

Steven P Jones

List of Publications by Year in descending order

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108
papers

8,330
citations

41323

49
h-index

46771

89
g-index

109
all docs

109
docs citations

109
times ranked

9911
citing authors

#	ARTICLE	IF	CITATIONS
1	The MEK1-ERK1/2 signaling pathway promotes compensated cardiac hypertrophy in transgenic mice. <i>EMBO Journal</i> , 2000, 19, 6341-6350.	3.5	690
2	Functional Integration of Electrically Active Cardiac Derivatives From Genetically Engineered Human Embryonic Stem Cells With Quiescent Recipient Ventricular Cardiomyocytes. <i>Circulation</i> , 2005, 111, 11-20.	1.6	455
3	The ubiquitous role of nitric oxide in cardioprotection. <i>Journal of Molecular and Cellular Cardiology</i> , 2006, 40, 16-23.	0.9	390
4	Guidelines for experimental models of myocardial ischemia and infarction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2018, 314, H812-H838.	1.5	372
5	Uncoupling Protein-2 Overexpression Inhibits Mitochondrial Death Pathway in Cardiomyocytes. <i>Circulation Research</i> , 2003, 93, 192-200.	2.0	292
6	Cardioprotection by <i>N</i> -Acetylglucosamine Linkage to Cellular Proteins. <i>Circulation</i> , 2008, 117, 1172-1182.	1.6	215
7	Endothelial nitric oxide synthase overexpression attenuates congestive heart failure in mice. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2003, 100, 4891-4896.	3.3	211
8	Simvastatin Exerts Both Anti-inflammatory and Cardioprotective Effects in Apolipoprotein E-deficient Mice. <i>Circulation</i> , 2001, 103, 2598-2603.	1.6	189
9	Myocardial ischemia-reperfusion injury is exacerbated in absence of endothelial cell nitric oxide synthase. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 1999, 276, H1567-H1573.	1.5	183
10	Endothelial nitric oxide synthase overexpression attenuates myocardial reperfusion injury. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2004, 286, H276-H282.	1.5	183
11	Metabolomic Analysis of Pressure-Overloaded and Infarcted Mouse Hearts. <i>Circulation: Heart Failure</i> , 2014, 7, 634-642.	1.6	181
12	PDGF-mediated autophagy regulates vascular smooth muscle cell phenotype and resistance to oxidative stress. <i>Biochemical Journal</i> , 2013, 451, 375-388.	1.7	175
13	O-linked <i>N</i> -acetylglucosamine transferase is indispensable in the failing heart. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2010, 107, 17797-17802.	3.3	170
14	O-GlcNAc Signaling Entrain the Circadian Clock by Inhibiting BMAL1/CLOCK Ubiquitination. <i>Cell Metabolism</i> , 2013, 17, 303-310.	7.2	169
15	The NHLBI-Sponsored Consortium for preclinical assessment of cardioprotective Therapies (CAESAR). <i>Circulation Research</i> , 2015, 116, 572-586.	2.0	164
16	Heart-targeted overexpression of caspase3 in mice increases infarct size and depresses cardiac function. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2001, 98, 9977-9982.	3.3	146
17	Augmented O-GlcNAc signaling attenuates oxidative stress and calcium overload in cardiomyocytes. <i>Amino Acids</i> , 2011, 40, 895-911.	1.2	145
18	<i>O</i> -GlcNAc Signaling in the Cardiovascular System. <i>Circulation Research</i> , 2010, 107, 171-185.	2.0	142

