Michael J May

List of Publications by Year
in descending order

76 papers	12,508 citations	53789 45 h-index	79691 73 g-index
76 all docs	76 docs citations	76 times ranked	14952 citing authors

#	Article	IF	Citations
1	Analysis of Calcium Control of Canonical NF-Î¹B Signaling in B Lymphocytes. Methods in Molecular Biology, 2021, 2366, 145-164.	0.9	2
2	Lymph node formation and B cell homeostasis require IKK-Î± in distinct endothelial cellâ€“derived compartments. Proceedings of the National Academy of Sciences of the United States of America, 2021, 118, .	7.1	1
3	4-1BB costimulation promotes CAR T cell survival through noncanonical NF-Î¹B signaling. Science Signaling, 2020, 13, .	3.6	115
4	BCR-Induced Ca2+ Signals Dynamically Tune Survival, Metabolic Reprogramming, and Proliferation of Naive B Cells. Cell Reports, 2020, 31, 107474.	6.4	54
5	STIM- and Orai-mediated calcium entry controls NF-Î¹B activity and function in lymphocytes. Cell Calcium, 2018, 74, 131-143.	2.4	61
6	4-1BB-Costimulated CAR-Mediated Non-Canonical NF-Kb Signaling Enhances CAR T Cell Survival and Suppresses Bim Expression. Blood, 2018, 132, 3713-3713.	1.4	1
7	T Cell Receptor-induced Nuclear Factor Î¹B (NF-Î¹B) Signaling and Transcriptional Activation Are Regulated by STIM1- and Orai1-mediated Calcium Entry. Journal of Biological Chemistry, 2016, 291, 8440-8452.	3.4	55
8	NF-kappa B. Methods in Molecular Biology, 2015, 1280, v-viii.	0.9	1
9	Understanding high endothelial venules: Lessons for cancer immunology. OncoImmunology, 2015, 4, e1008791.	4.6	70
10	Epithelial-intrinsic IKKÎ± expression regulates group 3 innate lymphoid cell responses and antibacterial immunity. Journal of Experimental Medicine, 2015, 212, 1513-1528.	8.5	79
11	Stable Reconstitution of IKK-Deficient Mouse Embryonic Fibroblasts. Methods in Molecular Biology, 2015, 1280, 181-195.	0.9	1
12	NEMO-Binding Domain Peptide Inhibition of Inflammatory Signal-Induced NF-Î¹B Activation In Vivo. Methods in Molecular Biology, 2015, 1280, 505-525.	0.9	5
13	Sneaking-Ligand Fusion Proteins Attenuate Serum Transfer Arthritis by Endothelium-Targeted NF-Î¹B Inhibition. Methods in Molecular Biology, 2015, 1280, 579-591.	0.9	8
14	Noncanonical NF-Î¹B Activation and SDF-1 Expression in Human Endothelial Cells. Methods in Molecular Biology, 2015, 1280, 155-180.	0.9	3
15	A Phase I Clinical Trial of Systemically Delivered NEMO Binding Domain Peptide in Dogs with Spontaneous Activated B-Cell like Diffuse Large B-Cell Lymphoma. PLoS ONE, 2014, 9, e95404.	2.5	39
16	Triggering ubiquitination of <scp>IFNAR</scp> 1 protects tissues from inflammatory injury. EMBO Molecular Medicine, 2014, 6, 384-397.	6.9	52
17	Noncanonical NF-Î¹B Signaling Is Limited by Classical NF-Î¹B Activity. Science Signaling, 2014, 7, ra13.	3.6	49
18	Negative feedback regulation of NF-Î¹B-inducing kinase is proteasome-dependent but does not require cellular inhibitors of apoptosis. Biochemical and Biophysical Research Communications, 2014, 450, 341-346.	2.1	7

