [et Al], 2002, 11, Unit12.8.	1.1	19
71	Neuroendocrine aspects of the response to stress. Metabolism: Clinical and Experimental, 2002, 51, 5-10.	1.5	400
72	Chronic treatment with supraphysiological levels of corticosterone enhances d-MDMA-induced dopaminergic neurotoxicity in the C57BL/6J female mouse. Brain Research, 2002, 933, 130-138.	1.1	38

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73	Prior exposure to a behaviorally sensitizing regimen of d-methamphetamine does not alter the striatal dopaminergic damage induced by a neurotoxic regimen. Addiction Biology, 2000, 5, 361-367.	1.4	1
74	Chronic dopaminergic signaling in the basal ganglia: a damage perspective on kinases and fos-related antigens. Addiction Biology, 2000, 5, 369-376.	1.4	1
75	Age as a Susceptibility Factor in the Striatal Dopaminergic Neurotoxicity Observed in the Mouse following Substituted Amphetamine Exposure. Annals of the New York Academy of Sciences, 2000, 914, 194-207.	1.8	31
76	Protein Phosphorylation Cascades Associated with Methamphetamineâ€induced Glial Activation. Annals of the New York Academy of Sciences, 2000, 914, 238-262.	1.8	81
77	Quantitative Immunoblots of Proteins Resolved from Brain Homogenates: Underestimation of Specific Protein Concentration and of Treatment Effects. Analytical Biochemistry, 1999, 274, 18-26.	1.1	31
78	The Impact of Gender and Estrogen on Striatal Dopaminergic Neurotoxicity. Annals of the New York Academy of Sciences, 1998, 844, 153-165.	1.8	236
79	A direct comparison of GFAP immunocytochemistry and GFAP concentration in various regions of ethanol-fixed rat and mouse brain. Journal of Neuroscience Methods, 1995, 58, 181-192.	1.3	49
80	Quantitative Features of Reactive Gliosis following Toxicant-induced Damage of the CNS. Annals of the New York Academy of Sciences, 1993, 679, 195-210.	1.8	126
81	The concentration of glial fibrillary acidic protein increases with age in the mouse and rat brain. Neurobiology of Aging, 1991, 12, 171-174.	1.5	141
82	Quantification of glial fibrillary acidic protein: Comparison of slot-immunobinding assays with a novel sandwich ELISA. Neurotoxicology and Teratology, 1991, 13, 275-281.	1.2	149
83	Diethyldithiocarbamate Potentiates the Neurotoxicity of In Vivo I-Methyl-4-Phenyl-1, 2, 3, 6-Tetrahydropyridine and of In Vitro 1-Methyl-4-Phenylpyridinium. Journal of Neurochemistry, 1991, 57, 541-549.	2.1	52
84	Glucocorticoids Regulate the Synthesis of Glial Fibrillary Acidic Protein in Intact and Adrenalectomized Rats but Do Not Affect Its Expression Following Brain Injury. Journal of Neurochemistry, 1991, 57, 860-869.	2.1	127
85	The Use of Glial Fibrillary Acidic Protein in First-Tier Assessments of Neurotoxicity. Journal of the American College of Toxicology, 1991, 10, 719-726.	0.2	13
86	1-Methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP)- induced damage of striatal dopaminergic fibers attenuates subsequent astrocyte response to MPTP. Neuroscience Letters, 1990, 117, 228-233.	1.0	28
87	Characterization of the origins of astrocyte response to injury using the dopaminergic neurotoxicant, 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine. Brain Research, 1990, 521, 73-80.	1.1	142
88	The neurotoxicant MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine) increases glial fibrillary acidic protein and decreases dopamine levels of the mouse striatum: Evidence for glial response to injury. Neuroscience Letters, 1988, 95, 246-251.	1.0	57
89	A method for dissection of discrete regions of rat brain following microwave irradiation. Brain Research Bulletin, 1983, 11, 31-42.	1.4	6
90	Neurotoxic Effects of Substituted Amphetamines in Rats and Mice: Challenges to the Current Dogma. , 0, , 269-302.		23

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CITATIONS

ARTICLE

The astrocyte response to neural injury. , 0, , 233-266.