Yiru Guo

List of Publications by Year in descending order

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94	7,885	43973 48	51492 86
papers	citations	h-index	g-index

99	99	99	7153
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Transient Cell Cycle Induction in Cardiomyocytes to Treat Subacute Ischemic Heart Failure. Circulation, 2022, 145, 1339-1355.	1.6	27
2	Comparison of One and Three Intraventricular Injections of Cardiac Progenitor Cells in a Murine Model of Chronic Ischemic Cardiomyopathy. Stem Cell Reviews and Reports, 2021, 17, 604-615.	1.7	9
3	Echocardiography-guided percutaneous left ventricular intracavitary injection as a cell delivery approach in infarcted mice. Molecular and Cellular Biochemistry, 2021, 476, 2135-2148.	1.4	5
4	After the storm: an objective appraisal of the efficacy of c-kit+ cardiac progenitor cells in preclinical models of heart disease. Canadian Journal of Physiology and Pharmacology, 2021, 99, 129-139.	0.7	25
5	Comparison of Repeated Doses of C-kit-Positive Cardiac Cells versus a Single Equivalent Combined Dose in a Murine Model of Chronic Ischemic Cardiomyopathy. International Journal of Molecular Sciences, 2021, 22, 3145.	1.8	2
6	Cardiac Mesenchymal Cells Cultured at Physiologic Oxygen Tension Have Superior Therapeutic Efficacy in Heart Failure Caused by Myocardial Infarction. Frontiers in Cell and Developmental Biology, 2021, 9, 662415.	1.8	1
7	Single dose of synthetic microRNA-199a or microRNA-149 mimic does not improve cardiac function in a murine model of myocardial infarction. Molecular and Cellular Biochemistry, 2021, 476, 4093-4106.	1.4	3
8	Exercise-induced late preconditioning in mice is triggered by eNOS-dependent generation of nitric oxide and activation of PKCÎµ and is mediated by increased iNOS activity. International Journal of Cardiology, 2021, 340, 68-78.	0.8	11
9	Physiological Oxygen Tension Enhances Competence and Functional Properties of Murine Cardiac Mesenchymal Cells. Stem Cell Reviews and Reports, 2021, 17, 900-910.	1.7	5
10	Effects of Heme Oxygenase-1 on c-Kit-Positive Cardiac Cells. International Journal of Molecular Sciences, 2021, 22, 13448.	1.8	2
11	Administration of cardiac mesenchymal cells modulates innate immunity in the acute phase of myocardial infarction in mice. Scientific Reports, 2020, 10, 14754.	1.6	10
12	Cardiospecific Overexpression of ATPGD1 (Carnosine Synthase) Increases Histidine Dipeptide Levels and Prevents Myocardial Ischemia Reperfusion Injury. Journal of the American Heart Association, 2020, 9, e015222.	1.6	27
13	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.	1.5	4
14	Inducible cardiac-specific overexpression of cyclooxygenase-2 (COX-2) confers resistance to ischemia/reperfusion injury. Basic Research in Cardiology, 2019, 114, 32.	2.5	13
15	TRPA1 channel contributes to myocardial ischemia-reperfusion injury. American Journal of Physiology - Heart and Circulatory Physiology, 2019, 316, H889-H899.	1.5	42
16	Cardiac mesenchymal cells from diabetic mice are ineffective for cell therapy-mediated myocardial repair. Basic Research in Cardiology, 2018, 113, 46.	2.5	41
17	Repeated doses of cardiac mesenchymal cells are therapeutically superior to a single dose in mice with old myocardial infarction. Basic Research in Cardiology, 2017, 112, 18.	2.5	76
18	Myocardial Reparative Properties of Cardiac Mesenchymal Cells IsolatedÂonÂtheÂBasis of Adherence. Journal of the American College of Cardiology, 2017, 69, 1824-1838.	1.2	45

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19	WLAN interference selfâ€optimization using som neural networks. Concurrency Computation Practice and Experience, 2017, 29, e3913.	1.4	6
