Rong Tian

List of Publications by Year
in descending order

147 papers	13,148 citations	22153 59 h-index	24982 109 g-index
155 all docs	155 docs citations	155 times ranked	14190 citing authors

#	Article	IF	Citations
1	Cardiac Metabolism and its Interactions With Contraction, Growth, and Survival of Cardiomyocytes. Circulation Research, 2013, 113, 603-616.	4.5	591
2	Mitochondrial dysfunction in pathophysiology of heart failure. Journal of Clinical Investigation, 2018, 128, 3716-3726.	8.2	498
3	Improvement of Cardiac Functions by Chronic Metformin Treatment Is Associated With Enhanced Cardiac Autophagy in Diabetic OVE26 Mice. Diabetes, 2011, 60, 1770-1778.	0.6	433
4	Cardiac Energy Metabolism in Heart Failure. Circulation Research, 2021, 128, 1487-1513.	4.5	433
5	Cardiac-Specific Expression of Heme Oxygenase-1 Protects Against Ischemia and Reperfusion Injury in Transgenic Mice. Circulation Research, 2001, 89, 168-173.	4.5	385
6	Mitochondrial Complex I Deficiency Increases Protein Acetylation and Accelerates Heart Failure. Cell Metabolism, 2013, 18, 239-250.	16.2	376
7	Acute Metformin Therapy Confers Cardioprotection Against Myocardial Infarction Via AMPK-eNOSâ€"Mediated Signaling. Diabetes, 2008, 57, 696-705.	0.6	373
8	Activation of AMP-Activated Protein Kinase by Metformin Improves Left Ventricular Function and Survival in Heart Failure. Circulation Research, 2009, 104, 403-411.	4.5	357
9	Cardiac hypertrophy with preserved contractile function after selective deletion of GLUT4 from the heart. Journal of Clinical Investigation, 1999, 104, 1703-1714.	8.2	310
10	Defective Branched-Chain Amino Acid Catabolism Disrupts Glucose Metabolism and Sensitizes the Heart to Ischemia-Reperfusion Injury. Cell Metabolism, 2017, 25, 374-385.	16.2	289
11	Cardiac-Specific Overexpression of GLUT1 Prevents the Development of Heart Failure Attributable to Pressure Overload in Mice. Circulation, 2002, 106, 2125-2131.	1.6	282
12	Increased Adenosine Monophosphateâ€"Activated Protein Kinase Activity in Rat Hearts With Pressure-Overload Hypertrophy. Circulation, 2001, 104, 1664-1669.	1.6	278
13	Normalization of NAD ⁺ Redox Balance as a Therapy for Heart Failure. Circulation, 2016, 134, 883-894.	1.6	250
14	Glucose metabolism and cardiac hypertrophy. Cardiovascular Research, 2011, 90, 194-201.	3.8	241
15	Cardiac-Specific Deletion of Acetyl CoA Carboxylase 2 Prevents Metabolic Remodeling During Pressure-Overload Hypertrophy. Circulation Research, 2012, 111, 728-738.	4.5	214
16	Impairment of energy metabolism in intact residual myocardium of rat hearts with chronic myocardial infarction.. Journal of Clinical Investigation, 1995, 95, 1092-1100.	8.2	208
17	Mechanisms for Increased Glycolysis in the Hypertrophied Rat Heart. Hypertension, 2004, 44, 662-667.	2.7	200
18	Responses of GLUT4-Deficient Hearts to Ischemia Underscore the Importance of Glycolysis. Circulation, 2001, 103, 2961-2966.	1.6	197