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19	Leukocyte-endothelial cell interactions in nitric oxide synthase-deficient mice. American Journal of Physiology - Heart and Circulatory Physiology, 1999, 276, H1943-H1950.	1.5	136
20	Bioenergetic function in cardiovascular cells: The importance of the reserve capacity and its biological regulation. Chemico-Biological Interactions, 2011, 191, 288-295.	1.7	134
21	Unique Hexosaminidase Reduces Metabolic Survival Signal and Sensitizes Cardiac Myocytes to Hypoxia/Reoxygenation Injury. Circulation Research, 2009, 104, 41-49.	2.0	132
22	Direct vascular and cardioprotective effects of rosuvastatin, a new HMG-CoA reductase inhibitor. Journal of the American College of Cardiology, 2002, 40, 1172-1178.	1.2	128
23	O-GlcNAc and the cardiovascular system. , 2014, 142, 62-71.		119
24	Simvastatin Attenuates Oxidant-Induced Mitochondrial Dysfunction in Cardiac Myocytes. Circulation Research, 2003, 93, 697-699.	2.0	114
25	Non-canonical glycosyltransferase modulates post-hypoxic cardiac myocyte death and mitochondrial permeability transition. Journal of Molecular and Cellular Cardiology, 2008, 45, 313-325.	0.9	106
26	Cariporide (HOE642), a Selective Na ⁺ -H ⁺ Exchange Inhibitor, Inhibits the Mitochondrial Death Pathway. Circulation, 2003, 108, 2275-2281.	1.6	105
27	Exercise-Induced Changes in Glucose Metabolism Promote Physiological Cardiac Growth. Circulation, 2017, 136, 2144-2157.	1.6	103
28	TRO40303, a New Cardioprotective Compound, Inhibits Mitochondrial Permeability Transition. Journal of Pharmacology and Experimental Therapeutics, 2010, 333, 696-706.	1.3	102
29	O-GlcNAc signaling attenuates ER stress-induced cardiomyocyte death. American Journal of Physiology - Heart and Circulatory Physiology, 2009, 297, H1711-H1719.	1.5	97
30	O-GlcNAc signaling is essential for NFAT-mediated transcriptional reprogramming during cardiomyocyte hypertrophy. American Journal of Physiology - Heart and Circulatory Physiology, 2012, 302, H2122-H2130.	1.5	96
31	Pretreatment With Simvastatin Attenuates Myocardial Dysfunction After Ischemia and Chronic Reperfusion. Arteriosclerosis, Thrombosis, and Vascular Biology, 2001, 21, 2059-2064.	1.1	84
32	HMG-CoA reductase inhibition protects the diabetic myocardium from ischemia-reperfusion injury. FASEB Journal, 2001, 15, 1454-1456.	0.2	83
33	Myocardial Ischemia/Reperfusion Injury in NADPH Oxidase-Deficient Mice. Circulation Research, 2000, 87, 812-817.	2.0	82
34	Differential Actions of Cardioprotective Agents on the Mitochondrial Death Pathway. Circulation Research, 2003, 92, 195-202.	2.0	78
35	Cardiomyocyte Ogt is essential for postnatal viability. American Journal of Physiology - Heart and Circulatory Physiology, 2014, 306, H142-H153.	1.5	78
36	Leukocyte and endothelial cell adhesion molecules in a chronic murine model of myocardial reperfusion injury. American Journal of Physiology - Heart and Circulatory Physiology, 2000, 279, H2196-H2201.	1.5	77

