# Steven P Jones

## List of Publications by Year in Descending Order



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| 99 papers | 6,835 citations | 47 h-index | 82 g-index |
| 109 ext. papers | 7,558 ext. citations | 7.4 avg, IF | 5.52 L-index |

| # | Paper | IF | Citations |
|---|---|---|---|
| 99 | Guidelines for in vivo mouse models of myocardial infarction. *American Journal of Physiology - Heart and Circulatory Physiology*, **2021**, 321, H1056-H1073 | 5.2 | 7 |
| 98 | Cardiac PANK1 deletion exacerbates ventricular dysfunction during pressure overload. *American Journal of Physiology - Heart and Circulatory Physiology*, **2021**, 321, H784-H797 | 5.2 | 0 |
| 97 | Cardiac mesenchymal cells from failing and nonfailing hearts limit ventricular dilation when administered late after infarction. *American Journal of Physiology - Heart and Circulatory Physiology*, **2020**, 319, H109-H122 | 5.2 | 2 |
| 96 | Cardiomyocyte Oga haploinsufficiency increases O-GlcNAcylation but hastens ventricular dysfunction following myocardial infarction. *PLoS ONE*, **2020**, 15, e0242250 | 3.7 | 2 |
| 95 | E2f1 deletion attenuates infarct-induced ventricular remodeling without affecting O-GlcNAcylation. *Basic Research in Cardiology*, **2019**, 114, 28 | 11.8 | 20 |
| 94 | Physiological Biomimetic Culture System for Pig and Human Heart Slices. *Circulation Research*, **2019** , 125, 628-642 | 15.7 | 29 |
| 93 | Metabolic regulation of Kv channels and cardiac repolarization by Kv subunits. *Journal of Molecular and Cellular Cardiology*, **2019**, 137, 93-106 | 5.8 | 7 |
| 92 | RDH10 function is necessary for spontaneous fetal mouth movement that facilitates palate shelf elevation. *DMM Disease Models and Mechanisms*, **2019**, 12, | 4.1 | 6 |
| 91 | Regulates Igf2bp2 Translation in Cardiomyocytes. *Circulation Research*, **2018**, 122, 1347-1353 | 15.7 | 17 |
| 90 | Deficiency of aldose reductase exacerbates early pressure overload-induced cardiac dysfunction and autophagy in mice. *Journal of Molecular and Cellular Cardiology*, **2018**, 118, 183-192 | 5.8 | 15 |
| 89 | RNA Editing: Unexplored Opportunities in the Cardiovascular System. *Circulation Research*, **2018**, 122, 399-401 | 15.7 | 17 |
| 88 | Guidelines for experimental models of myocardial ischemia and infarction. *American Journal of Physiology - Heart and Circulatory Physiology*, **2018**, 314, H812-H838 | 5.2 | 249 |
| 87 | Cardiac-specific overexpression of aldehyde dehydrogenase 2 exacerbates cardiac remodeling in response to pressure overload. *Redox Biology*, **2018**, 17, 440-449 | 11.3 | 7 |
| 86 | Leukocyte iNOS is required for inflammation and pathological remodeling in ischemic heart failure. *Basic Research in Cardiology*, **2017**, 112, 19 | 11.8 | 46 |
| 85 | Cardiomyocyte Ogt limits ventricular dysfunction in mice following pressure overload without affecting hypertrophy. *Basic Research in Cardiology*, **2017**, 112, 23 | 11.8 | 24 |
| 84 | Exercise-Induced Changes in Glucose Metabolism Promote Physiological Cardiac Growth. *Circulation*, **2017**, 136, 2144-2157 | 16.7 | 63 |
| 83 | Integration of flux measurements to resolve changes in anabolic and catabolic metabolism in cardiac myocytes. *Biochemical Journal*, **2017**, 474, 2785-2801 | 3.8 | 36 |

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| 82 | A New Method to Stabilize C-Kit Expression in Reparative Cardiac Mesenchymal Cells. *Frontiers in Cell and Developmental Biology*, **2016**, 4, 78 | 5.7 | 27 |
