Markus Kipp

List of Publications by Year
in descending order

		109321	95266
94	5,108	35	68
papers	citations	h-index	g-index

95	95	95	6100
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Cuprizone Intoxication Results in Myelin Vacuole Formation. Frontiers in Cellular Neuroscience, 2022, 16, 709596.	3.7	4
2	Transmembrane protein 119 is neither a specific nor a reliable marker for microglia. Glia, 2022, 70, 1170-1190.	4.9	33
3	The Role of Glial Cells in Regulating Feeding Behavior: Potential Relevance to Anorexia Nervosa. Journal of Clinical Medicine, 2022, 11, 186.	2.4	10
4	Spontaneous Hind Limb Paralysis Due to Acute Precursor B Cell Leukemia in RAG1-deficient Mice. Journal of Molecular Neuroscience, 2022, 72, 1646-1655.	2.3	1
5	Expression of Ectonucleoside Triphosphate Diphosphohydrolase 2 (NTPDase2) Is Negatively Regulated Under Neuroinflammatory Conditions<i>In Vivo</i>and<i>In Vitro</i>. ASN Neuro, 2022, 14, 175909142211020.	2.7	2
6	Different Methods for Evaluating Microglial Activation Using Anti-Ionized Calcium-Binding Adaptor Protein-1 Immunohistochemistry in the Cuprizone Model. Cells, 2022, 11, 1723.	4.1	18
7	Cuprizoneâ€induced demyelination triggers a <scp>CD8</scp>â€pronounced T cell recruitment. Glia, 2021, 69, 925-942.	4.9	24
8	Interleukin-17 and Th17 Lymphocytes Directly Impair Motoneuron Survival of Wildtype and FUS-ALS Mutant Human iPSCs. International Journal of Molecular Sciences, 2021, 22, 8042.	4.1	19
9	Focal white matter lesions induce long-lasting axonal degeneration, neuroinflammation and behavioral deficits. Neurobiology of Disease, 2021, 155, 105371.	4.4	4
10	What Guides Peripheral Immune Cells into the Central Nervous System?. Cells, 2021, 10, 2041.	4.1	10
11	Ectopic lymphoid follicles in progressive multiple sclerosis: From patients to animal models. Immunology, 2021, 164, 450-466.	4.4	18
12	Impact of the COVID-19 Pandemic on the Acceptance and Use of an E-Learning Platform. International Journal of Environmental Research and Public Health, 2021, 18, 11372.	2.6	6
13	Oligodendrocyte Lineage Marker Expression in eGFP-GFAP Transgenic Mice. Journal of Molecular Neuroscience, 2021, 71, 2237-2248.	2.3	8
14	Laquinimod ameliorates secondary brain inflammation. Neurobiology of Disease, 2020, 134, 104675.	4.4	11
15	Aquaporin-4 Expression during Toxic and Autoimmune Demyelination. Cells, 2020, 9, 2187.	4.1	14
16	CD44 expression in the cuprizone model. Brain Research, 2020, 1745, 146950.	2.2	3
17	Does Siponimod Exert Direct Effects in the Central Nervous System?. Cells, 2020, 9, 1771.	4.1	24
18	Oligodendrocyte Physiology and Pathology Function. Cells, 2020, 9, 2078.	4.1	15

