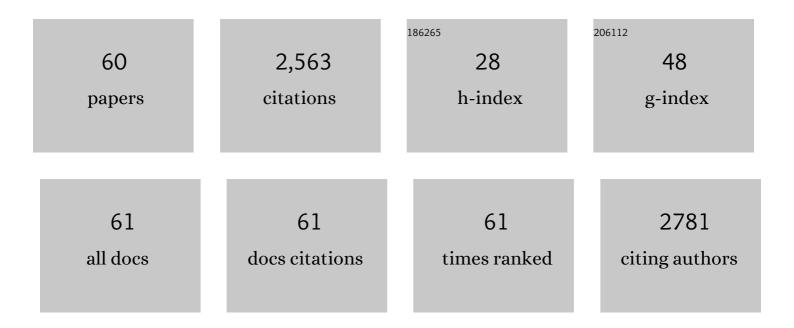
## Michael A O'reilly

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Neonatal Hyperoxia Activates ATF4 to Stimulate Folate Metabolism and AT2 Cell Proliferation. American Journal of Respiratory Cell and Molecular Biology, 2022, , .	2.9	2
2	Ataxia telangiectasia mutated is required for efficient proximal airway epithelial cell regeneration following influenza A virus infection. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2022, 322, L581-L592.	2.9	0
3	Neonatal hyperoxia inhibits proliferation and survival of atrial cardiomyocytes by suppressing fatty acid synthesis. JCI Insight, 2021, 6, .	5.0	16
4	Neonatal hyperoxia impairs adipogenesis of bone marrow-derived mesenchymal stem cells and fat accumulation in adult mice. Free Radical Biology and Medicine, 2021, 167, 287-298.	2.9	2
5	Low-dose hyperoxia primes airways for fibrosis in mice after influenza A infection. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2021, 321, L750-L763.	2.9	1
6	Early Neonatal Oxygen Exposure Predicts Pulmonary Morbidity and Functional Deficits at 1ÂYear. Journal of Pediatrics, 2020, 223, 20-28.e2.	1.8	14
7	Alveolar regeneration through a Krt8+ transitional stem cell state that persists in human lung fibrosis. Nature Communications, 2020, 11, 3559.	12.8	378
8	Tumor Necrosis Factor Induces Obliterative Pulmonary Vascular Disease in a Novel Model of Connective Tissue Disease–Associated Pulmonary Arterial Hypertension. Arthritis and Rheumatology, 2020, 72, 1759-1770.	5.6	14
9	Lung SOD3 limits neurovascular reperfusion injury and systemic immune activation following transient global cerebral ischemia. Journal of Stroke and Cerebrovascular Diseases, 2020, 29, 104942.	1.6	1
10	Pulmonary mechanics and structural lung development after neonatal hyperoxia in mice. Pediatric Research, 2020, 87, 1201-1210.	2.3	24
11	Neonatal hyperoxia enhances age-dependent expression of SARS-CoV-2 receptors in mice. Scientific Reports, 2020, 10, 22401.	3.3	16
12	Ataxia-telangiectasia mutated is required for the development of protective immune memory after influenza A virus infection. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2019, 317, L591-L601.	2.9	6
13	Lung-Specific Extracellular Superoxide Dismutase Improves Cognition of Adult Mice Exposed to Neonatal Hyperoxia. Frontiers in Medicine, 2018, 5, 334.	2.6	8
14	Inflammation and transcriptional responses of peripheral blood mononuclear cells in classic ataxia telangiectasia. PLoS ONE, 2018, 13, e0209496.	2.5	20
15	Cognitive flexibility deficits in male mice exposed to neonatal hyperoxia followed by concentrated ambient ultrafine particles. Neurotoxicology and Teratology, 2018, 70, 51-59.	2.4	9
16	Neonatal hyperoxia depletes pulmonary vein cardiomyocytes in adult mice via mitochondrial oxidation. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2018, 314, L846-L859.	2.9	25
17	Alternative Progenitor Lineages Regenerate the Adult Lung Depleted of Alveolar Epithelial Type 2 Cells. American Journal of Respiratory Cell and Molecular Biology, 2017, 56, 453-464.	2.9	44
18	SMG-1 kinase attenuates mitochondrial ROS production but not cell respiration deficits during hyperoxia. Experimental Lung Research, 2017, 43, 229-239.	1.2	2

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19	Oxygen-dependent changes in lung development do not affect epithelial infection with influenza A virus. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2017, 313, L940-L949.	2.9	4
