

# Mark D Mannie

## List of Publications by Year in descending order

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59  
papers

1,543  
citations

393982

19  
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360668

35  
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all docs

59  
docs citations

59  
times ranked

1258  
citing authors

#	ARTICLE	IF	CITATIONS
1	Hypoxia-inducible factor-1 drives divergent immunomodulatory functions in the pathogenesis of autoimmune diseases. <i>Immunology</i> , 2021, 164, 31-42.	2.0	20
2	Low-Zone IL-2 Signaling: Fusion Proteins Containing Linked CD25 and IL-2 Domains Sustain Tolerogenic Vaccination in vivo and Promote Dominance of FOXP3+ Tregs in vitro. <i>Frontiers in Immunology</i> , 2020, 11, 541619.	2.2	8
3	Tolerogenic vaccines: Targeting the antigenic and cytokine niches of FOXP3+ regulatory T cells. <i>Cellular Immunology</i> , 2020, 355, 104173.	1.4	8
4	A GM-CSF-neuroantigen tolerogenic vaccine elicits inefficient antigen recognition events below the CD40L triggering threshold to expand CD4+ CD25+ FOXP3+ Tregs that inhibit experimental autoimmune encephalomyelitis (EAE). <i>Journal of Neuroinflammation</i> , 2020, 17, 180.	3.1	6
5	A GMCSF-Neuroantigen Tolerogenic Vaccine Elicits Systemic Lymphocytosis of CD4+ CD25high FOXP3+ Regulatory T Cells in Myelin-Specific TCR Transgenic Mice Contingent Upon Low-Efficiency T Cell Antigen Receptor Recognition. <i>Frontiers in Immunology</i> , 2019, 9, 3119.	2.2	7
6	Partial CD25 Antagonism Enables Dominance of Antigen-Inducible CD25high FOXP3+ Regulatory T Cells As a Basis for a Regulatory T Cell-Based Adoptive Immunotherapy. <i>Frontiers in Immunology</i> , 2017, 8, 1782.	2.2	12
7	IFN- $\gamma$ Facilitates Neuroantigen-Dependent Induction of CD25+ FOXP3+ Regulatory T Cells That Suppress Experimental Autoimmune Encephalomyelitis. <i>Journal of Immunology</i> , 2016, 197, 2992-3007.	0.4	21
8	Depletion of CD4+ CD25+ regulatory T cells confers susceptibility to experimental autoimmune encephalomyelitis (EAE) in GM-CSF-deficient <i>Csf2</i> <sup>-/-</sup> mice. <i>Journal of Leukocyte Biology</i> , 2016, 100, 747-760.	1.5	18
9	Enhanced stability of tristetraprolin mRNA protects mice against immune-mediated inflammatory pathologies. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2016, 113, 1865-1870.	3.3	79
10	The Immune Basis of Allergic Lung Disease. , 2015, , 683-719.		3
11	GM-CSF-Neuroantigen Fusion Proteins Reverse Experimental Autoimmune Encephalomyelitis and Mediate Tolerogenic Activity in Adjuvant-Primed Environments: Association with Inflammation-Dependent, Inhibitory Antigen Presentation. <i>Journal of Immunology</i> , 2014, 193, 2317-2329.	0.4	13
12	The Extracellular Domain of Myelin Oligodendrocyte Glycoprotein Elicits Atypical Experimental Autoimmune Encephalomyelitis in Rat and Macaque Species. <i>PLoS ONE</i> , 2014, 9, e110048.	1.1	6
13	Tolerogenic vaccines for Multiple Sclerosis. <i>Human Vaccines and Immunotherapeutics</i> , 2013, 9, 1032-1038.	1.4	21
14	Airway Anatomy, Physiology, and Inflammation. , 2013, , 19-61.		3
15	Cytokine-Neuroantigen Fusion Proteins as a New Class of Tolerogenic, Therapeutic Vaccines for Treatment of Inflammatory Demyelinating Disease in Rodent Models of Multiple Sclerosis. <i>Frontiers in Immunology</i> , 2012, 3, 255.	2.2	24
16	Neuroantigen-specific, tolerogenic vaccines: GM-CSF is a fusion partner that facilitates tolerance rather than immunity to dominant self-epitopes of myelin in murine models of experimental autoimmune encephalomyelitis (EAE). <i>BMC Immunology</i> , 2011, 12, 72.	0.9	19
17	Autoimmunity and asthma: The dirt on the hygiene hypothesis. <i>Self/nonself</i> , 2010, 1, 123-128.	2.0	3
18	Experimental Autoimmune Encephalomyelitis in Lewis rats: IFN- $\gamma$ Acts As a Tolerogenic Adjuvant for Induction of Neuroantigen-Dependent Tolerance. <i>Journal of Immunology</i> , 2009, 182, 5331-5341.	0.4	15