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19	NF-ÎºB inhibitor targeted to activated endothelium demonstrates a critical role of endothelial NF-ÎºB in immune-mediated diseases. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 16556-16561.	7.1	77
20	Atypical mechanism of NF-ÎºB activation by TRE17/ubiquitin-specific protease 6 (USP6) oncogene and its requirement in tumorigenesis. Oncogene, 2012, 31, 3525-3535.	5.9	58
21	IL-17R signaling: new players get in on the Act1. Nature Immunology, 2011, 12, 813-815.	14.5	23
22	The NFÎºB paradox: RelB induces and inhibits gene expression. Cell Cycle, 2011, 10, 6-7.	2.6	19
23	A Critical Role for SOCS3 in Innate Resistance to Toxoplasma gondii. Cell Host and Microbe, 2011, 10, 224-236.	11.0	69
24	The RET/PTC3 oncogene activates classical NF-ÎºB by stabilizing NIK. Oncogene, 2011, 30, 87-96.	5.9	29
25	NEMO-Binding Domain Peptide Inhibits Constitutive NF-ÎºB Activity and Reduces Tumor Burden in a Canine Model of Relapsed, Refractory Diffuse Large B-Cell Lymphoma. Clinical Cancer Research, 2011, 17, 4661-4671.	7.0	48
26	Requirement of FADD, NEMO, and BAX/BAK for Aberrant Mitochondrial Function in Tumor Necrosis Factor Alpha-Induced Necrosis. Molecular and Cellular Biology, 2011, 31, 3745-3758.	2.3	97
27	Classical NF-ÎºB Activation Negatively Regulates Noncanonical NF-ÎºB-dependent CXCL12 Expression. Journal of Biological Chemistry, 2010, 285, 38069-38077.	3.4	39
28	Cutting Edge: Association with IÎºB Kinase Î² Regulates the Subcellular Localization of Homer3. Journal of Immunology, 2010, 185, 2665-2669.	0.8	7
29	Constitutive noncanonical NFÎºB signaling in pancreatic cancer cells. Cancer Biology and Therapy, 2009, 8, 1567-1576.	3.4	74
30	NEMO-binding Domains of Both IKKÎ± and IKKÎ² Regulate IÎºB Kinase Complex Assembly and Classical NF-ÎºB Activation. Journal of Biological Chemistry, 2009, 284, 27596-27608.	3.4	40
31	Inhibiting Proinflammatory NF-ÎºB Signaling Using Cell-Penetrating NEMO Binding Domain Peptides. Methods in Molecular Biology, 2009, 512, 209-232.	0.9	13
32	Cell penetrating peptide inhibitors of Nuclear Factor-kappa B. Cellular and Molecular Life Sciences, 2008, 65, 3564-3591.	5.4	62
33	The IÎºB kinase complex: master regulator of NF-ÎºB signaling. Immunologic Research, 2008, 42, 3-18.	2.9	216
34	DNA double-strand breaks activate a multi-functional genetic program in developing lymphocytes. Nature, 2008, 456, 819-823.	27.8	137
35	Hypomorphic nuclear factor-ÎºB essential modulator mutation database and reconstitution system identifies phenotypic and immunologic diversity. Journal of Allergy and Clinical Immunology, 2008, 122, 1169-1177.e16.	2.9	240
36	NFAT Binding and Regulation of T Cell Activation by the Cytoplasmic Scaffolding Homer Proteins. Science, 2008, 319, 476-481.	12.6	100

