Robert Maile

List of Publications by Year in descending order

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POREDT MAILE

#	Article	IF	CITATIONS
1	A prospective study of asymptomatic SARS-CoV-2 infection among individuals involved in academic research under limited operations during the COVID-19 pandemic. PLoS ONE, 2022, 17, e0267353.	2.5	5
2	Role of Nitric Oxide-Releasing Glycosaminoglycans in Wound Healing. ACS Biomaterials Science and Engineering, 2022, 8, 2537-2552.	5.2	9
3	Characterization of extracellular vesicle miRNA identified in peripheral blood of chronic pancreatitis patients. Molecular and Cellular Biochemistry, 2021, 476, 4331-4341.	3.1	7
4	Plasma extracellular vesicles released after severe burn injury modulate macrophage phenotype and function. Journal of Leukocyte Biology, 2021, 111, 33-49.	3.3	19
5	Burn Injury Induces Proinflammatory Plasma Extracellular Vesicles That Associate with Length of Hospital Stay in Women: CRP and SAA1 as Potential Prognostic Indicators. International Journal of Molecular Sciences, 2021, 22, 10083.	4.1	9
6	Nitric Oxide-Releasing Hyaluronic Acid as an Antibacterial Agent for Wound Therapy. Biomacromolecules, 2021, 22, 867-879.	5.4	19
7	One-hit wonder: Late after burn injury, granulocytes can clear one bacterial infection but cannot control a subsequent infection. Burns, 2019, 45, 627-640.	1.9	10
8	Mammalian target of rapamycin regulates a hyperresponsive state in pulmonary neutrophils late after burn injury. Journal of Leukocyte Biology, 2018, 103, 909-918.	3.3	17
9	Blocking CXCL1-dependent neutrophil recruitment prevents immune damage and reduces pulmonary bacterial infection after inhalation injury. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2018, 314, L822-L834.	2.9	22
10	HMGB1/IL-1β complexes in plasma microvesicles modulate immune responses to burn injury. PLoS ONE, 2018, 13, e0195335.	2.5	33
11	Innate Immune Cell Recovery Is Positively Regulated by NLRP12 during Emergency Hematopoiesis. Journal of Immunology, 2017, 198, 2426-2433.	0.8	18
12	Pseudomonas aeruginosa exoproducts determine antibiotic efficacy against Staphylococcus aureus. PLoS Biology, 2017, 15, e2003981.	5.6	141
13	Differential regulation of innate immune cytokine production through pharmacological activation of Nuclear Factor-Erythroid-2-Related Factor 2 (NRF2) in burn patient immune cells and monocytes. PLoS ONE, 2017, 12, e0184164.	2.5	18
14	Direct detection of blood nitric oxide reveals a burn-dependent decrease of nitric oxide in response to Pseudomonas aeruginosa infection. Burns, 2016, 42, 1522-1527.	1.9	13
15	Timeline of health care–associated infections and pathogens after burn injuries. American Journal of Infection Control, 2016, 44, 1511-1516.	2.3	59
16	Association between early airway damage-associated molecular patterns and subsequent bacterial infection in patients with inhalational and burn injury. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2015, 308, L855-L860.	2.9	31
17	Flagellin Treatment Prevents Increased Susceptibility to Systemic Bacterial Infection after Injury by Inhibiting Anti-Inflammatory IL-10+ IL-12- Neutrophil Polarization. PLoS ONE, 2014, 9, e85623.	2.5	52
18	Bronchoscopy-Derived Correlates of Lung Injury following Inhalational Injuries: A Prospective Observational Study. PLoS ONE, 2013, 8, e64250.	2.5	30

ROBERT MAILE

#	Article	IF	CITATIONS
19	Burn injury induces high levels of phosphorylated insulin-like growth factor binding protein-1. International Journal of Burns and Trauma, 2013, 3, 180-9.	0.2	4
20	Radiation Combined With Thermal Injury Induces Immature Myeloid Cells. Shock, 2012, 38, 532-542.	2.1	18
21	Th17 (IFNÎ ³ - IL17+) CD4+ T Cells Generated After Burn Injury May Be a Novel Cellular Mechanism for Postburn Immunosuppression. Journal of Trauma, 2011, 70, 681-690.	2.3	22
22	Toll-like Receptor 2 and 4 Ligation Results in Complex Altered Cytokine Profiles Early and Late After Burn Injury. Journal of Trauma, 2008, 64, 1069-1078.	2.3	34
23	CD8+ T Cell Activation Is Governed by TCR-Peptide/MHC Affinity, Not Dissociation Rate. Journal of Immunology, 2007, 179, 2952-2960.	0.8	111
24	Downregulation of Immune Signaling Genes in Patients With Large Surface Burn Injury. Journal of Burn Care and Research, 2007, 28, 879-887.	0.4	14
25	Increased Toll-Like Receptor 4 Expression on T Cells May Be a Mechanism for Enhanced T cell Response Late After Burn Injury. Journal of Trauma, 2006, 61, 293-299.	2.3	45
26	The Effect of Burn Injury on CD8+ and CD4+ T Cells in an Irradiation Model of Homeostatic Proliferation. Journal of Trauma, 2006, 61, 1062-1068.	2.3	10
27	Lowâ€avidity CD8 ^{lo} T cells induced by incomplete antigen stimulation <i>in vivo</i> regulate naive higher avidity CD8 ^{hi} T cell responses to the same antigen. European Journal of Immunology, 2006, 36, 397-410.	2.9	32
28	Lymphopenia-Induced Homeostatic Proliferation of CD8+T Cells Is a Mechanism for Effective Allogeneic Skin Graft Rejection following Burn Injury. Journal of Immunology, 2006, 176, 6717-6726.	0.8	22
29	Memory CD8+ T cells require CD8 coreceptor engagement for calcium mobilization and proliferation, but not cytokine production. Immunology, 2005, 114, 44-52.	4.4	5
30	Peripheral "CD8 Tuning―Dynamically Modulates the Size and Responsiveness of an Antigen-Specific T Cell Pool In Vivo. Journal of Immunology, 2005, 174, 619-627.	0.8	73
31	Interplay between TCR Affinity and Necessity of Coreceptor Ligation: High-Affinity Peptide-MHC/TCR Interaction Overcomes Lack of CD8 Engagement. Journal of Immunology, 2003, 171, 4493-4503.	0.8	80
32	Peptidic Termini Play a Significant Role in TCR Recognition. Journal of Immunology, 2002, 169, 3137-3145.	0.8	21
33	Antigen-Specific Modulation of an Immune Response by In Vivo Administration of Soluble MHC Class I Tetramers. Journal of Immunology, 2001, 167, 3708-3714.	0.8	71
34	Modulation of immune response with MHC class i allotetramers. Journal of the American College of Surgeons, 2000, 191, S49.	0.5	0
35	Naive CD8+ T Cells Do Not Require Costimulation for Proliferation and Differentiation into Cytotoxic Effector Cells. Journal of Immunology, 2000, 164, 1216-1222.	0.8	99