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19	Vaccinia virus decreases major histocompatibility complex (MHC) class II antigen presentation, T cell priming, and peptide association with MHC class II. <i>Immunology</i> , 2009, 128, 381-392.	2.0	32
20	Experimental Autoimmune Encephalomyelitis in the Rat. <i>Current Protocols in Immunology</i> , 2009, 85, Unit 15.2.	3.6	25
21	A GMCSF-neuroantigen fusion protein is a potent tolerogen in experimental autoimmune encephalomyelitis (EAE) that is associated with efficient targeting of neuroantigen to APC. <i>Journal of Leukocyte Biology</i> , 2009, 87, 509-521.	1.5	25
22	IL-2/Neuroantigen Fusion Proteins as Antigen-Specific Tolerogens in Experimental Autoimmune Encephalomyelitis (EAE): Correlation of T Cell-Mediated Antigen Presentation and Tolerance Induction. <i>Journal of Immunology</i> , 2007, 178, 2835-2843.	0.4	14
23	A Fusion Protein Consisting of IL-16 and the Encephalitogenic Peptide of Myelin Basic Protein Constitutes an Antigen-Specific Tolerogenic Vaccine That Inhibits Experimental Autoimmune Encephalomyelitis. <i>Journal of Immunology</i> , 2007, 179, 1458-1465.	0.4	17
24	Cytokine neuroantigen fusion proteins: New tools for modulation of myelin basic protein (MBP)-specific T cell responses in experimental autoimmune encephalomyelitis. <i>Journal of Immunological Methods</i> , 2007, 319, 118-132.	0.6	7
25	Activation-dependent phases of T cells distinguished by use of optical tweezers and near infrared Raman spectroscopy. <i>Journal of Immunological Methods</i> , 2005, 297, 53-60.	0.6	60
26	MHC class II biosynthesis by activated rat CD4+ T cells: development of repression in vitro and modulation by APC-derived signals. <i>Cellular Immunology</i> , 2004, 230, 33-43.	1.4	9
27	IL-4 responsive CD4+ T cells specific for myelin basic protein: IL-2 confers a prolonged postactivation refractory phase. <i>Immunology and Cell Biology</i> , 2003, 81, 8-19.	1.0	18
28	Acquisition of functional MHC class II/peptide complexes by T cells during thymic development and CNS-directed pathogenesis. <i>Cellular Immunology</i> , 2002, 218, 13-25.	1.4	20
29	Interleukin-2 Promotes Antigenic Reactivity of Rested T Cells but Prolongs the Postactivation Refractory Phase of Activated T Cells. <i>Cellular Immunology</i> , 2001, 211, 51-60.	1.4	14
30	MHC Class-II-Restricted Antigen Presentation by Myelin Basic Protein-Specific CD4+ T Cells Causes Prolonged Desensitization and Outgrowth of CD4+ Responders. <i>Cellular Immunology</i> , 2001, 212, 51-62.	1.4	25
31	Intercellular Exchange of Class II Major Histocompatibility Complex/Peptide Complexes Is a Conserved Process That Requires Activation of T Cells But Is Constitutive in Other Types of Antigen Presenting Cell. <i>Cellular Immunology</i> , 2001, 214, 165-172.	1.4	27
32	Intercellular Exchange of Class II MHC Complexes: Ultrastructural Localization and Functional Presentation of Adsorbed I-A/Peptide Complexes. <i>Cellular Immunology</i> , 2001, 214, 21-34.	1.4	34
33	T Cell-Mediated Antigen Presentation: A Potential Mechanism of Infectious Tolerance. <i>Immunologic Research</i> , 2001, 23, 01-22.	1.3	11
34	Immunological self/nonself discrimination. <i>Immunologic Research</i> , 1999, 19, 65-87.	1.3	196
35	Vesicles bearing MHC class II molecules mediate transfer of antigen from antigen-presenting cells to CD4+ T cells. <i>European Journal of Immunology</i> , 1999, 29, 1363-1373.	1.6	91
36	An autologous self-antigen differentially regulates expression of I-A glycoproteins and B7 costimulatory molecules on CD4+ CD8+ T helper cells. <i>Journal of Leukocyte Biology</i> , 1999, 66, 120-126.	1.5	9

