

# Michael V Autieri

## List of Publications by Year in descending order

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48  
papers

1,406  
citations

430874

18  
h-index

345221

36  
g-index

49  
all docs

49  
docs citations

49  
times ranked

1361  
citing authors

#	ARTICLE	IF	CITATIONS
1	Challenging the Paradigm: Anti-Inflammatory Interleukins and Angiogenesis. <i>Cells</i> , 2022, 11, 587.	4.1	24
2	Adipocyte Phenotype Flexibility and Lipid Dysregulation. <i>Cells</i> , 2022, 11, 882.	4.1	7
3	The Role of FXR1 and Senescence in Vascular Biology and Intimal Hyperplasia. <i>FASEB Journal</i> , 2022, 36, .	0.5	0
4	IL-19 Regulates Atherosclerotic Plaque Progression via Lymphangiogenesis. <i>FASEB Journal</i> , 2022, 36, .	0.5	0
5	FXR1 Decreases Blood Pressure by Regulating Vascular Contractility. <i>FASEB Journal</i> , 2022, 36, .	0.5	0
6	Deletion of LDLRAP1 Induces Atherosclerotic Plaque Formation, Insulin Resistance, and Dysregulated Insulin Response in Adipose Tissue. <i>FASEB Journal</i> , 2022, 36, .	0.5	0
7	Adipose tissue inflammation and metabolic dysfunction in obesity. <i>American Journal of Physiology - Cell Physiology</i> , 2021, 320, C375-C391.	4.6	510
8	Resolution of inflammation in immune and nonimmune cells by interleukin-19. <i>American Journal of Physiology - Cell Physiology</i> , 2020, 319, C457-C464.	4.6	21
9	Angiotensin II, Hypercholesterolemia, and Vascular Smooth Muscle Cells: A Perfect Trio for Vascular Pathology. <i>International Journal of Molecular Sciences</i> , 2020, 21, 4525.	4.1	23
10	Postprandial activation of leukocyte-endothelium interaction by fatty acids in the visceral adipose tissue microcirculation. <i>FASEB Journal</i> , 2019, 33, 11993-12007.	0.5	9
11	RNA stability protein ILF3 mediates cytokine-induced angiogenesis. <i>FASEB Journal</i> , 2019, 33, 3304-3316.	0.5	19
12	Global Knockout of LDLRAP1 Regulates Atherosclerosis, Insulin Resistance, and VSMC Foam Cell Formation. <i>FASEB Journal</i> , 2019, 33, 496.51.	0.5	0
13	FXR1 Is an IL-19-Responsive RNA-Binding Protein that Destabilizes Pro-inflammatory Transcripts in Vascular Smooth Muscle Cells. <i>Cell Reports</i> , 2018, 24, 1176-1189.	6.4	29
14	IL-19 and Other IL-20 Family Member Cytokines in Vascular Inflammatory Diseases. <i>Frontiers in Immunology</i> , 2018, 9, 700.	4.8	39
15	Cardiovascular disease, inflammation, and mRNA stability. <i>Aging</i> , 2018, 10, 3046-3047.	3.1	5
16	Investigation of inhomogeneous and anisotropic material behavior of porcine thoracic aorta using nano-indentation tests. <i>Journal of the Mechanical Behavior of Biomedical Materials</i> , 2017, 69, 50-56.	3.1	14
17	Inflammation-regulated mRNA stability and the progression of vascular inflammatory diseases. <i>Clinical Science</i> , 2017, 131, 2687-2699.	4.3	14
18	Interleukin-19 is cardioprotective in dominant negative cyclic adenosine monophosphate response-element binding protein-mediated heart failure in a sex-specific manner. <i>World Journal of Cardiology</i> , 2017, 9, 673.	1.5	5