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37	Differential response to myocardial reperfusion injury in eNOS-deficient mice. American Journal of Physiology - Heart and Circulatory Physiology, 2002, 282, H2422-H2426.	1.5	77
38	Myocardial ischemia-reperfusion injury in CD18- and ICAM-1-deficient mice. American Journal of Physiology - Heart and Circulatory Physiology, 1998, 275, H2300-H2307.	1.5	70
39	Role of intracellular antioxidant enzymes after in vivo myocardial ischemia and reperfusion. American Journal of Physiology - Heart and Circulatory Physiology, 2003, 284, H277-H282.	1.5	70
40	Post-transcriptional gene silencing of KChIP2 and Nav ^{1.2} 1 in neonatal rat cardiac myocytes reveals a functional association between Na and Ito currents. Journal of Molecular and Cellular Cardiology, 2008, 45, 336-346.	0.9	69
41	High Fat Feeding in Mice Is Insufficient to Induce Cardiac Dysfunction and Does Not Exacerbate Heart Failure. PLoS ONE, 2013, 8, e83174.	1.1	69
42	CD8 + -T-Cell Depletion Ameliorates Circulatory Shock in Plasmodium berghei -Infected Mice. Infection and Immunity, 2001, 69, 7341-7348.	1.0	68
43	PR-39, a potent neutrophil inhibitor, attenuates myocardial ischemia-reperfusion injury in mice. American Journal of Physiology - Heart and Circulatory Physiology, 2000, 279, H2824-H2828.	1.5	66
44	Standardized bioenergetic profiling of adult mouse cardiomyocytes. Physiological Genomics, 2012, 44, 1208-1213.	1.0	64
45	MicroRNA-539 Is Up-regulated in Failing Heart, and Suppresses O-GlcNAcase Expression. Journal of Biological Chemistry, 2014, 289, 29665-29676.	1.6	63
46	Recent Developments in Heart Failure. Circulation Research, 2015, 117, e58-63.	2.0	60
47	Leukocyte iNOS is required for inflammation and pathological remodeling in ischemic heart failure. Basic Research in Cardiology, 2017, 112, 19.	2.5	60
48	Physiological Biomimetic Culture System for Pig and Human Heart Slices. Circulation Research, 2019, 125, 628-642.	2.0	60
49	Effects of Hypercholesterolemia on Myocardial Ischemia-Reperfusion Injury in LDL Receptor-Deficient Mice. Arteriosclerosis, Thrombosis, and Vascular Biology, 1999, 19, 2776-2781.	1.1	57
50	Integration of flux measurements to resolve changes in anabolic and catabolic metabolism in cardiac myocytes. Biochemical Journal, 2017, 474, 2785-2801.	1.7	55
51	Reperfusion injury is not affected by blockade of P-selectin in the diabetic mouse heart. American Journal of Physiology - Heart and Circulatory Physiology, 1999, 277, H763-H769.	1.5	54
52	Protein O-GlcNAcylation Is a Novel Cytoprotective Signal in Cardiac Stem Cells. Stem Cells, 2013, 31, 765-775.	1.4	54
53	Guidelines for in vivo mouse models of myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2021, 321, H1056-H1073.	1.5	53
54	Coronary endothelial P-selectin in pathogenesis of myocardial ischemia-reperfusion injury. American Journal of Physiology - Heart and Circulatory Physiology, 1998, 275, H1865-H1872.	1.5	46

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55	Cardiac overexpression of 8-oxoguanine DNA glycosylase 1 protects mitochondrial DNA and reduces cardiac fibrosis following transaortic constriction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2011, 301, H2073-H2080.	1.5	43
56	Cardioprotective actions of endogenous IL-10 are independent of iNOS. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2001, 281, H48-H52.	1.5	41
57	Cardiac phosphatase-deficient 6-phosphofructo-2-kinase/fructose-2,6-bisphosphatase increases glycolysis, hypertrophy, and myocyte resistance to hypoxia. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2008, 294, H2889-H2897.	1.5	40
58	Cardioprotective actions of acute HMG-CoA reductase inhibition in the setting of myocardial infarction. <i>Acta Physiologica Scandinavica</i> , 2001, 173, 139-143.	2.3	39
59	High glucose induces mitochondrial dysfunction independently of protein O-GlcNAcylation. <i>Biochemical Journal</i> , 2015, 467, 115-126.	1.7	39
60	Cardiomyocyte Ogt limits ventricular dysfunction in mice following pressure overload without affecting hypertrophy. <i>Basic Research in Cardiology</i> , 2017, 112, 23.	2.5	38
61	Endothelial Cell Overexpression of Fas Ligand Attenuates Ischemia-Reperfusion Injury in the Heart. <i>Journal of Biological Chemistry</i> , 2003, 278, 15185-15191.	1.6	36
62	Deficiency of iNOS does not attenuate severe congestive heart failure in mice. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2005, 288, H365-H370.	1.5	35
63	A New Method to Stabilize C-Kit Expression in Reparative Cardiac Mesenchymal Cells. <i>Frontiers in Cell and Developmental Biology</i> , 2016, 4, 78.	1.8	33
64	<i>Airn</i> Regulates Igf2bp2 Translation in Cardiomyocytes. <i>Circulation Research</i> , 2018, 122, 1347-1353.	2.0	33
65	Ischemic preconditioning prevents postischemic P-selectin expression in the rat small intestine. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 1999, 277, H2476-H2481.	1.5	32
66	Low-dose simvastatin improves survival and ventricular function via eNOS in congestive heart failure. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2006, 291, H2743-H2751.	1.5	30
67	The COX-2/PGI2 Receptor Axis Plays an Obligatory Role in Mediating the Cardioprotection Conferred by the Late Phase of Ischemic Preconditioning. <i>PLoS ONE</i> , 2012, 7, e41178.	1.1	30
68	New Insights into Metabolic Signaling and Cell Survival: The Role of $\hat{1}^2$ -O-Linkage of N-Acetylglucosamine. <i>Journal of Pharmacology and Experimental Therapeutics</i> , 2008, 327, 602-609.	1.3	29
69	E2F1 Transcription Factor Regulates O-linked N-acetylglucosamine (O-GlcNAc) Transferase and O-GlcNAcase Expression. <i>Journal of Biological Chemistry</i> , 2015, 290, 31013-31024.	1.6	28
70	Myocardial reperfusion injury in neuronal nitric oxide synthase deficient mice. <i>Coronary Artery Disease</i> , 2000, 11, 593-597.	0.3	26
71	Responses of hypertrophied myocytes to reactive species: implications for glycolysis and electrophile metabolism. <i>Biochemical Journal</i> , 2011, 435, 519-528.	1.7	26
72	RNA Editing. <i>Circulation Research</i> , 2018, 122, 399-401.	2.0	25