| 81 | TNF receptor signaling inhibits cardiomyogenic differentiation of cardiac stem cells and promotes a neuroadrenergic-like fate. *American Journal of Physiology - Heart and Circulatory Physiology*, **2016**, 311, H1189-H1201 | 5.2 | 9 |
| 80 | High glucose induces mitochondrial dysfunction independently of protein O-GlcNAcylation. *Biochemical Journal*, **2015**, 467, 115-26 | 3.8 | 31 |
| 79 | E2F1 Transcription Factor Regulates O-linked N-acetylglucosamine (O-GlcNAc) Transferase and O-GlcNAcase Expression. *Journal of Biological Chemistry*, **2015**, 290, 31013-24 | 5.4 | 22 |
| 78 | Induction of activating transcription factor 3 limits survival following infarct-induced heart failure in mice. *American Journal of Physiology - Heart and Circulatory Physiology*, **2015**, 309, H1326-35 | 5.2 | 12 |
| 77 | Recent Developments in Heart Failure. *Circulation Research*, **2015**, 117, e58-63 | 15.7 | 36 |
| 76 | O-GlcNAcylation Negatively Regulates Cardiomyogenic Fate in Adult Mouse Cardiac Mesenchymal Stromal Cells. *PLoS ONE*, **2015**, 10, e0142939 | 3.7 | 4 |
| 75 | The NHLBI-sponsored Consortium for preclinicAl assESsment of cARdioprotective therapies (CAESAR): a new paradigm for rigorous, accurate, and reproducible evaluation of putative infarct-sparing interventions in mice, rabbits, and pigs. *Circulation Research*, **2015**, 116, 572-86 | 15.7 | 111 |
| 74 | Metabolomic analysis of pressure-overloaded and infarcted mouse hearts. *Circulation: Heart Failure*, **2014**, 7, 634-42 | 7.6 | 130 |
| 73 | Cardiomyocyte Ogt is essential for postnatal viability. *American Journal of Physiology - Heart and Circulatory Physiology*, **2014**, 306, H142-53 | 5.2 | 47 |
| 72 | O-GlcNAc and the cardiovascular system. *Pharmacology & Therapeutics*, **2014**, 142, 62-71 | 13.9 | 93 |
| 71 | Endothelial dysfunction as a nexus for endothelial cell-cardiomyocyte miscommunication. *Frontiers in Physiology*, **2014**, 5, 328 | 4.6 | 21 |
| 70 | MicroRNA-539 is up-regulated in failing heart, and suppresses O-GlcNAcase expression. *Journal of Biological Chemistry*, **2014**, 289, 29665-76 | 5.4 | 55 |
| 69 | O-GlcNAc signaling entrains the circadian clock by inhibiting BMAL1/CLOCK ubiquitination. *Cell Metabolism*, **2013**, 17, 303-10 | 24.6 | 137 |
| 68 | PDGF-mediated autophagy regulates vascular smooth muscle cell phenotype and resistance to oxidative stress. *Biochemical Journal*, **2013**, 451, 375-88 | 3.8 | 147 |
| 67 | Protein O-GlcNAcylation is a novel cytoprotective signal in cardiac stem cells. *Stem Cells*, **2013**, 31, 765-75 | 5.8 | 47 |
| 66 | Reduced cardiac fructose 2,6 bisphosphate increases hypertrophy and decreases glycolysis following aortic constriction. *PLoS ONE*, **2013**, 8, e53951 | 3.7 | 16 |
| 65 | High fat feeding in mice is insufficient to induce cardiac dysfunction and does not exacerbate heart failure. *PLoS ONE*, **2013**, 8, e83174 | 3.7 | 60 |

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| 64 | O-GlcNAc signaling is essential for NFAT-mediated transcriptional reprogramming during cardiomyocyte hypertrophy. *American Journal of Physiology - Heart and Circulatory Physiology*, **2012**, 302, H2122-30 | 5.2 | 77 |
| 63 | Standardized bioenergetic profiling of adult mouse cardiomyocytes. *Physiological Genomics*, **2012**, 44, 1208-13 | 3.6 | 44 |
| 62 | The COX-2/PGI2 receptor axis plays an obligatory role in mediating the cardioprotection conferred by the late phase of ischemic preconditioning. *PLoS ONE*, **2012**, 7, e41178 | 3.7 | 26 |
| 61 | Protein O-GlcNAcylation □A Novel Cell Survival Signal in Cardiac Stem Cells. *FASEB Journal*, **2012**, 26, 693.1 | 0.9 | 1 |