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19	The Cuprizone Model: Dos and Do Nots. Cells, 2020, 9, 843.	4.1	82
20	G-Protein-Coupled Receptor Gpr17 Expression in Two Multiple Sclerosis Remyelination Models. Molecular Neurobiology, 2019, 56, 1109-1123.	4.0	27
21	Blocking Inflammasome Activation Caused by Î²-Amyloid Peptide (AÎ²) and Islet Amyloid Polypeptide (IAPP) through an IAPP Mimic. ACS Chemical Neuroscience, 2019, 10, 3703-3717.	3.5	16
22	Laquinimod Supports Remyelination in Non-Supportive Environments. Cells, 2019, 8, 1363.	4.1	13
23	Mechanism of Siponimod: Anti-Inflammatory and Neuroprotective Mode of Action. Cells, 2019, 8, 24.	4.1	73
24	Stereological Investigation of Regional Brain Volumes after Acute and Chronic Cuprizone-Induced Demyelination. Cells, 2019, 8, 1024.	4.1	6
25	Water-Soluble Cuprizone Derivative: Synthesis, Characterization, and in Vitro Studies. ACS Omega, 2019, 4, 1685-1689.	3.5	6
26	Expression of Translocator Protein and [18F]-GE180 Ligand Uptake in Multiple Sclerosis Animal Models. Cells, 2019, 8, 94.	4.1	32
27	Continuous cuprizone intoxication allows active experimental autoimmune encephalomyelitis induction in C57BL/6 mice. Histochemistry and Cell Biology, 2019, 152, 119-131.	1.7	11
28	Oligodendrocyte degeneration and concomitant microglia activation directs peripheral immune cells into the forebrain. Neurochemistry International, 2019, 126, 139-153.	3.8	17
29	Animal Weight Is an Important Variable for Reliable Cuprizone-Induced Demyelination. Journal of Molecular Neuroscience, 2019, 68, 522-528.	2.3	12
30	High Speed Ventral Plane Videography as a Convenient Tool to Quantify Motor Deficits during Pre-Clinical Experimental Autoimmune Encephalomyelitis. Cells, 2019, 8, 1439.	4.1	19
31	Mitochondrial Impairment in Oligodendroglial Cells Induces Cytokine Expression and Signaling. Journal of Molecular Neuroscience, 2019, 67, 265-275.	2.3	13
32	Evaluation strategy to determine reliable demyelination in the cuprizone model. Metabolic Brain Disease, 2019, 34, 681-685.	2.9	10
33	Visualization of the Breakdown of the Axonal Transport Machinery: a Comparative Ultrastructural and Immunohistochemical Approach. Molecular Neurobiology, 2019, 56, 3984-3998.	4.0	12
34	Cuprizoneâ€induced graded oligodendrocyte vulnerability is regulated by the transcription factor DNA damageâ€inducible transcript 3. Glia, 2019, 67, 263-276.	4.9	31
35	Effect of Intrastriatal 6-OHDA Lesions on Extrastriatal Brain Structures in the Mouse. Molecular Neurobiology, 2018, 55, 4240-4252.	4.0	24
36	Phosphatidylcholine 36:1 concentration decreasesÂalong with demyelination in the cuprizone animal model and in postâ€mortem multiple sclerosis brain tissue. Journal of Neurochemistry, 2018, 145, 504-515.	3.9	15

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37	Toll-Like Receptor 2-Mediated Glial Cell Activation in a Mouse Model of Cuprizone-Induced Demyelination. Molecular Neurobiology, 2018, 55, 6237-6249.	4.0	22
38	Do pre-clinical multiple sclerosis models allow us to measure neurodegeneration and clinical progression?. Expert Review of Neurotherapeutics, 2018, 18, 351-353.	2.8	5
39	Estrogen Attenuates Local Inflammasome Expression and Activation after Spinal Cord Injury. Molecular Neurobiology, 2018, 55, 1364-1375.	4.0	98
40	Chemical hypoxiaâ€induced integrated stress response activation in oligodendrocytes is mediated by the transcription factor nuclear factor (erythroidâ€derived 2)â€like 2 (<scp>NRF</scp>2). Journal of Neurochemistry, 2018, 144, 285-301.	3.9	14
41	An Extensible Semantic Search Engine for Biomedical Publications. , 2018, , .		2
42	Lack of chemokine (C-C motif) ligand 3 leads to decreased survival and reduced immune response after bacterial meningitis. Cytokine, 2018, 111, 246-254.	3.2	7
43	Identification of highly connected hub genes in the protective response program of human macrophages and microglia activated by alpha Bâ€crystallin. Glia, 2017, 65, 460-473.	4.9	16
44	Cuprizone-Containing Pellets Are Less Potent to Induce Consistent Demyelination in the Corpus Callosum of C57BL/6 Mice. Journal of Molecular Neuroscience, 2017, 61, 617-624.	2.3	32
45	Combination of cuprizone and experimental autoimmune encephalomyelitis to study inflammatory brain lesion formation and progression. Glia, 2017, 65, 1900-1913.	4.9	56
46	Neurofilament light as an immune target for pathogenic antibodies. Immunology, 2017, 152, 580-588.	4.4	14
47	Synaptophysin Is a Reliable Marker for Axonal Damage. Journal of Neuropathology and Experimental Neurology, 2017, 76, 109-125.	1.7	61
48	Design-Based Stereology for Evaluation of Histological Parameters. Journal of Molecular Neuroscience, 2017, 61, 325-342.	2.3	13
49	Multiple sclerosis animal models: a clinical and histopathological perspective. Brain Pathology, 2017, 27, 123-137.	4.1	174
50	ViLiP â€” A visual literature research platform for biomedical publications. , 2017, , .		1
51	Cuprizone as a model of myelin and axonal damage. Drug Discovery Today: Disease Models, 2017, 25-26, 63-68.	1.2	6
52	Thalamus Degeneration and Inflammation in Two Distinct Multiple Sclerosis Animal Models. Journal of Molecular Neuroscience, 2016, 60, 102-114.	2.3	24
53	Acute axonal damage in three different murine models of multiple sclerosis: A comparative approach. Brain Research, 2016, 1650, 125-133.	2.2	38
54	Pentraxinâ€3 is upregulated in the central nervous system during MS and EAE, but does not modulate experimental neurological disease. European Journal of Immunology, 2016, 46, 701-711.	2.9	22