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73	Reduced Cardiac Fructose 2,6 Bisphosphate Increases Hypertrophy and Decreases Glycolysis following Aortic Constriction. <i>PLoS ONE</i> , 2013, 8, e53951.	1.1	24
74	Endothelial dysfunction as a nexus for endothelial cell-cardiomyocyte miscommunication. <i>Frontiers in Physiology</i> , 2014, 5, 328.	1.3	23
75	Deficiency of aldose reductase exacerbates early pressure overload-induced cardiac dysfunction and autophagy in mice. <i>Journal of Molecular and Cellular Cardiology</i> , 2018, 118, 183-192.	0.9	23
76	E2f1 deletion attenuates infarct-induced ventricular remodeling without affecting O-GlcNAcylation. <i>Basic Research in Cardiology</i> , 2019, 114, 28.	2.5	23
77	Metabolic regulation of Kv channels and cardiac repolarization by Kv β 2 subunits. <i>Journal of Molecular and Cellular Cardiology</i> , 2019, 137, 93-106.	0.9	21
78	Induction of activating transcription factor 3 limits survival following infarct-induced heart failure in mice. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2015, 309, H1326-H1335.	1.5	20
79	TNF receptor signaling inhibits cardiomyogenic differentiation of cardiac stem cells and promotes a neuroadrenergic-like fate. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2016, 311, H1189-H1201.	1.5	18
80	A Bittersweet Modification. <i>Circulation Research</i> , 2005, 96, 925-926.	2.0	13
81	Cardiac-specific overexpression of aldehyde dehydrogenase 2 exacerbates cardiac remodeling in response to pressure overload. <i>Redox Biology</i> , 2018, 17, 440-449.	3.9	13
82	Acute exposure to a high cholesterol diet attenuates myocardial ischemia-reperfusion injury in cholesteryl ester transfer protein mice. <i>Coronary Artery Disease</i> , 2001, 12, 37-44.	0.3	12
83	Cardiomyocyte Oga haploinsufficiency increases O-GlcNAcylation but hastens ventricular dysfunction following myocardial infarction. <i>PLoS ONE</i> , 2020, 15, e0242250.	1.1	11
84	RDH10 function is necessary for spontaneous fetal mouth movement that facilitates palate shelf elevation. <i>DMM Disease Models and Mechanisms</i> , 2019, 12, .	1.2	9
85	Using gene-targeted mice to investigate the pathophysiology of myocardial reperfusion injury. <i>Basic Research in Cardiology</i> , 2000, 95, 499-502.	2.5	8
86	Metabolic signatures of pregnancy-induced cardiac growth. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2022, 323, H146-H164.	1.5	8
87	Influence of biological sex and exercise on murine cardiac metabolism. <i>Journal of Sport and Health Science</i> , 2022, 11, 479-494.	3.3	8
88	Myocardial Reperfusion Injury: Insights Gained from Gene-Targeted Mice. <i>Physiology</i> , 2000, 15, 303-308.	1.6	7
89	Have the Rigor, but Hold the Mortis. <i>Circulation Research</i> , 2017, 120, 1852-1854.	2.0	7
90	O-GlcNAcylation Negatively Regulates Cardiomyogenic Fate in Adult Mouse Cardiac Mesenchymal Stromal Cells. <i>PLoS ONE</i> , 2015, 10, e0142939.	1.1	6