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37	Partial Agonism Elicits an Enduring Phase of T-Cell-Mediated Antigen Presentation. Cellular Immunology, 1998, 186, 83-93.	1.4	16
38	Class II MHC/Peptide Complexes on T Cell Antigen-Presenting Cells: Agonistic Antigen Recognition Inhibits Subsequent Antigen Presentation. Cellular Immunology, 1998, 186, 111-120.	1.4	18
39	A Novel Monoclonal Antibody Against Rat LFA-1: Blockade of LFA-1 and CD4 Augments Class II MHC Expression on T Cells. Hybridoma, 1998, 17, 331-338.	0.9	4
40	Acquired Resistance to Experimental Autoimmune Encephalomyelitis Is Independent of V $\beta$ 2 Usage. Cellular Immunology, 1997, 179, 55-65.	1.4	12
41	Modulation of outward K <sup>+</sup> conductance is a post-activational event in rat T lymphocytes responsible for the adoptive transfer of experimental allergic encephalomyelitis. Journal of Biomedical Science, 1997, 4, 98-110.	2.6	11
42	Potassium channel blockers inhibit adoptive transfer of experimental allergic encephalomyelitis by myelin-basic-protein-stimulated rat T lymphocytes. Journal of Biomedical Science, 1997, 4, 169-178.	2.6	10
43	Antigen presentation by T cells: T cell receptor ligation promotes antigen acquisition from professional antigen-presenting cells. European Journal of Immunology, 1997, 27, 3198-3205.	1.6	64
44	The Post-activation refractory phase: A mechanism to measure antigenic complexity and ensure self-tolerance among mature peripheral T lymphocytes. Medical Hypotheses, 1996, 47, 467-470.	0.8	4
45	T-Helper Lymphocytes Specific for Myelin Basic Protein: Low-Density Activation Prolongs a Postactivation Refractory Phase Marked by Decreased Pathogenicity and Enhanced Sensitivity to Anergy. Cellular Immunology, 1996, 172, 108-117.	1.4	9
46	Prostaglandin E2 promotes the induction of anergy during T helper cell recognition of myelin basic protein. Cellular Immunology, 1995, 160, 132-138.	1.4	35
47	Parallel Costimulatory Pathways Promote Myelin Basic Protein-Stimulated Proliferation of Encephalitogenic Rat T Cells. Cellular Immunology, 1994, 153, 312-328.	1.4	7
48	T-Helper Lymphocytes Specific for Myelin Basic Protein: Activation-Induced Refractoriness of IL-2 Production Pathways Augments an Anti-CD4-Mediated Proliferative Deficit. Cellular Immunology, 1994, 154, 484-497.	1.4	6
49	Inhibition of activation-induced death in T cell hybridomas by thiol antioxidants: oxidative stress as a mediator of apoptosis. Journal of Leukocyte Biology, 1994, 55, 221-226.	1.5	227
50	Emergence of a radioresistant population of co-stimulatory splenocytes during remission of experimental autoimmune encephalomyelitis in Lewis rats. Immunology Letters, 1993, 38, 237-242.	1.1	0
51	A Unique Costimulatory Pathway Defined with T Cell Hybridomas Specific for Myelin Basic Protein: Third Party Costimulators Restrict Antigenic Responses in Time and Space. Cellular Immunology, 1993, 147, 25-40.	1.4	3
52	Immune discrimination of self and nonself: A unified theory for the induction of self tolerance among thymocytes and mature peripheral T cells. Medical Hypotheses, 1993, 40, 105-112.	0.8	8
53	Subset-specific co-stimulatory signals are required for IL-2 production but not growth inhibition responses by T cell hybrids specific for myelin basic protein. Cellular Immunology, 1992, 140, 219-236.	1.4	6
54	An alternative pathway of B cell activation: stilbene disulfonates interact with a Cl $\alpha$ binding motif on AEn-related proteins to stimulate mitogenesis. European Journal of Immunology, 1992, 22, 1165-1171.	1.6	16

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55	A unified model for T cell antigen recognition and thymic selection of the T cell repertoire. <i>Journal of Theoretical Biology</i> , 1991, 151, 169-192.	0.8	36
56	The N- and C-terminal boundaries of myelin basic protein determinants required for encephalitogenic and proliferative responses of Lewis rat T cells. <i>Journal of Neuroimmunology</i> , 1990, 26, 201-211.	1.1	20
57	Indomethacin augments in vitro proliferative responses of Lewis rat lymphocytes to myelin basic protein. <i>Cellular Immunology</i> , 1989, 121, 196-212.	1.4	12
58	Clonotypic heterogeneity of lewis rat T cells specific for the encephalitogenic 68-86 region of myelin basic protein. <i>Cellular Immunology</i> , 1989, 122, 534-547.	1.4	15
59	Induction of experimental allergic encephalomyelitis in Lewis rats with purified synthetic peptides: delineation of antigenic determinants for encephalitogenicity, in vitro activation of cellular transfer, and proliferation of lymphocytes. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 1985, 82, 5515-5519.	3.3	84