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19	Glucose Metabolism and Energy Homeostasis in Mouse Hearts Overexpressing Dominant Negative Î±2 Subunit of AMP-activated Protein Kinase. Journal of Biological Chemistry, 2003, 278, 28372-28377.	3.4	197
20	Defective DNA Replication Impairs Mitochondrial Biogenesis In Human Failing Hearts. Circulation Research, 2010, 106, 1541-1548.	4.5	192
21	Interaction of Insulin and AMPK in the Ischemic Heart. Circulation Research, 2006, 99, 3-5.	4.5	190
22	Targeting Mitochondria-Inflammation Circuit by Î²-Hydroxybutyrate Mitigates HFpEF. Circulation Research, 2021, 128, 232-245.	4.5	190
23	Metabolism in cardiomyopathy: every substrate matters. Cardiovascular Research, 2017, 113, 411-421.	3.8	188
24	An open-label, non-randomized study of the pharmacokinetics of the nutritional supplement nicotinamide riboside (NR) and its effects on blood NAD+ levels in healthy volunteers. PLoS ONE, 2017, 12, e0186459.	2.5	188
25	Fatty Acids Enhance the Maturation of Cardiomyocytes Derived from Human Pluripotent Stem Cells. Stem Cell Reports, 2019, 13, 657-668.	4.8	187
26	Impaired Cardiac Energetics in Mice Lacking Muscle-Specific Isoenzymes of Creatine Kinase. Circulation Research, 1998, 82, 898-907.	4.5	178
27	Glucose Transporters in Cardiac Metabolism and Hypertrophy. , 2015, 6, 331-351.		174
28	Increased Glucose Uptake and Oxidation in Mouse Hearts Prevent High Fatty Acid Oxidation but Cause Cardiac Dysfunction in Diet-Induced Obesity. Circulation, 2009, 119, 2818-2828.	1.6	168
29	Mitochondrial protein interactome elucidated by chemical cross-linking mass spectrometry. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, 1732-1737.	7.1	165
30	CaMKII induces permeability transition through Drp1 phosphorylation during chronic Î²-AR stimulation. Nature Communications, 2016, 7, 13189.	12.8	151
31	Decreased Contractile and Metabolic Reserve in Peroxisome Proliferatorâ€"Activated Receptor-Î±â€"Null Hearts Can Be Rescued by Increasing Glucose Transport and Utilization. Circulation, 2005, 112, 2339-2346.	1.6	148
32	Metabolic Remodeling Promotes Cardiac Hypertrophy by Directing Glucose to Aspartate Biosynthesis. Circulation Research, 2020, 126, 182-196.	4.5	135
33	Deletion of thioredoxin-interacting protein in mice impairs mitochondrial function but protects the myocardium from ischemia-reperfusion injury. Journal of Clinical Investigation, 2012, 122, 267-279.	8.2	135
34	AMP-Activated Protein Kinase Deficiency Enhances Myocardial Ischemia/Reperfusion Injury but Has Minimal Effect on the Antioxidant/Antinitrative Protection of Adiponectin. Circulation, 2009, 119, 835-844.	1.6	128
35	Broad Suppression of NADPH Oxidase Activity Exacerbates Ischemia/Reperfusion Injury Through Inadvertent Downregulation of Hypoxia-inducible Factor-1Î± and Upregulation of Peroxisome Proliferatorâ€"activated Receptor-Î±. Circulation Research, 2013, 112, 1135-1149.	4.5	127
36	Global Proteomics and Pathway Analysis of Pressure-Overloadâ€"Induced Heart Failure and Its Attenuation by Mitochondrial-Targeted Peptides. Circulation: Heart Failure, 2013, 6, 1067-1076.	3.9	126

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37	AMP-activated protein kinase mediates preconditioning in cardiomyocytes by regulating activity and trafficking of sarcolemmal ATP-sensitive K+ channels. Journal of Cellular Physiology, 2007, 210, 224-236.	4.1	122
38	Chemical Crosslinking Mass Spectrometry Analysis of Protein Conformations and Supercomplexes in Heart Tissue. Cell Systems, 2018, 6, 136-141.e5.	6.2	118
39	Boosting NAD level suppresses inflammatory activation of PBMCs in heart failure. Journal of Clinical Investigation, 2020, 130, 6054-6063.	8.2	117
40	Glucose promotes cell growth by suppressing branched-chain amino acid degradation. Nature Communications, 2018, 9, 2935.	12.8	115
41	Long-Term Effects of Increased Glucose Entry on Mouse Hearts During Normal Aging and Ischemic Stress. Circulation, 2007, 116, 901-909.	1.6	112
42	A novel approach to measure mitochondrial respiration in frozen biological samples. EMBO Journal, 2020, 39, e104073.	7.8	110
43	Novel targets for mitochondrial medicine. Science Translational Medicine, 2016, 8, 326rv3.	12.4	106
44	Failure to Maintain a Low ADP Concentration Impairs Diastolic Function in Hypertrophied Rat Hearts. Circulation, 1997, 96, 1313-1319.	1.6	106
45	Rapamycin transiently induces mitochondrial remodeling to reprogram energy metabolism in old hearts. Aging, 2016, 8, 314-327.	3.1	104
46	Increasing Fatty Acid Oxidation Prevents High-Fat Dietâ€"Induced Cardiomyopathy Through Regulating Parkin-Mediated Mitophagy. Circulation, 2020, 142, 983-997.	1.6	103
47	Depletion of Energy Reserve via the Creatine Kinase Reaction During the Evolution of Heart Failure in Cardiomyopathic Hamsters. Journal of Molecular and Cellular Cardiology, 1996, 28, 755-765.	1.9	95
48	Aberrant activation of AMP-activated protein kinase remodels metabolic network in favor of cardiac glycogen storage. Journal of Clinical Investigation, 2007, 117, 1432-1439.	8.2	95
49	Impaired Mitochondrial Biogenesis Precedes Heart Failure in Right Ventricular Hypertrophy in Congenital Heart Disease. Circulation: Heart Failure, 2011, 4, 707-713.	3.9	94
50	Unlocking the Secrets of Mitochondria in the Cardiovascular System. Circulation, 2019, 140, 1205-1216.	1.6	91
51	Regulation of mitochondrial functions by protein phosphorylation and dephosphorylation. Cell and Bioscience, 2016, 6, 25.	4.8	85
52	Increased Î±2 Subunitâ€"Associated AMPK Activity and PRKAG2 Cardiomyopathy. Circulation, 2005, 112, 3140-3148.	1.6	83
53	Targeting NAD+ Metabolism as Interventions for Mitochondrial Disease. Scientific Reports, 2019, 9, 3073.	3.3	82
54	Substrain specific response to cardiac pressure overload in C57BL/6 mice. American Journal of Physiology - Heart and Circulatory Physiology, 2013, 305, H397-H402.	3.2	74