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37	Raising the price of platinum: Inhibition of NF-Î¹B in human tumor epithelial cells. Cancer Biology and Therapy, 2008, 7, 1415-1417.	3.4	1
38	Lymphotoxin-Î±1Î²2 and LIGHT Induce Classical and Noncanonical NF-Î¹B-Dependent Proinflammatory Gene Expression in Vascular Endothelial Cells. Journal of Immunology, 2008, 180, 3467-3477.	0.8	71
39	Strong Neuroprotection by Inhibition of NF-Î¹B After Neonatal Hypoxia-Ischemia Involves Apoptotic Mechanisms but Is Independent of Cytokines. Stroke, 2008, 39, 2129-2137.	2.0	112
40	A Dual Role of the NF-Î¹B Pathway in Neonatal Hypoxic-Ischemic Brain Damage. Stroke, 2008, 39, 2578-2586.	2.0	101
41	Interleukin-1-induced NF-Î¹B Activation Is NEMO-dependent but Does Not Require IKKÎ². Journal of Biological Chemistry, 2007, 282, 8724-8733.	3.4	75
42	Caspase Inhibition Sensitizes Inhibitor of NF-Î¹B Kinase Î²-deficient Fibroblasts to Caspase-independent Cell Death via the Generation of Reactive Oxygen Species. Journal of Biological Chemistry, 2007, 282, 16105-16116.	3.4	39
43	Inhibition of Nuclear Factor-Î¹B Ameliorates Bowel Injury and Prolongs Survival in a Neonatal Rat Model of Necrotizing Enterocolitis. Pediatric Research, 2007, 61, 716-721.	2.3	84
44	NKp30 Ligation Induces Rapid Activation of the Canonical NF-Î¹B Pathway in NK Cells. Journal of Immunology, 2007, 179, 7385-7396.	0.8	29
45	G Protein-Coupled Receptor Ca²⁺-Linked Mitochondrial Reactive Oxygen Species Are Essential for Endothelial/Leukocyte Adherence. Molecular and Cellular Biology, 2007, 27, 7582-7593.	2.3	45
46	171 Intestinal Epithelial Cell-derived TSLP Regulates DC and CD4 T Cell Responses in the Gastrointestinal Tract. Cytokine, 2007, 39, 47.	3.2	0
47	Noncanonical NF-Î¹B signaling in dendritic cells is required for indoleamine 2,3-dioxygenase (IDO) induction and immune regulation. Blood, 2007, 110, 1540-1549.	1.4	143
48	Epithelial-cell-intrinsic IKK-Î² expression regulates intestinal immune homeostasis. Nature, 2007, 446, 552-556.	27.8	479
49	Local treatment with the selective IkappaB kinase beta inhibitor NEMO-binding domain peptide ameliorates synovial inflammation. Arthritis Research and Therapy, 2006, 8, R86.	3.5	69
50	Lipopolysaccharide induces CXCL2/macrophage inflammatory protein-2 gene expression in enterocytes via NF-kappaB activation: independence from endogenous TNF-alpha and platelet-activating factor. Immunology, 2006, 118, 153-163.	4.4	66
51	Selective Inhibition of Nuclear Factor-Î¹B Activation After Hypoxia/Ischemia in Neonatal Rats Is Not Neuroprotective. Pediatric Research, 2006, 59, 232-236.	2.3	17
52	A Nuclear Factor in B Cells and Beyond. Journal of Immunology, 2006, 177, 7483-7484.	0.8	7
53	Intestinal epithelial cells release CXCLâ€2 in response to lipopolysaccharide via NFâ€ŁB and IKK activation. FASEB Journal, 2006, 20, A1094.	0.5	0
54	NFÎ¹B Activatesin vivothe Synthesis of Inducible Cox-2 in the Brain. Journal of Cerebral Blood Flow and Metabolism, 2005, 25, 1047-1059.	4.3	73

