List of Publications by Year in descending order

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ΗινισΤλι

#	Article	IF	CITATIONS
1	Cardiac natriuretic peptide deficiency sensitizes the heart to stress-induced ventricular arrhythmias via impaired CREB signalling. Cardiovascular Research, 2022, 118, 2124-2138.	3.8	8
2	Isoform-Specific Role of GSK-3 in High Fat Diet Induced Obesity and Glucose Intolerance. Cells, 2022, 11, 559.	4.1	7
3	Targeting 5-HT _{2B} Receptor Signaling Prevents Border Zone Expansion and Improves Microstructural Remodeling After Myocardial Infarction. Circulation, 2021, 143, 1317-1330.	1.6	36
4	Repurposing Nintedanib for pathological cardiac remodeling and dysfunction. Pharmacological Research, 2021, 169, 105605.	7.1	10
5	Mechanisms of Fibroblast Activation and Myocardial Fibrosis: Lessons Learned from FB-Specific Conditional Mouse Models. Cells, 2021, 10, 2412.	4.1	27
6	Novel Mechanisms of Exosome-Mediated Phagocytosis of Dead Cells in Injured Heart. Circulation Research, 2021, 129, 1006-1020.	4.5	32
7	Abstract 117: Ponatinib Mediated Cardiotoxicity Is Driven By Pro-inflammatory S100A8/A9-NLRP3-IL-1β Signaling Circuit. Circulation Research, 2021, 129, .	4.5	0
8	Abstract P317: Cardiac Fibroblast GSK3α Promotes Myocardial Fibrotic Remodeling Through GSK3α-ERK-IL11 Signaling Circuit. Circulation Research, 2021, 129, .	4.5	0
9	Abstract 11334: Metabolic Labeling and Systemic Tracking of Cardiomyocyte-Derived Exosomal MiRNAs. Circulation, 2021, 144, .	1.6	0
10	The BDNF rs6265 Polymorphism is a Modifier of Cardiomyocyte Contractility and Dilated Cardiomyopathy. International Journal of Molecular Sciences, 2020, 21, 7466.	4.1	6
11	Deletion of Cardiomyocyte Glycogen Synthase Kinase-3 Beta (GSK-3β) Improves Systemic Glucose Tolerance with Maintained Heart Function in Established Obesity. Cells, 2020, 9, 1120.	4.1	7
12	Cardiotoxicity of the BCR-ABL1 tyrosine kinase inhibitors: Emphasis on ponatinib. International Journal of Cardiology, 2020, 316, 214-221.	1.7	38
13	A Pharmacovigilance Study of Hydroxychloroquine Cardiac Safety Profile: Potential Implication in COVID-19 Mitigation. Journal of Clinical Medicine, 2020, 9, 1867.	2.4	21
14	Neutrophil-Derived S100A8/A9 Amplify Granulopoiesis After Myocardial Infarction. Circulation, 2020, 141, 1080-1094.	1.6	155
15	Mouse Models of Heart Failure with Preserved or Reduced Ejection Fraction. American Journal of Pathology, 2020, 190, 1596-1608.	3.8	28
16	Abstract MP151: Cardiomyocyte HIPK2 is a Critical Regulator of Purinergic Signaling Regulated Myocardial Inflammation. Circulation Research, 2020, 127, .	4.5	0
17	Abstract 365: Ponatinib Triggers Cardiac Inflammation by STAT-3 Dependent Immune Checkpoint Blockade. Circulation Research, 2020, 127,	4.5	0
18	Abstract 285: High Throughput Profiling of Gsk3α Regulated Fibroblast Kinome Reveals Raf as a Mediator of Fibrosis in Failing Heart. Circulation Research, 2020, 127, .	4.5	0

