

Braden C Mcfarland

List of Publications by Year in descending order

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Version: 2024-02-01

26
papers

1,576
citations

489802

18
h-index

759306

22
g-index

29
all docs

29
docs citations

29
times ranked

3208
citing authors

#	ARTICLE	IF	CITATIONS
1	Glioma and the gut-brain axis: opportunities and future perspectives. <i>Neuro-Oncology Advances</i> , 2022, 4, vdac054.	0.4	10
2	Human gut microbial communities dictate efficacy of anti-PD-1 therapy in a humanized microbiome mouse model of glioma. <i>Neuro-Oncology Advances</i> , 2021, 3, vdab023.	0.4	10
3	Changes in the gut microbiome community of nonhuman primates following radiation injury. <i>BMC Microbiology</i> , 2021, 21, 93.	1.3	35
4	An individualized mosaic of maternal microbial strains is transmitted to the infant gut microbial community. <i>Royal Society Open Science</i> , 2020, 7, 192200.	1.1	24
5	Reactive astrocytes foster brain metastases via STAT3 signaling. <i>Annals of Translational Medicine</i> , 2019, 7, S83-S83.	0.7	10
6	Protein kinase CK2 is important for the function of glioblastoma brain tumor initiating cells. <i>Journal of Neuro-Oncology</i> , 2017, 132, 219-229.	1.4	24
7	Attenuation of PKR-like ER Kinase (PERK) Signaling Selectively Controls Endoplasmic Reticulum Stress-induced Inflammation Without Compromising Immunological Responses. <i>Journal of Biological Chemistry</i> , 2016, 291, 15830-15840.	1.6	68
8	Loss of SOCS3 in myeloid cells prolongs survival in a syngeneic model of glioma. <i>Oncotarget</i> , 2016, 7, 20621-20635.	0.8	23
9	Loss of tumor suppressive microRNA-31 enhances TRADD/NF- κ B signaling in glioblastoma. <i>Oncotarget</i> , 2015, 6, 17805-17816.	0.8	43
10	TMIC-17LOSS OF SOCS3 IN MYELOID CELLS PROMOTES A DECREASED M2 MACROPHAGE PHENOTYPE AND AN INCREASED CYTOTOXIC T-CELL RESPONSE IN A SYNGENEIC MODEL OF GLIOMA. <i>Neuro-Oncology</i> , 2015, 17, v218.5-v218.	0.6	0
11	SOCS3 Deficiency in Myeloid Cells Promotes Tumor Development: Involvement of STAT3 Activation and Myeloid-Derived Suppressor Cells. <i>Cancer Immunology Research</i> , 2015, 3, 727-740.	1.6	54
12	Protein Kinase CK2 and Dysregulated Oncogenic Inflammatory Signaling Pathways. , 2015, , 259-280.		2
13	Abstract A25: SOCS3 deficiency in myeloid cells promotes prostate tumor development. , 2015, , .		0
14	Therapeutic Efficacy of Suppressing the JAK/STAT Pathway in Multiple Models of Experimental Autoimmune Encephalomyelitis. <i>Journal of Immunology</i> , 2014, 192, 59-72.	0.4	122
15	NF- κ B and STAT3 in glioblastoma: therapeutic targets coming of age. <i>Expert Review of Neurotherapeutics</i> , 2014, 14, 1293-1306.	1.4	89
16	Involvement of the Janus Kinase/Signal Transducer and Activator of Transcription Signaling Pathway in Multiple Sclerosis and the Animal Model of Experimental Autoimmune Encephalomyelitis. <i>Journal of Interferon and Cytokine Research</i> , 2014, 34, 577-588.	0.5	85
17	Therapeutic CK2 inhibition attenuates diverse prosurvival signaling cascades and decreases cell viability in human breast cancer cells. <i>Oncotarget</i> , 2014, 5, 6484-6496.	0.8	68
18	Targeting Protein Kinase CK2 Suppresses Prosurvival Signaling Pathways and Growth of Glioblastoma. <i>Clinical Cancer Research</i> , 2013, 19, 6484-6494.	3.2	124

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19	Activation of the NF- κ B Pathway by the STAT3 Inhibitor JSI-124 in Human Glioblastoma Cells. <i>Molecular Cancer Research</i> , 2013, 11, 494-505.	1.5	51
20	NF- κ B-Induced IL-6 Ensures STAT3 Activation and Tumor Aggressiveness in Glioblastoma. <i>PLoS ONE</i> , 2013, 8, e78728.	1.1	118
21	The role of Rac proteins in glioblastoma stem cells.. <i>Journal of Clinical Oncology</i> , 2013, 31, e13011-e13011.	0.8	0
22	Therapeutic Potential of AZD1480 for the Treatment of Human Glioblastoma. <i>Molecular Cancer Therapeutics</i> , 2011, 10, 2384-2393.	1.9	81
23	Plasminogen Kringle 5 Induces Apoptosis of Brain Microvessel Endothelial Cells: Sensitization by Radiation and Requirement for GRP78 and LRP1. <i>Cancer Research</i> , 2009, 69, 5537-5545.	0.4	46
24	New molecular targets in angiogenic vessels of glioblastoma tumours. <i>Expert Reviews in Molecular Medicine</i> , 2008, 10, e23.	1.6	62
25	HEF1 is a necessary and specific downstream effector of FAK that promotes the migration of glioblastoma cells. <i>Oncogene</i> , 2006, 25, 1721-1732.	2.6	173
26	New concepts regarding focal adhesion kinase promotion of cell migration and proliferation. <i>Journal of Cellular Biochemistry</i> , 2006, 99, 35-52.	1.2	254