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55	Selective inhibition of NF-?B in dendritic cells by the NEMO-binding domain peptide blocks maturation and prevents T cell proliferation and polarization. European Journal of Immunology, 2005, 35, 1164-1174.	2.9	63
56	Initiation and termination of NF-ÎºB signaling by the intracellular protozoan parasite Toxoplasma gondii. Journal of Cell Science, 2005, 118, 3501-3508.	2.0	61
57	Inactivation of the Cerebral NFÎºB Pathway Inhibits Interleukin-1Î²-Induced Sickness Behavior and c-Fos Expression in Various Brain Nuclei. Neuropsychopharmacology, 2005, 30, 1492-1499.	5.4	118
58	Inhibition of inhibitor of ÎºB kinases stimulates hepatic stellate cell apoptosis and accelerated recovery from rat liver fibrosis. Gastroenterology, 2005, 128, 108-120.	1.3	256
59	A Novel Ubiquitin-like Domain in IÎºB Kinase Î² Is Required for Functional Activity of the Kinase. Journal of Biological Chemistry, 2004, 279, 45528-45539.	3.4	52
60	Selective inhibition of NF-ÎºB blocks osteoclastogenesis and prevents inflammatory bone destruction in vivo. Nature Medicine, 2004, 10, 617-624.	30.7	465
61	RelB Forms Transcriptionally Inactive Complexes with RelA/p65. Journal of Biological Chemistry, 2003, 278, 19852-19860.	3.4	130
62	Tumor Necrosis Factor-Î± Induces Nuclear Factor-ÎºB-dependent TRPC1 Expression in Endothelial Cells. Journal of Biological Chemistry, 2003, 278, 37195-37203.	3.4	87
63	Characterization of the IÎºB-kinase NEMO Binding Domain. Journal of Biological Chemistry, 2002, 277, 45992-46000.	3.4	137
64	Inhibition of Nuclear Factor Kappa B (NF-B):: An Emerging Theme in Anti-Inflammatory Therapies. Molecular Interventions: Pharmacological Perspectives From Biology, Chemistry and Genomics, 2002, 2, 22-35.	3.4	218
65	The Phosphorylation Status of Nuclear NF-ÎšB Determines Its Association with CBP/p300 or HDAC-1. Molecular Cell, 2002, 9, 625-636.	9.7	896
66	Selective targeting of the nuclear factor-ÎºB pathway enhances tumor necrosis factorâ€“related apoptosis-inducing ligand-mediated pancreatic cancer cell death. Surgery, 2002, 132, 127-134.	1.9	67
67	SIGNAL TRANSDUCTION:IkB Kinases: Kinsmen with Different Crafts. Science, 1999, 284, 271-273.	12.6	127
68	Signal transduction through NF-ÎºB. Trends in Immunology, 1998, 19, 80-88.	7.5	1,045
69	NF-ÎºB AND REL PROTEINS: Evolutionarily Conserved Mediators of Immune Responses. Annual Review of Immunology, 1998, 16, 225-260.	21.8	4,878
70	Activation of p42mapkin human umbilical vein endothelial cells by interleukin-1Î± and tumor necrosis factor-Î±. American Journal of Physiology - Cell Physiology, 1998, 274, C789-C798.	4.6	20
71	Rel/NF-ÎºB and IÎºB proteins: an overview. Seminars in Cancer Biology, 1997, 8, 63-73.	9.6	335
72	Effects of protein tyrosine kinase inhibitors on cytokineâ€induced adhesion molecule expression by human umbilical vein endothelial cells. British Journal of Pharmacology, 1996, 118, 1761-1771.	5.4	60

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73	Inhibition of MAP kinase kinase (MEK) blocks endothelial PGI2release but has no effect on von Willebrand factor secretion or E-selectin expression. FEBS Letters, 1996, 388, 180-184.	2.8	40
74	Protein tyrosine kinases regulate agonist-stimulated prostacyclin release but not von Willebrand factor secretion from human umbilical vein endothelial cells. Biochemical Journal, 1996, 315, 407-416.	3.7	25
75	ICAM-1-independent lymphocyte transmigration across high endothelium : Differential up-regulation by interferon Î³, tumor necrosis factor-Î± and interleukin 1Î². European Journal of Immunology, 1992, 22, 219-226.	2.9	66
76	BCR-Induced Ca ²⁺ Signals Dynamically Tune Key Checkpoints that Control the Survival, Metabolic Reprogramming, and Proliferation of NaÃ¯ve B Cells. SSRN Electronic Journal, 0, , .	0.4	0