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91	Cardiac PANK1 deletion exacerbates ventricular dysfunction during pressure overload. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2021, 321, H784-H797.	1.5	6
92	Collagen type XIX regulates cardiac extracellular matrix structure and ventricular function. <i>Matrix Biology</i> , 2022, 109, 49-69.	1.5	6
93	Chronic Benzene Exposure Aggravates Pressure Overload-Induced Cardiac Dysfunction. <i>Toxicological Sciences</i> , 2021, 185, 64-76.	1.4	5
94	Why publish in the <i>American Journal of Physiology-Heart and Circulatory Physiology</i> ?. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2017, 313, H221-H223.	1.5	4
95	Cardiac mesenchymal cells from failing and nonfailing hearts limit ventricular dilation when administered late after infarction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2020, 319, H109-H122.	1.5	4
96	Loss of O ⁶ -GlcNAc transferase activity sensitizes cardiac myocytes to post-hypoxic death. <i>FASEB Journal</i> , 2008, 22, 750.10.	0.2	2
97	O ⁶ -GlcNAc signaling attenuates mitochondrial permeability transition. <i>FASEB Journal</i> , 2008, 22, .	0.2	2
98	AMP-Dependent Protein Kinase Activators. <i>Circulation Research</i> , 2009, 104, 282-284.	2.0	1
99	Angiotensinogen Takes Some of the Spotlight From Angiotensin II in the Cardiohepatic Axis. <i>Circulation Research</i> , 2021, 129, 565-567.	2.0	1
100	Protein O ⁶ -GlcNAcylation â€” A Novel Cell Survival Signal in Cardiac Stem Cells. <i>FASEB Journal</i> , 2012, 26, 693.1.	0.2	1
101	The Sweet Smell of Progress With Hyaluronan and Heart Failure. <i>Hypertension</i> , 2021, 77, 1928-1930.	1.3	0
102	Leukocyte-Endothelial Interactions Following Myocardial Ischemia. , 2003, , 427-438.		0
103	O ⁶ -GlcNAcase Exacerbates Post-hypoxic Cardiac Myocyte Death. <i>FASEB Journal</i> , 2007, 21, A1376.	0.2	0
104	O ⁶ -GlcNAc Transferase is a Pro-survival Enzyme in Post-hypoxic Cardiac Myocytes. <i>FASEB Journal</i> , 2007, 21, A800.	0.2	0
105	Hexosamine signaling reduces ER stress-induced cardiomyocyte death. <i>FASEB Journal</i> , 2009, 23, 991.7.	0.2	0
106	TRO40303 attenuates oxidant-induced mitochondrial dysfunction in cardiac myocytes. <i>FASEB Journal</i> , 2009, 23, LB71.	0.2	0
107	Protein O ⁶ -GlcNAcylation Exerts Mitogenic Effects in Cardiac Progenitor Cells. <i>FASEB Journal</i> , 2011, 25, 1043.16.	0.2	0
108	Protein O ⁶ -GlcNAcylation Promotes Post-hypoxic Survival of Cardiac Progenitor Cells. <i>FASEB Journal</i> , 2011, 25, 861.12.	0.2	0