20	An Optimal Routing Algorithm in Service Customized 5G Networks. Mobile Information Systems, 2016, 2016, 1-7.	0.4	14
21	Preconditioning Human Cardiac Stem Cells with an HO-1 Inducer Exerts Beneficial Effects After Cell Transplantation in the Infarcted Murine Heart. Stem Cells, 2015, 33, 3596-3607.	1.4	39
22	The NHLBI-Sponsored Consortium for preclinicAl assESsment of cARdioprotective Therapies (CAESAR). Circulation Research, 2015, 116, 572-586.	2.0	164
23	Genetic Deficiency of Glutathione S -Transferase P Increases Myocardial Sensitivity to Ischemiaâ€“Reperfusion Injury. Circulation Research, 2015, 117, 437-449.	2.0	34
24	c-kit+ Cardiac Stem Cells Alleviate Post-Myocardial Infarction Left Ventricular Dysfunction Despite Poor Engraftment and Negligible Retention in the Recipient Heart. PLoS ONE, 2014, 9, e96725.	1.1	158
25	Endoplasmic reticulum stress-dependent activation of ATF3 mediates the late phase of ischemic preconditioning. Journal of Molecular and Cellular Cardiology, 2014, 76, 138-147.	0.9	34
26	Sodium Nitrite Fails to Limit Myocardial Infarct Size: Results from the CAESAR Cardioprotection Consortium (LB645). FASEB Journal, 2014, 28, LB645.	0.2	18
27	Administration of Sildenafil at Reperfusion Fails to Reduce Infarct Size: Results from the CAESAR Cardioprotection Consortium (LB650). FASEB Journal, 2014, 28, LB650.	0.2	15
28	A highly sensitive and accurate method to quantify absolute numbers of c-kit+ cardiac stem cells following transplantation in mice. Basic Research in Cardiology, 2013, 108, 346.	2.5	114
29	A Self-Organizing Distributed Memory Cache for Data Sharing Applications in Cluster Environment. , 2013, , .		1
30	GCMR: A GPU Cluster-Based MapReduce Framework for Large-Scale Data Processing. , 2013, , .		2
31	The Heme Oxygenase 1 Inducer (CoPP) Protects Human Cardiac Stem Cells against Apoptosis through Activation of the Extracellular Signal-regulated Kinase (ERK)/NRF2 Signaling Pathway and Cytokine Release. Journal of Biological Chemistry, 2012, 287, 33720-33732.	1.6	89
32	Carbon monoxide induces a late preconditioning-mimetic cardioprotective and antiapoptotic milieu in the myocardium. Journal of Molecular and Cellular Cardiology, 2012, 52, 228-236.	0.9	78
33	Genetic background, gender, age, body temperature, and arterial blood pH have a major impact on myocardial infarct size in the mouse and need to be carefully measured and/or taken into account: results of a comprehensive analysis of determinants of infarct size in 1,074 mice. Basic Research in Cardiology, 2012, 107, 288.	2.5	44
34	Identification of inducible nitric oxide synthase in peripheral blood cells as a mediator of myocardial ischemia/reperfusion injury. Basic Research in Cardiology, 2012, 107, 253.	2.5	25
35	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.	1.1	30
36	A murine model of inducible, cardiac-specific deletion of STAT3: Its use to determine the role of STAT3 in the upregulation of cardioprotective proteins by ischemic preconditioning. Journal of Molecular and Cellular Cardiology, 2011, 50, 589-597.	0.9	87

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37	Transplantation of expanded bone marrowâ€derived very small embryonicâ€like stem cells (VSELâ€SCs) improves left ventricular function and remodelling after myocardial infarction. Journal of Cellular and Molecular Medicine, 2011, 15, 1319-1328.	1.6	73
38	Intracoronary administration of cardiac stem cells in mice: a new, improved technique for cell therapy in murine models. Basic Research in Cardiology, 2011, 106, 849-864.	2.5	106