| 60 | Cardiac overexpression of 8-oxoguanine DNA glycosylase 1 protects mitochondrial DNA and reduces cardiac fibrosis following transaortic constriction. *American Journal of Physiology - Heart and Circulatory Physiology*, **2011**, 301, H2073-80 | 5.2 | 39 |
| 59 | Responses of hypertrophied myocytes to reactive species: implications for glycolysis and electrophile metabolism. *Biochemical Journal*, **2011**, 435, 519-28 | 3.8 | 23 |
| 58 | Bioenergetic function in cardiovascular cells: the importance of the reserve capacity and its biological regulation. *Chemico-Biological Interactions*, **2011**, 191, 288-95 | 5 | 111 |
| 57 | Augmented O-GlcNAc signaling attenuates oxidative stress and calcium overload in cardiomyocytes. *Amino Acids*, **2011**, 40, 895-911 | 3.5 | 121 |
| 56 | Protein O-GlcNAcylation Exerts Mitogenic Effects in Cardiac Progenitor Cells. *FASEB Journal*, **2011**, 25, 1043.16 | 0.9 | |
| 55 | Protein O-GlcNAcylation Promotes Post-hypoxic Survival of Cardiac Progenitor Cells. *FASEB Journal* , **2011**, 25, 861.12 | 0.9 | |
| 54 | TRO40303, a new cardioprotective compound, inhibits mitochondrial permeability transition. *Journal of Pharmacology and Experimental Therapeutics*, **2010**, 333, 696-706 | 4.7 | 94 |
| 53 | O-linked □N-acetylglucosamine transferase is indispensable in the failing heart. *Proceedings of the National Academy of Sciences of the United States of America*, **2010**, 107, 17797-802 | 11.5 | 140 |
| 52 | O-GlcNAc signaling in the cardiovascular system. *Circulation Research*, **2010**, 107, 171-85 | 15.7 | 125 |
| 51 | Unique hexosaminidase reduces metabolic survival signal and sensitizes cardiac myocytes to hypoxia/reoxygenation injury. *Circulation Research*, **2009**, 104, 41-9 | 15.7 | 93 |
| 50 | O-GlcNAc signaling attenuates ER stress-induced cardiomyocyte death. *American Journal of Physiology - Heart and Circulatory Physiology*, **2009**, 297, H1711-9 | 5.2 | 84 |
| 49 | Hexosamine signaling reduces ER stress-induced cardiomyocyte death. *FASEB Journal*, **2009**, 23, 991.7 | 0.9 | |
| 48 | TRO40303 attenuates oxidant-induced mitochondrial dysfunction in cardiac myocytes. *FASEB Journal*, **2009**, 23, LB71 | 0.9 | |
| 47 | Non-canonical glycosyltransferase modulates post-hypoxic cardiac myocyte death and mitochondrial permeability transition. *Journal of Molecular and Cellular Cardiology*, **2008**, 45, 313-25 | 5.8 | 92 |

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| 46 | Post-transcriptional gene silencing of KChIP2 and Navbeta1 in neonatal rat cardiac myocytes reveals a functional association between Na and Ito currents. *Journal of Molecular and Cellular Cardiology*, **2008**, 45, 336-46 | 5.8 | 64 |
| 45 | New insights into metabolic signaling and cell survival: the role of beta-O-linkage of N-acetylglucosamine. *Journal of Pharmacology and Experimental Therapeutics*, **2008**, 327, 602-9 | 4.7 | 29 |
| 44 | Cardiac phosphatase-deficient 6-phosphofructo-2-kinase/fructose-2,6-bisphosphatase increases glycolysis, hypertrophy, and myocyte resistance to hypoxia. *American Journal of Physiology - Heart and Circulatory Physiology*, **2008**, 294, H2889-97 | 5.2 | 27 |
| 43 | Cardioprotection by N-acetylglucosamine linkage to cellular proteins. *Circulation*, **2008**, 117, 1172-82 | 16.7 | 179 |
| 42 | Loss of O-GlcNAc transferase activity sensitizes cardiac myocytes to post-hypoxic death. *FASEB Journal*, **2008**, 22, 750.10 | 0.9 | 2 |