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55	Absence of CCL2 and CCL3 Ameliorates Central Nervous System Grey Matter But Not White Matter Demyelination in the Presence of an Intact Bloodâ€"Brain Barrier. Molecular Neurobiology, 2016, 53, 1551-1564.	4.0	29
56	Activation and Regulation of NLRP3 Inflammasome by Intrathecal Application of SDF-1a in a Spinal Cord Injury Model. Molecular Neurobiology, 2016, 53, 3063-3075.	4.0	129
57	Neurodegeneration Triggers Peripheral Immune Cell Recruitment into the Forebrain. Journal of Neuroscience, 2016, 36, 1410-1415.	3.6	59
58	Oxidative stress in multiple sclerosis: Central and peripheral mode of action. Experimental Neurology, 2016, 277, 58-67.	4.1	230
59	Remyelination strategies in multiple sclerosis: a critical reflection. Expert Review of Neurotherapeutics, 2016, 16, 1-3.	2.8	23
60	Induction of regulatory T cells in Th1-/Th17-driven experimental autoimmune encephalomyelitis by zinc administration. Journal of Nutritional Biochemistry, 2016, 29, 116-123.	4.2	69
61	Female sex steroids and glia cells: Impact on multiple sclerosis lesion formation and fine tuning of the local neurodegenerative cellular network. Neuroscience and Biobehavioral Reviews, 2016, 67, 125-136.	6.1	28
62	Lesion Expansion in Experimental Demyelination Animal Models and Multiple Sclerosis Lesions. Molecular Neurobiology, 2016, 53, 4905-4917.	4.0	13
63	Thalamus pathology in multiple sclerosis: from biology to clinical application. Cellular and Molecular Life Sciences, 2015, 72, 1127-1147.	5.4	54
64	CXCL10 Triggers Early Microglial Activation in the Cuprizone Model. Journal of Immunology, 2015, 194, 3400-3413.	0.8	115
65	Extracellular ATP Selectively Upregulates Ecto-Nucleoside Triphosphate Diphosphohydrolase 2 and Ecto-5â€²-Nucleotidase by Rat Cortical Astrocytes In Vitro. Journal of Molecular Neuroscience, 2015, 57, 452-462.	2.3	32
66	Anatomical Distribution of Cuprizone-Induced Lesions in C57BL6 Mice. Journal of Molecular Neuroscience, 2015, 57, 166-175.	2.3	73
67	Short-Term Cuprizone Feeding Verifies N-Acetylaspartate Quantification as a Marker of Neurodegeneration. Journal of Molecular Neuroscience, 2015, 55, 733-748.	2.3	20
68	Oligodendrocyteâ€microglia crossâ€talk in the central nervous system. Immunology, 2014, 141, 302-313.	4.4	299
69	Short-Term Cuprizone Feeding Induces Selective Amino Acid Deprivation with Concomitant Activation of an Integrated Stress Response in Oligodendrocytes. Cellular and Molecular Neurobiology, 2013, 33, 1087-1098.	3.3	51
70	Astrocytes regulate myelin clearance through recruitment of microglia during cuprizone-induced demyelination. Brain, 2013, 136, 147-167.	7.6	298
71	Cuprizone-Induced Demyelination as a Tool to Study Remyelination and Axonal Protection. Journal of Molecular Neuroscience, 2013, 51, 567-572.	2.3	79
72	Endogeneous Remyelination: Findings in Human Studies. CNS and Neurological Disorders - Drug Targets, 2012, 11, 598-609.	1.4	31