39	Gene transfer as a strategy to achieve permanent cardioprotection I: rAAV-mediated gene therapy with inducible nitric oxide synthase limits infarct size 1Âyear later without adverse functional consequences. Basic Research in Cardiology, 2011, 106, 1355-1366.	2.5	21
40	Gene transfer as a strategy to achieve permanent cardioprotection II: rAAV-mediated gene therapy with heme oxygenase-1 limits infarct size 1Âyear later without adverse functional consequences. Basic Research in Cardiology, 2011, 106, 1367-1377.	2.5	34
41	Cardiac Progenitor Cells and Bone Marrow-Derived Very Small Embryonic-Like Stem Cells for Cardiac Repair After Myocardial Infarction. Circulation Journal, 2010, 74, 390-404.	0.7	62
42	Gene Transfer of Inducible Nitric Oxide Synthase Affords Cardioprotection by Upregulating Heme Oxygenase-1 Via a Nuclear Factor-ÎºB-Dependent Pathway. Circulation, 2009, 120, 1222-1230.	1.6	50
43	The beneficial effects of postinfarct cytokine combination therapy are sustained during long-term follow-up. Journal of Molecular and Cellular Cardiology, 2009, 47, 528-535.	0.9	12
44	Transplantation of Bone Marrow-Derived Very Small Embryonic-Like Stem Cells Attenuates Left Ventricular Dysfunction and Remodeling After Myocardial Infarction. Stem Cells, 2008, 26, 1646-1655.	1.4	138
45	Endothelial nitric oxide synthase is not necessary for the early phase of ischemic preconditioning in the mouse. Journal of Molecular and Cellular Cardiology, 2008, 44, 496-501.	0.9	26
46	Bone marrow-derived pluripotent very small embryonic-like stem cells (VSELs) are mobilized after acute myocardial infarction. Journal of Molecular and Cellular Cardiology, 2008, 44, 865-873.	0.9	75
47	Acrolein consumption exacerbates myocardial ischemic injury and blocks nitric oxide-induced PKCÎµ signaling and cardioprotection. Journal of Molecular and Cellular Cardiology, 2008, 44, 1016-1022.	0.9	86
48	The role of TNF-Î± receptors p55 and p75 in acute myocardial ischemia/reperfusion injury and late preconditioning. Journal of Molecular and Cellular Cardiology, 2008, 45, 735-741.	0.9	42
49	Cardiac Myocyteâ€“Specific Expression of Inducible Nitric Oxide Synthase Protects Against Ischemia/Reperfusion Injury by Preventing Mitochondrial Permeability Transition. Circulation, 2008, 118, 1970-1978.	1.6	109
50	Cardioprotection of glutathione Sâ€transferase P against ischemiaâ€reperfusion injury. FASEB Journal, 2008, 22, 750.18.	0.2	0
51	Cardioprotection Afforded by Inducible Nitric Oxide Synthase Gene Therapy Is Mediated by Cyclooxygenase-2 via a Nuclear Factor-ÎºBâ€“Dependent Pathway. Circulation, 2007, 116, 1577-1584.	1.6	54
52	CRYAB and HSPB2 deficiency alters cardiac metabolism and paradoxically confers protection against myocardial ischemia in aging mice. American Journal of Physiology - Heart and Circulatory Physiology, 2007, 293, H3201-H3209.	1.5	40
53	Endothelial Nitric Oxide Synthase Plays an Obligatory Role in the Late Phase of Ischemic Preconditioning by Activating the Protein Kinase Cİµâ€“p44/42 Mitogen-Activated Protein Kinaseâ€“pSer-Signal Transducers and Activators of Transcription1/3 Pathway. Circulation, 2007, 116, 535-544.	1.6	73
54	Stromal Cellâ€“Derived Factor-1Î± Confers Protection Against Myocardial Ischemia/Reperfusion Injury. Circulation, 2007, 116, 654-663.	1.6	295

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55	Targeted disruption of cox-2 gene abrogates the late phase of ischemic preconditioning. Journal of Molecular and Cellular Cardiology, 2007, 42, S181.	0.9	0
56	Glutathione-S-transferase-P deficiency in mice enhances myocardial sensitivity to ischemiaâ€“reperfusion. Journal of Molecular and Cellular Cardiology, 2007, 42, S198.	0.9	0