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19	CARDIAC NATRIURETIC PEPTIDE DEFICIENCY PREDISPOSES TO ARRHYTHMIAS AND SUDDEN DEATH FOLLOWING ACUTE CARDIAC STRESS. Journal of the American College of Cardiology, 2019, 73, 891.	2.8	0
20	IL-10–producing B cells are enriched in murine pericardial adipose tissues and ameliorate the outcome of acute myocardial infarction. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 21673-21684.	7.1	62
21	Ponatinib-induced cardiotoxicity: delineating the signalling mechanisms and potential rescue strategies. Cardiovascular Research, 2019, 115, 966-977.	3.8	56
22	Cardiomyocyte Homeodomain-Interacting Protein Kinase 2 Maintains Basal Cardiac Function via Extracellular Signal-Regulated Kinase Signaling. Circulation, 2019, 140, 1820-1833.	1.6	21
23	Cardiomyocyte-CSK-3α promotes mPTP opening and heart failure in mice with chronic pressure overload. Journal of Molecular and Cellular Cardiology, 2019, 130, 65-75.	1.9	34
24	Generation of Nppaâ€ŧagBFP reporter knockâ€in mouse line for studying cardiac chamber specification. Genesis, 2019, 57, e23294.	1.6	2
25	Cardiomyocyte SMAD4-Dependent TGF-β Signaling is Essential to MaintainÂAdult Heart Homeostasis. JACC Basic To Translational Science, 2019, 4, 41-53.	4.1	35
26	Inhibition of GSK-3 to induce cardiomyocyte proliferation: a recipe for in situ cardiac regeneration. Cardiovascular Research, 2019, 115, 20-30.	3.8	31
27	Cadherin-11 blockade reduces inflammation-driven fibrotic remodeling and improves outcomes after myocardial infarction. JCI Insight, 2019, 4, .	5.0	33
28	Abstract 738: Comparative Cardiotoxicity of Tyrosine Kinase Inhibitors Ponatinib and PF114. Circulation Research, 2019, 125, .	4.5	0
29	Abstract 184: Cardioprotective Effects of Brain-derived Neurotrophic Factor rs6265 Polymorphism in Duchenne Cardiomyopathy. Circulation Research, 2019, 125, .	4.5	0
30	Abstract 530: Cardiac Fibroblast GSK-3α Contributes to Ventricular Remodeling and Dysfunction of the Failing Heart. Circulation Research, 2019, 125, .	4.5	0
31	Cardiomyocyte-specific deletion of CSK-3β leads to cardiac dysfunction in a diet induced obesity model. International Journal of Cardiology, 2018, 259, 145-152.	1.7	20
32	Abstract 361: Analysis of Cardiotoxic Mechanisms Associated With Tyrosine Kinase Inhibitor Ponatinib. Circulation Research, 2018, 123, .	4.5	0
33	Abstract 114: A Role for Brain-derived Neurotrophic Factor in Duchenne Cardiomyopathy. Circulation Research, 2018, 123, .	4.5	0
34	Activation of the Amino Acid Response Pathway Blunts the Effects of Cardiac Stress. Journal of the American Heart Association, 2017, 6, .	3.7	26
35	Chronic Neuregulin-1β Treatment Mitigates the Progression of Postmyocardial Infarction Heart Failure in the Setting of Type 1 Diabetes Mellitus by Suppressing Myocardial Apoptosis, Fibrosis, and Key Oxidant-Producing Enzymes. Journal of Cardiac Failure, 2017, 23, 887-899.	1.7	20
36	Mechanistic Insights of Empagliflozin-Mediated Cardiac Benefits: Nearing the Starting Line. Cardiovascular Drugs and Therapy, 2017, 31, 229-232.	2.6	5

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37	Entanglement of GSK-3β, β-catenin and TGF-β1 signaling network to regulate myocardial fibrosis. Journal of Molecular and Cellular Cardiology, 2017, 110, 109-120.	1.9	118
38	Abstract 249: Chronic Neuregulin-1β Treatment Mitigates the Progression of Post-myocardial Infarction Heart Failure in the Setting of Type 1 Diabetes Mellitus. Circulation Research, 2017, 121, .	4.5	0
39	Abstract 41: Cardiomyocyte-specific Conditional Deletion of GSK-3β Leads to Cardiac Dysfunction in a High Fat Diet Induced Obesity Model. Circulation Research, 2017, 121, .	4.5	0
40	Abstract 98: Canonical TGF-β1 Signaling in Cardiomyocytes is Essential to Maintain Basal Cardiac Function. Circulation Research, 2017, 121, .	4.5	0
41	Response by Zhou et al to Letter Regarding Article, "Loss of Adult Cardiac Myocyte GSK-3 Leads to Mitotic Catastrophe Resulting in Fatal Dilated Cardiomyopathy― Circulation Research, 2016, 119, e29-e30.	4.5	10
42	Loss of Adult Cardiac Myocyte GSK-3 Leads to Mitotic Catastrophe Resulting in Fatal Dilated Cardiomyopathy. Circulation Research, 2016, 118, 1208-1222.	4.5	92
43	Abstract 75: Cardiomyocyte-specific Conditional Deletion of GSK-3Î ² Leads to Global Metabolic Defects and Cardiac Dysfunction in a HFD Induced Obesity Model. Circulation Research, 2016, 119, .	4.5	0
44	Abstract 280: Cardiac Fibroblast Specific Deletion of Gsk3α Alleviate From Cardiac Dysfunction and Fibrotic Remodeling in Ischemic Heart. Circulation Research, 2016, 119, .	4.5	0
45	Abstract 69: Cardiomyocyte GSK-3α Signaling Exacerbate Pressure Overload-induced Dilated Cardiomyopathy and Heart Failure. Circulation Research, 2016, 119, .	4.5	0
46	The GSK-3 Family as Therapeutic Target for Myocardial Diseases. Circulation Research, 2015, 116, 138-149.	4.5	174
47	Prevention of liver cancer cachexia-induced cardiac wasting and heart failure. European Heart Journal, 2014, 35, 932-941.	2.2	167
48	Sorafenib Cardiotoxicity Increases Mortality After Myocardial Infarction. Circulation Research, 2014, 114, 1700-1712.	4.5	69
49	Cardiomyocyte-Specific Deletion of Gsk3αÂMitigates Post–Myocardial InfarctionÂRemodeling, Contractile Dysfunction, and Heart Failure. Journal of the American College of Cardiology, 2014, 64, 696-706.	2.8	63
50	Cardiac Fibroblast Glycogen Synthase Kinase-3Î ² Regulates Ventricular Remodeling and Dysfunction in Ischemic Heart. Circulation, 2014, 130, 419-430.	1.6	148
51	Troponin I-Interacting Protein Kinase. Circulation Journal, 2014, 78, 1514-1519.	1.6	20
52	Caveolin and β1-integrin coordinate angiotensinogen expression in cardiac myocytes. International Journal of Cardiology, 2013, 168, 436-445.	1.7	7
53	Anthrax lethal toxin induces acute diastolic dysfunction in rats through disruption of the phospholamban signaling network. International Journal of Cardiology, 2013, 168, 3884-3895.	1.7	8
54	Cancer Genetics and the Cardiotoxicity of the Therapeutics. Journal of the American College of Cardiology, 2013, 61, 267-274.	2.8	56