| 41 | O-GlcNAc signaling attenuates mitochondrial permeability transition. *FASEB Journal*, **2008**, 22, | 0.9 | 2 |
| 40 | O-GlcNAcase Exacerbates Post-Hypoxic Cardiac Myocyte Death. *FASEB Journal*, **2007**, 21, A1376 | 0.9 | |
| 39 | O-GlcNAc Transferase is a Pro-Survival Enzyme in Post-Hypoxic Cardiac Myocytes. *FASEB Journal*, **2007**, 21, A800 | 0.9 | |
| 38 | Low-dose simvastatin improves survival and ventricular function via eNOS in congestive heart failure. *American Journal of Physiology - Heart and Circulatory Physiology*, **2006**, 291, H2743-51 | 5.2 | 28 |
| 37 | The ubiquitous role of nitric oxide in cardioprotection. *Journal of Molecular and Cellular Cardiology*, **2006**, 40, 16-23 | 5.8 | 356 |
| 36 | Functional integration of electrically active cardiac derivatives from genetically engineered human embryonic stem cells with quiescent recipient ventricular cardiomyocytes: insights into the development of cell-based pacemakers. *Circulation*, **2005**, 111, 11-20 | 16.7 | 416 |
| 35 | Deficiency of iNOS does not attenuate severe congestive heart failure in mice. *American Journal of Physiology - Heart and Circulatory Physiology*, **2005**, 288, H365-70 | 5.2 | 33 |
| 34 | A bittersweet modification: O-GlcNAc and cardiac dysfunction. *Circulation Research*, **2005**, 96, 925-6 | 15.7 | 13 |
| 33 | Endothelial nitric oxide synthase overexpression attenuates myocardial reperfusion injury. *American Journal of Physiology - Heart and Circulatory Physiology*, **2004**, 286, H276-82 | 5.2 | 163 |
| 32 | Role of intracellular antioxidant enzymes after in vivo myocardial ischemia and reperfusion. *American Journal of Physiology - Heart and Circulatory Physiology*, **2003**, 284, H277-82 | 5.2 | 63 |
| 31 | Differential actions of cardioprotective agents on the mitochondrial death pathway. *Circulation Research*, **2003**, 92, 195-202 | 15.7 | 73 |
| 30 | Uncoupling protein-2 overexpression inhibits mitochondrial death pathway in cardiomyocytes. *Circulation Research*, **2003**, 93, 192-200 | 15.7 | 267 |
| 29 | Endothelial cell overexpression of fas ligand attenuates ischemia-reperfusion injury in the heart. *Journal of Biological Chemistry*, **2003**, 278, 15185-91 | 5.4 | 33 |

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| 28 | Cariporide (HOE642), a selective Na+-H+ exchange inhibitor, inhibits the mitochondrial death pathway. *Circulation*, **2003**, 108, 2275-81 | 16.7 | 99 |
| 27 | Endothelial nitric oxide synthase overexpression attenuates congestive heart failure in mice. *Proceedings of the National Academy of Sciences of the United States of America*, **2003**, 100, 4891-6 | 11.5 | 190 |
| 26 | Simvastatin attenuates oxidant-induced mitochondrial dysfunction in cardiac myocytes. *Circulation Research*, **2003**, 93, 697-9 | 15.7 | 98 |
| 25 | Leukocyte-Endothelial Interactions Following Myocardial Ischemia **2003**, 427-438 | | |
| 24 | Differential response to myocardial reperfusion injury in eNOS-deficient mice. *American Journal of Physiology - Heart and Circulatory Physiology*, **2002**, 282, H2422-6 | 5.2 | 63 |
| 23 | Direct vascular and cardioprotective effects of rosuvastatin, a new HMG-CoA reductase inhibitor. *Journal of the American College of Cardiology*, **2002**, 40, 1172-8 | 15.1 | 116 |
| 22 | Cardioprotective actions of acute HMG-CoA reductase inhibition in the setting of myocardial infarction. *Acta Physiologica Scandinavica*, **2001**, 173, 139-43 | | 36 |
| 21 | Simvastatin exerts both anti-inflammatory and cardioprotective effects in apolipoprotein E-deficient mice. *Circulation*, **2001**, 103, 2598-603 | 16.7 | 174 |