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73	Pathology of Multiple Sclerosis. CNS and Neurological Disorders - Drug Targets, 2012, 11, 506-517.	1.4	57
74	In Vitro and In Vivo Models of Multiple Sclerosis. CNS and Neurological Disorders - Drug Targets, 2012, 11, 570-588.	1.4	119
75	Clusters of activated microglia in normal-appearing white matter show signs of innate immune activation. Journal of Neuroinflammation, 2012, 9, 156.	7.2	153
76	Myelin debris regulates inflammatory responses in an experimental demyelination animal model and multiple sclerosis lesions. Glia, 2012, 60, 1468-1480.	4.9	131
77	Experimental in vivo and in vitro models of multiple sclerosis: EAE and beyond. Multiple Sclerosis and Related Disorders, 2012, 1, 15-28.	2.0	78
78	Multiple sclerosis: Neuroprotective alliance of estrogenâ€"progesterone and gender. Frontiers in Neuroendocrinology, 2012, 33, 1-16.	5.2	73
79	Phagocytosis of neuronal debris by microglia is associated with neuronal damage in multiple sclerosis. Glia, 2012, 60, 422-431.	4.9	85
80	Gonadal steroids prevent cell damage and stimulate behavioral recovery after transient middle cerebral artery occlusion in male and female rats. Brain, Behavior, and Immunity, 2011, 25, 715-726.	4.1	119
81	Glial Amyloid Precursor Protein Expression is Restricted to Astrocytes in an Experimental Toxic Model of Multiple Sclerosis. Journal of Molecular Neuroscience, 2011, 43, 268-274.	2.3	23
82	Sulforaphane suppresses LPS-induced inflammation in primary rat microglia. Inflammation Research, 2010, 59, 443-450.	4.0	116
83	ADAM12 is expressed by astrocytes during experimental demyelination. Brain Research, 2010, 1326, 1-14.	2.2	29
84	TTC staining of damaged brain areas after MCA occlusion in the rat does not constrict quantitative gene and protein analyses. Journal of Neuroscience Methods, 2010, 187, 84-89.	2.5	93
85	Combined Application of 17-Estradiol and Progesterone Enhance Vascular Endothelial Growth Factor and Surfactant Protein Expression in Cultured Embryonic Lung Cells of Mice. International Journal of Pediatrics (United Kingdom), 2009, 2009, 1-8.	0.8	23
86	Impact of sex steroids on neuroinflammatory processes and experimental multiple sclerosis. Frontiers in Neuroendocrinology, 2009, 30, 188-200.	5.2	97
87	Cuprizone effect on myelination, astrogliosis and microglia attraction in the mouse basal ganglia. Brain Research, 2009, 1305, 137-149.	2.2	69
88	17Î²â€estradiol and progesterone prevent cuprizone provoked demyelination of corpus callosum in male mice. Glia, 2009, 57, 807-814.	4.9	175
89	Cuprizone treatment induces demyelination and astrocytosis in the mouse hippocampus. Journal of Neuroscience Research, 2009, 87, 1343-1355.	2.9	96
90	The cuprizone animal model: new insights into an old story. Acta Neuropathologica, 2009, 118, 723-736.	7.7	415

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91	Cuprizone Treatment Induces Distinct Demyelination, Astrocytosis, and Microglia Cell Invasion or Proliferation in the Mouse Cerebellum. Cerebellum, 2009, 8, 163-174.	2.5	95
92	Selective regulation of growth factor expression in cultured cortical astrocytes by neuro-pathological toxins. Neurochemistry International, 2009, 55, 610-618.	3.8	32
93	Brain-Region-Specific Astroglial Responses In Vitro After LPS Exposure. Journal of Molecular Neuroscience, 2008, 35, 235-243.	2.3	77
94	Estrogen and the development and protection of nigrostriatal dopaminergic neurons: Concerted action of a multitude of signals, protective molecules, and growth factors. Frontiers in Neuroendocrinology, 2006, 27, 376-390.	5.2	73