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55	GSK-3α is a central regulator of age-related pathologies in mice. Journal of Clinical Investigation, 2013, 123, 1821-1832.	8.2	137
56	Glycogen Synthase Kinase-3α Limits Ischemic Injury, Cardiac Rupture, Post–Myocardial Infarction Remodeling and Death. Circulation, 2012, 125, 65-75.	1.6	64
57	Cardiac Wasting in Experimental Cancer Cachexia: Prevention by Bisoprolol and Spironolactone. Journal of Cardiac Failure, 2011, 17, S10.	1.7	0
58	A novel cardioprotective p38-MAPK/mTOR pathway. Experimental Cell Research, 2011, 317, 2938-2949.	2.6	74
59	Rac1 and RhoA differentially regulate angiotensinogen gene expression in stretched cardiac fibroblasts. Cardiovascular Research, 2011, 90, 88-96.	3.8	52
60	Abstract P335: Is GSK-31 \pm a Regulator of Aging?. Circulation Research, 2011, 109, .	4.5	0
61	GSK-3α directly regulates β-adrenergic signaling and the response of the heart to hemodynamic stress in mice. Journal of Clinical Investigation, 2010, 120, 2280-2291.	8.2	54
62	Glycogen Synthase Kinase-3β Regulates Post–Myocardial Infarction Remodeling and Stress-Induced Cardiomyocyte Proliferation In Vivo. Circulation Research, 2010, 106, 1635-1645.	4.5	108
63	Anthrax toxin: pathologic effects on the cardiovascular system. Frontiers in Bioscience - Landmark, 2009, Volume, 2335.	3.0	23
64	Integrins and proximal signaling mechanisms in cardiovascular disease. Frontiers in Bioscience - Landmark, 2009, Volume, 2307.	3.0	70
65	Molecular Signaling Mechanisms of Myocardial Stretch: Implications for Heart Disease. , 2009, , 55-81.		3
66	Stretch-induced regulation of angiotensinogen gene expression in cardiac myocytes and fibroblasts: Opposing roles of JNK1/2 and p38α MAP kinases. Journal of Molecular and Cellular Cardiology, 2008, 45, 770-778.	1.9	33
67	The Sodium Pump: Bridging the Basic and Clinical Cardiovascular Sciences. Recent Patents on Endocrine, Metabolic & Immune Drug Discovery, 2007, 1, 224-246.	0.6	0
68	Integrins: Novel Therapeutic Targets for Cardiovascular Diseases. Cardiovascular and Hematological Agents in Medicinal Chemistry, 2007, 5, 109-132.	1.0	30
69	Stretch-induced MAP kinase activation in cardiac myocytes: Differential regulation through β1-integrin and focal adhesion kinase. Journal of Molecular and Cellular Cardiology, 2007, 43, 137-147.	1.9	84
70	Lethal and edema toxins of anthrax induce distinct hemodynamic dysfunction. Frontiers in Bioscience - Landmark, 2007, 12, 4670.	3.0	34
71	Stress induced phosphate solubilization in bacteria isolated from alkaline soils. FEMS Microbiology Letters, 2000, 182, 291-296.	1.8	325
72	Stress induced phosphate solubilization in bacteria isolated from alkaline soils. FEMS Microbiology Letters, 2000, 182, 291-296.	1.8	11