57	The late phase of ischemic preconditioning induces a prosurvival genetic program that results in marked attenuation of apoptosis. Journal of Molecular and Cellular Cardiology, 2007, 42, 1075-1085.	0.9	24
58	The late phase of preconditioning and its natural clinical applicationâ€”gene therapy. Heart Failure Reviews, 2007, 12, 189-199.	1.7	64
59	An obligatory role of STAT1 in the upregulation of cardioprotective proteins and delayed cardioprotection in ischemic preconditioning. FASEB Journal, 2007, 21, A1376.	0.2	0
60	An In Vivo Evidence That Murine Very Small Embryonic Like (VSEL) Stem Cells Are Mobilized into Peripheral Blood after Acute Myocardial Infarction (AMI) and Contribute to Myocardiac Regeneration.. Blood, 2007, 110, 3694-3694.	0.6	0
61	Gene therapy with iNOS provides long-term protection against myocardial infarction without adverse functional consequences. American Journal of Physiology - Heart and Circulatory Physiology, 2006, 290, H584-H589.	1.5	45
62	Postinfarct Cytokine Therapy Regenerates Cardiac Tissue and Improves Left Ventricular Function. Circulation Research, 2006, 98, 1098-1105.	2.0	82
63	Late preconditioning induced by NO donors, adenosine A1 receptor agonists, and Î´1-opioid receptor agonists is mediated by iNOS. American Journal of Physiology - Heart and Circulatory Physiology, 2005, 289, H2251-H2257.	1.5	48
64	Proapoptotic Effects of Caspase-1/Interleukin-Converting Enzyme Dominate in Myocardial Ischemia. Circulation Research, 2005, 96, 1103-1109.	2.0	71
65	Role of the Protein Kinase C-Îµâ€“Raf-1â€“MEK-1/2â€“p44/42 MAPK Signaling Cascade in the Activation of Signal Transducers and Activators of Transcription 1 and 3 and Induction of Cyclooxygenase-2 After Ischemic Preconditioning. Circulation, 2005, 112, 1971-1978.	1.6	126
66	Overexpression of Mitogen-activated Protein Kinase Kinase 6 in the Heart Improves Functional Recovery from Ischemia in Vitro and Protects against Myocardial Infarction in Vivo. Journal of Biological Chemistry, 2005, 280, 669-676.	1.6	77
67	Administration of a CO-releasing molecule induces late preconditioning against myocardial infarction. Journal of Molecular and Cellular Cardiology, 2005, 38, 127-134.	0.9	137
68	IL-6 plays an obligatory role in late preconditioning via JAK?STAT signaling and upregulation of iNOS and COX-2. Cardiovascular Research, 2004, 64, 61-71.	1.8	183
69	Cells Expressing Early Cardiac Markers Reside in the Bone Marrow and Are Mobilized Into the Peripheral Blood After Myocardial Infarction. Circulation Research, 2004, 95, 1191-1199.	2.0	325
70	Delayed Adaptation of the Heart to Stress: Late Preconditioning. Stroke, 2004, 35, 2676-2679.	1.0	94
71	Administration of a CO-releasing molecule at the time of reperfusion reduces infarct size in vivo. American Journal of Physiology - Heart and Circulatory Physiology, 2004, 286, H1649-H1653.	1.5	193
72	Tumor necrosis factor-? does not modulate ischemia/reperfusion injury in naï¿½ve myocardium but is essential for the development of late preconditioning*1. Journal of Molecular and Cellular Cardiology, 2004, 37, 51-61.	0.9	64

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73	CXCR4+ CD45â" Tissue-Committed Stem Cells (TCSC) for Myocardium Reside in the Bone Marrow, Are Mobilized into the Peripheral Blood during Myocardial Infarction, and â€œHomeâ€•to Infarcted Myocardium in CXCR4-SDF-1 and HGF/SF-c-Met Dependent Manner.. Blood, 2004, 104, 2131-2131.	0.6	0
74	Mechanism of cyclooxygenase-2 upregulation in late preconditioning. Journal of Molecular and Cellular Cardiology, 2003, 35, 525-537.	0.9	92