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19	Interleukin-19 induces angiogenesis in the absence of hypoxia by direct and indirect immune mechanisms. American Journal of Physiology - Cell Physiology, 2016, 310, C931-C941.	4.6	23
20	Regulation of mitogen-activated protein kinase by protein kinase C and mitogen-activated protein kinase phosphatase-1 in vascular smooth muscle. American Journal of Physiology - Cell Physiology, 2016, 310, C921-C930.	4.6	10
21	IL-19 Halts Progression of Atherosclerotic Plaque, Polarizes, and Increases Cholesterol Uptake and Efflux in Macrophages. American Journal of Pathology, 2016, 186, 1361-1374.	3.8	39
22	Interleukin-19 can enhance angiogenesis by Macrophage Polarization. Macrophage, 2015, 2, e562.	1.0	9
23	Correlations between transmural mechanical and morphological properties in porcine thoracic descending aorta. Journal of the Mechanical Behavior of Biomedical Materials, 2015, 47, 12-20.	3.1	12
24	Interleukin-19 increases angiogenesis in ischemic hind limbs by direct effects on both endothelial cells and macrophage polarization. Journal of Molecular and Cellular Cardiology, 2015, 79, 21-31.	1.9	43
25	IL-19 Reduces Ligation-Mediated Neointimal Hyperplasia by Reducing Vascular Smooth Muscle Cell Activation. American Journal of Pathology, 2014, 184, 2134-2143.	3.8	29
26	Abstract 45: Interleukin-19 Increases Angiogenic Gene Expression and Perfusion in Ischemic Hind-Limbs. Arteriosclerosis, Thrombosis, and Vascular Biology, 2014, 34, .	2.4	0
27	Increasing our IQ of vascular smooth muscle cell migration with IQGAP1 links PDGF receptor- $\beta$ signal to focal adhesions involved in vascular smooth muscle cell migration: role in neointimal formation after vascular injury. American Journal of Physiology - Cell Physiology, 2013, 305, C579-C580.	4.6	2
28	Attenuation of Experimental Atherosclerosis by Interleukin-19. Arteriosclerosis, Thrombosis, and Vascular Biology, 2013, 33, 2316-2324.	2.4	52
29	Identifying Mechanisms of Interleukin-19-Mediated HuR-Dependent Reduction in Vascular Cell mRNA Stability. FASEB Journal, 2013, 27, 648.5.	0.5	0
30	Attenuation of Experimental Atherosclerosis by Interleukin-19. FASEB Journal, 2013, 27, 869.7.	0.5	0
31	Anti-Inflammatory Effects of Interleukin-19 in Vascular Disease. International Journal of Inflammation, 2012, 2012, 1-10.	1.5	25
32	Pro- and Anti-Inflammatory Cytokine Networks in Atherosclerosis. ISRN Vascular Medicine, 2012, 2012, 1-17.	0.7	15
33	Interleukin-19 (IL-19) Induces Heme Oxygenase-1 (HO-1) Expression and Decreases Reactive Oxygen Species in Human Vascular Smooth Muscle Cells. Journal of Biological Chemistry, 2012, 287, 2477-2484.	3.4	40
34	The Anti-Inflammatory Cytokine Interleukin 19 Is Expressed By and Angiogenic for Human Endothelial Cells. Arteriosclerosis, Thrombosis, and Vascular Biology, 2011, 31, 167-175.	2.4	47
35	IL-19 reduces VSMC activation by regulation of mRNA regulatory factor HuR and reduction of mRNA stability. Journal of Molecular and Cellular Cardiology, 2010, 49, 647-654.	1.9	59
36	IL-19 decreases oxLDL induced VSMC activation by suppression of scavenger receptor expression and reduction of oxLDL uptake. FASEB Journal, 2010, 24, .	0.5	0

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37	IL-19 reduces VSMC activation by regulation of mRNA binding proteins and reduction of inflammatory gene expression. <i>FASEB Journal</i> , 2009, 23, 357.6.	0.5	0
38	Expression and Suppressive Effects of Interleukin-19 on Vascular Smooth Muscle Cell Pathophysiology and Development of Intimal Hyperplasia. <i>American Journal of Pathology</i> , 2008, 173, 901-909.	3.8	71
39	NFAT regulates the alternative splicing of Allograft Inflammatory Factor-1 gene: Role in neointima formation. <i>FASEB Journal</i> , 2008, 22, 49-49.	0.5	1
40	The anti-inflammatory cytokine IL-19 suppresses VSMC activation by attenuation of proliferative and inflammatory gene expression. <i>FASEB Journal</i> , 2008, 22, 900.6.	0.5	0
41	The ability of AIF-1 to activate human vascular smooth muscle cells is lost by mutations in the EF-hand calcium-binding region. <i>Experimental Cell Research</i> , 2005, 307, 204-211.	2.6	18
42	Inducible expression of the signal transduction protein 14-3-3 in injured arteries and stimulated human vascular smooth muscle cells. <i>Experimental and Molecular Pathology</i> , 2004, 76, 99-107.	2.1	15
43	Early Growth Responsive Gene (Egr)-1 Expression Correlates with Cardiac Allograft Rejection. <i>Transplantation</i> , 2004, 78, 107-111.	1.0	7
44	Antiproliferative effects of immunosuppressant drugs on vascular smooth muscle cells: An additional advantage for attenuating transplant vasculopathy. <i>Drug News and Perspectives</i> , 2004, 17, 110.	1.5	3
45	AIF-1 Is an Actin-Polymerizing and Rac1-Activating Protein That Promotes Vascular Smooth Muscle Cell Migration. <i>Circulation Research</i> , 2003, 92, 1107-1114.	4.5	66
46	Allograft-Induced Proliferation of Vascular Smooth Muscle Cells: Potential Targets for Treating Transplant Vasculopathy. <i>Current Vascular Pharmacology</i> , 2003, 1, 1-9.	1.7	30
47	Regulating the regulators: Transcription factors as targets for attenuating proliferative arteriopathies. <i>Drug News and Perspectives</i> , 2003, 16, 149.	1.5	4
48	Allograft Inflammatory Factor-1 Expression Correlates With Cardiac Rejection and Development of Cardiac Allograft Vasculopathy. <i>Circulation</i> , 2002, 106, 2218-2223.	1.6	65