20	The Oxygen Environment at Birth Specifies the Population of Alveolar Epithelial Stem Cells in the Adult Lung. Stem Cells, 2016, 34, 1396-1406.	3.2	28
21	Cumulative neonatal oxygen exposure predicts response of adult mice infected with influenza A virus. Pediatric Pulmonology, 2015, 50, 222-230.	2.0	17
22	Bronchopulmonary Dysplasia Early Changes Leading to Long-Term Consequences. Frontiers in Medicine, 2015, 2, 2.	2.6	51
23	Affect of Early Life Oxygen Exposure on Proper Lung Development and Response to Respiratory Viral Infections. Frontiers in Medicine, 2015, 2, 55.	2.6	18
24	Hyperoxia activates ATM independent from mitochondrial ROS and dysfunction. Redox Biology, 2015, 5, 176-185.	9.0	44
25	Neonatal hyperoxia leads to persistent alterations in NK responses to influenza A virus infection. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2015, 308, L76-L85.	2.9	13
26	Neonatal Hyperoxia Stimulates the Expansion of Alveolar Epithelial Type II Cells. American Journal of Respiratory Cell and Molecular Biology, 2014, 50, 757-766.	2.9	52
27	The genome-wide transcriptional response to neonatal hyperoxia identifies Ahr as a key regulator. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2014, 307, L516-L523.	2.9	36
28	DNA double-strand breaks activate ATM independent of mitochondrial dysfunction in A549 cells. Free Radical Biology and Medicine, 2014, 75, 30-39.	2.9	7
29	Neonatal Hyperoxia Increases Sensitivity of Adult Mice to Bleomycin-Induced Lung Fibrosis. American Journal of Respiratory Cell and Molecular Biology, 2013, 48, 258-266.	2.9	22
30	The role of hyperoxia in the pathogenesis of experimental BPD. Seminars in Perinatology, 2013, 37, 69-78.	2.5	120
31	Transdifferentiation of alveolar epithelial type II to type I cells is controlled by opposing TGF-β and BMP signaling. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2013, 305, L409-L418.	2.9	83
32	Neonatal hyperoxia alters the host response to influenza A virus infection in adult mice through multiple pathways. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2013, 305, L282-L290.	2.9	44
33	Angiotensin II: tapping the cell cycle machinery to kill endothelial cells. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2012, 303, L575-L576.	2.9	2
34	Memory CD8 <sup>+</sup> T Cells Are Sufficient To Alleviate Impaired Host Resistance to Influenza A Virus Infection Caused by Neonatal Oxygen Supplementation. Vaccine Journal, 2012, 19, 1432-1441.	3.1	18
35	Neonatal Oxygen Increases Sensitivity to Influenza A Virus Infection in Adult Mice by Suppressing Epithelial Expression of Ear1. American Journal of Pathology, 2012, 181, 441-451.	3.8	37
36	Neonatal Hyperoxia Causes Pulmonary Vascular Disease and Shortens Life Span in Aging Mice. American Journal of Pathology, 2011, 178, 2601-2610.	3.8	106

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37	The RNA surveillance protein SMG1 activates p53 in response to DNA double-strand breaks but not exogenously oxidized mRNA. Cell Cycle, 2011, 10, 2561-2567.	2.6	41
38	Bcl-X <sub>L</sub> Is the primary mediator of p21 protection against hyperoxia-induced cell death. Experimental Lung Research, 2011, 37, 82-91.	1.2	13
39	Neonatal oxygen adversely affects lung function in adult mice without altering surfactant composition or activity. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2009, 297, L641-L649.	2.9	113
40	p21Cip1 protects against oxidative stress by suppressing ER-dependent activation of mitochondrial death pathways. Free Radical Biology and Medicine, 2009, 46, 33-41.	2.9	36