75	Gene Therapy With Inducible Nitric Oxide Synthase Protects Against Myocardial Infarction via a Cyclooxygenase-2â€"Dependent Mechanism. Circulation Research, 2003, 92, 741-748.	2.0	76
76	MCC-134, a Single Pharmacophore, Opens Surface ATPâ€"Sensitive Potassium Channels, Blocks Mitochondrial ATPâ€"Sensitive Potassium Channels, and Suppresses Preconditioning. Circulation, 2003, 107, 1183-1188.	1.6	31
77	Protein Kinase CÎµ Interacts With and Inhibits the Permeability Transition Pore in Cardiac Mitochondria. Circulation Research, 2003, 92, 873-880.	2.0	433
78	Gene Dosage-Dependent Effects of Cardiac-Specific Overexpression of the A3Adenosine Receptor. Circulation Research, 2002, 91, 165-172.	2.0	77
79	Discovery of a new function of cyclooxygenase (COX)-2: COX-2 is a cardioprotective protein that alleviates ischemia/reperfusion injury and mediates the late phase of preconditioning. Cardiovascular Research, 2002, 55, 506-519.	1.8	220
80	Ischemic Preconditioning Upregulates Inducible Nitric Oxide Synthase in Cardiac Myocyte. Journal of Molecular and Cellular Cardiology, 2002, 34, 5-15.	0.9	82
81	Formation of protein kinase CÎµ-Lck signaling modules confers cardioprotection. Journal of Clinical Investigation, 2002, 109, 499-507.	3.9	101
82	Formation of protein kinase CÎµ-Lck signaling modules confers cardioprotection. Journal of Clinical Investigation, 2002, 109, 499-507.	3.9	79
83	Targeted Deletion of the A3Adenosine Receptor Confers Resistance to Myocardial Ischemic Injury and does not Prevent Early Preconditioning. Journal of Molecular and Cellular Cardiology, 2001, 33, 825-830.	0.9	74
84	Exercise-induced late preconditioning is triggered by generation of nitric oxide. Journal of Molecular and Cellular Cardiology, 2001, 33, A41.	0.9	5
85	Gene Therapy With Extracellular Superoxide Dismutase Protects Conscious Rabbits Against Myocardial Infarction. Circulation, 2001, 103, 1893-1898.	1.6	140
86	An essential role of the JAK-STAT pathway in ischemic preconditioning. Proceedings of the National Academy of Sciences of the United States of America, 2001, 98, 9050-9055.	3.3	256
87	Opposing cardioprotective actions and parallel hypertrophic effects of ÂPKC and ÂPKC. Proceedings of the National Academy of Sciences of the United States of America, 2001, 98, 11114-11119.	3.3	510
88	Evidence for an essential role of cyclooxygenase-2 as a mediator of the late phase of ischemic preconditioning in mice. Basic Research in Cardiology, 2000, 95, 479-484.	2.5	94
89	The late phase of ischemic preconditioning is abrogated by targeted disruption of the inducible NO synthase gene. Proceedings of the National Academy of Sciences of the United States of America, 1999, 96, 11507-11512.	3.3	377
90	The nitric oxide hypothesis of late preconditioning. Basic Research in Cardiology, 1998, 93, 325-338.	2.5	255

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91	Nitric Oxide Donors Induce Late Preconditioning Against Myocardial Stunning and Infarction in Conscious Rabbits via an Antioxidant-Sensitive Mechanism. Circulation Research, 1998, 83, 73-84.	2.0	230
92	Demonstration of an early and a late phase of ischemic preconditioning in mice. American Journal of Physiology - Heart and Circulatory Physiology, 1998, 275, H1375-H1387.	1.5	141
93	Evidence That Late Preconditioning Against Myocardial Stunning in Conscious Rabbits Is Triggered by the Generation of Nitric Oxide. Circulation Research, 1997, 81, 42-52.	2.0	211
94	The Protective Effect of Late Preconditioning Against Myocardial Stunning in Conscious Rabbits Is Mediated by Nitric Oxide Synthase. Circulation Research, 1997, 81, 1094-1107.	2.0	272