| 20 | Pretreatment with simvastatin attenuates myocardial dysfunction after ischemia and chronic reperfusion. *Arteriosclerosis, Thrombosis, and Vascular Biology*, **2001**, 21, 2059-64 | 9.4 | 80 |
| 19 | Acute exposure to a high cholesterol diet attenuates myocardial ischemia-reperfusion injury in cholesteryl ester transfer protein mice. *Coronary Artery Disease*, **2001**, 12, 37-44 | 1.4 | 12 |
| 18 | Heart-targeted overexpression of caspase3 in mice increases infarct size and depresses cardiac function. *Proceedings of the National Academy of Sciences of the United States of America*, **2001**, 98, 9977-82 | 11.5 | 132 |
| 17 | HMG-CoA reductase inhibition protects the diabetic myocardium from ischemia-reperfusion injury. *FASEB Journal*, **2001**, 15, 1454-6 | 0.9 | 77 |
| 16 | Cardioprotective actions of endogenous IL-10 are independent of iNOS. *American Journal of Physiology - Heart and Circulatory Physiology*, **2001**, 281, H48-52 | 5.2 | 36 |
| 15 | CD8(+)-T-cell depletion ameliorates circulatory shock in Plasmodium berghei-infected mice. *Infection and Immunity*, **2001**, 69, 7341-8 | 3.7 | 64 |
| 14 | Myocardial reperfusion injury in neuronal nitric oxide synthase deficient mice. *Coronary Artery Disease*, **2000**, 11, 593-7 | 1.4 | 25 |
| 13 | The MEK1-ERK1/2 signaling pathway promotes compensated cardiac hypertrophy in transgenic mice. *EMBO Journal*, **2000**, 19, 6341-50 | 13 | 585 |
| 12 | Using gene-targeted mice to investigate the pathophysiology of myocardial reperfusion injury. *Basic Research in Cardiology*, **2000**, 95, 499-502 | 11.8 | 8 |
| 11 | PR-39, a potent neutrophil inhibitor, attenuates myocardial ischemia-reperfusion injury in mice. *American Journal of Physiology - Heart and Circulatory Physiology*, **2000**, 279, H2824-8 | 5.2 | 53 |

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| 10 | 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, H2196-201 | 5.2 | 64 |
| 9 | Myocardial Reperfusion Injury: Insights Gained from Gene-Targeted Mice. *Physiology*, **2000**, 15, 303-308 | 9.8 | 6 |
| 8 | Myocardial ischemia/reperfusion injury in NADPH oxidase-deficient mice. *Circulation Research*, **2000**, 87, 812-7 | 15.7 | 79 |
| 7 | Myocardial ischemia-reperfusion injury is exacerbated in absence of endothelial cell nitric oxide synthase. *American Journal of Physiology - Heart and Circulatory Physiology*, **1999**, 276, H1567-73 | 5.2 | 133 |
| 6 | Ischemic preconditioning prevents postischemic P-selectin expression in the rat small intestine. *American Journal of Physiology - Heart and Circulatory Physiology*, **1999**, 277, H2476-81 | 5.2 | 22 |
| 5 | Leukocyte-endothelial cell interactions in nitric oxide synthase-deficient mice. *American Journal of Physiology - Heart and Circulatory Physiology*, **1999**, 276, H1943-50 | 5.2 | 117 |
| 4 | 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, H763-9 | 5.2 | 48 |
| 3 | Effects of hypercholesterolemia on myocardial ischemia-reperfusion injury in LDL receptor-deficient mice. *Arteriosclerosis, Thrombosis, and Vascular Biology*, **1999**, 19, 2776-81 | 9.4 | 53 |
| 2 | Myocardial ischemia-reperfusion injury in CD18- and ICAM-1-deficient mice. *American Journal of Physiology - Heart and Circulatory Physiology*, **1998**, 275, H2300-7 | 5.2 | 51 |
| 1 | Coronary endothelial P-selectin in pathogenesis of myocardial ischemia-reperfusion injury. *American Journal of Physiology - Heart and Circulatory Physiology*, **1998**, 275, H1865-72 | 5.2 | 37 |