41	Hyperoxia augments ER-stress-induced cell death independent of BiP loss. Free Radical Biology and Medicine, 2009, 47, 1742-1752.	2.9	26
42	PUMA inactivation protects against oxidative stress through p21/Bcl-XL inhibition of bax death. Free Radical Biology and Medicine, 2008, 44, 367-374.	2.9	11
43	Neonatal Hyperoxia Enhances the Inflammatory Response in Adult Mice Infected with Influenza A Virus. American Journal of Respiratory and Critical Care Medicine, 2008, 177, 1103-1110.	5.6	110
44	p21Cip1/Waf1/Sdi1 protects against hyperoxia by maintaining expression of Bcl-XL. Free Radical Biology and Medicine, 2006, 41, 601-609.	2.9	15
45	Type II epithelial cells are critical target for hyperoxia-mediated impairment of postnatal lung development. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2006, 291, L1101-L1111.	2.9	133
46	p21Cip1/WAF1/Sdi1Does Not Affect Expression of Base Excision DNA Repair Enzymes During Chronic Oxidative Stress. Antioxidants and Redox Signaling, 2005, 7, 719-725.	5.4	4
47	Redox Activation of p21Cip1/WAF1/Sdi1: A Multifunctional Regulator of Cell Survival and Death. Antioxidants and Redox Signaling, 2005, 7, 108-118.	5.4	99
48	The Cdk and PCNA domains on p21Cip1 both function to inhibit G1/S progression during hyperoxia. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2004, 286, L506-L513.	2.9	31
49	In vivo exposure to hyperoxia induces DNA damage in a population of alveolar type II epithelial cells. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2004, 286, L1045-L1054.	2.9	65
50	Activation of the G2 cell cycle checkpoint enhances survival of epithelial cells exposed to hyperoxia. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2003, 284, L368-L375.	2.9	28
51	Induced p21Cip1 in premature baboons with CLD: implications for alveolar hypoplasia. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2003, 285, L964-L971.	2.9	7
52	Normal Remodeling of the Oxygen-Injured Lung Requires the Cyclin-Dependent Kinase Inhibitor p21Cip1/WAF1/Sdi1. American Journal of Pathology, 2002, 161, 1383-1393.	3.8	28
53	Increased epithelial cell proliferation in very premature baboons with chronic lung disease. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2002, 283, L991-L1001.	2.9	41
54	Growth arrest in G1 protects against oxygen-induced DNA damage and cell death. Journal of Cellular Physiology, 2002, 193, 26-36.	4.1	60

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55	DNA damage and cell cycle checkpoints in hyperoxic lung injury: braking to facilitate repair. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2001, 281, L291-L305.	2.9	110
56	The Cyclin-Dependent Kinase Inhibitor p21 Protects the Lung from Oxidative Stress. American Journal of Respiratory Cell and Molecular Biology, 2001, 24, 703-710.	2.9	102
57	Bcl-2 Family Gene Expression during Severe Hyperoxia Induced Lung Injury. Laboratory Investigation, 2000, 80, 1845-1854.	3.7	59
58	p53-independent induction of <i>GADD45</i> and <i>GADD153</i> in mouse lungs exposed to hyperoxia. American Journal of Physiology - Lung Cellular and Molecular Physiology, 2000, 278, L552-L559.	2.9	60
59	Accumulation of p21 <sup>Cip1/WAF1</sup> during Hyperoxic Lung Injury in Mice. American Journal of Respiratory Cell and Molecular Biology, 1998, 19, 777-785.	2.9	62
60	Epithelial-mesenchymal interactions in the alteration of gene expression and morphology following lung injury. Microscopy Research and Technique, 1997, 38, 473-479.	2.2	20