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55	Targeting AMPK for cardiac protection: Opportunities and challenges. Journal of Molecular and Cellular Cardiology, 2011, 51, 548-553.	1.9	72
56	Proposed Regulation of Gene Expression by Glucose in Rodent Heart. Gene Regulation and Systems Biology, 2007, 1, GRSB.S222.	2.3	65
57	Mitochondrial Maturation in Human Pluripotent Stem Cell Derived Cardiomyocytes. Stem Cells International, 2017, 2017, 1-10.	2.5	65
58	Role of Extracellular and Intracellular Acidosis for Hypercapnia-Induced Inhibition of Tension of Isolated Rat Cerebral Arteries. Circulation Research, 1995, 76, 269-275.	4.5	64
59	Mutation in the Î³2-Subunit of AMP-Activated Protein Kinase Stimulates Cardiomyocyte Proliferation and Hypertrophy Independent of Glycogen Storage. Circulation Research, 2014, 114, 966-975.	4.5	63
60	Elimination of NADPH Oxidase Activity Promotes Reductive Stress and Sensitizes the Heart to Ischemic Injury. Journal of the American Heart Association, 2014, 3, e000555.	3.7	62
61	Preservation of myocardial fatty acid oxidation prevents diastolic dysfunction in mice subjected to angiotensin II infusion. Journal of Molecular and Cellular Cardiology, 2016, 100, 64-71.	1.9	61
62	Functional role of AMP-activated protein kinase in the heart during exercise. FEBS Letters, 2005, 579, 2045-2050.	2.8	60
63	AMPK isoform expression in the normal and failing hearts. Journal of Molecular and Cellular Cardiology, 2012, 52, 1066-1073.	1.9	59
64	Ketones Step to the Plate. Circulation, 2016, 133, 689-691.	1.6	59
65	Rearrangement of energetic and substrate utilization networks compensate for chronic myocardial creatine kinase deficiency. Journal of Physiology, 2011, 589, 5193-5211.	2.9	53
66	Simultaneous Analysis of Major Coenzymes of Cellular Redox Reactions and Energy Using ex Vivo 1H NMR Spectroscopy. Analytical Chemistry, 2016, 88, 4817-4824.	6.5	53
67	Effects of endothelin-1 in the isolated heart in ischemia/reperfusion and hypoxia/reoxygenation injury. Journal of Molecular and Cellular Cardiology, 1991, 23, 1397-1409.	1.9	49
68	Effects of chronic Akt activation on glucose uptake in the heart. American Journal of Physiology - Endocrinology and Metabolism, 2006, 290, E789-E797.	3.5	49
69	Effects of Insulin Replacements, Inhibitors of Angiotensin, and PKCÂ's Actions to Normalize Cardiac Gene Expression and Fuel Metabolism in Diabetic Rats. Diabetes, 2007, 56, 1410-1420.	0.6	49
70	Activation of Î³2-AMPK Suppresses Ribosome Biogenesis and Protects Against Myocardial Ischemia/Reperfusion Injury. Circulation Research, 2017, 121, 1182-1191.	4.5	49
71	Cellular Interactome Dynamics during Paclitaxel Treatment. Cell Reports, 2019, 29, 2371-2383.e5.	6.4	45
72	Cardioprotective effect of adiponectin is partially mediated by its AMPK-independent antinitrative action. American Journal of Physiology - Endocrinology and Metabolism, 2009, 297, E384-E391.	3.5	44

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73	Altered Creatine Kinase Enzyme Kinetics in Diabetic Cardiomyopathy. A31P NMR Magnetization Transfer Study of the Intact Beating Rat Heart. Journal of Molecular and Cellular Cardiology, 1999, 31, 2175-2189.	1.9	43
74	N488I Mutation of the Î³2-Subunit Results in Bidirectional Changes in AMP-Activated Protein Kinase Activity. Circulation Research, 2005, 97, 323-328.	4.5	43
75	Revealing Pathway Dynamics in Heart Diseases by Analyzing Multiple Differential Networks. PLoS Computational Biology, 2015, 11, e1004332.	3.2	43
76	Endogenous nitric oxide enhances coupling between O2 consumption and ATP synthesis in guinea pig hearts. American Journal of Physiology - Heart and Circulatory Physiology, 2001, 281, H838-H846.	3.2	42
77	Raising NAD in Heart Failure. Circulation, 2018, 137, 2274-2277.	1.6	42
78	Pharmacologic therapy for engraftment arrhythmia induced by transplantation of human cardiomyocytes. Stem Cell Reports, 2021, 16, 2473-2487.	4.8	42
79	Transgenic overexpression of ribonucleotide reductase improves cardiac performance. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 6187-6192.	7.1	40
80	Mitochondrion as a Target for Heart Failure Therapyã€€â€“ Role of Protein Lysine Acetylation â€“. Circulation Journal, 2015, 79, 1863-1870.	1.6	37
81	Long-term beta-blocker treatment prevents chronic creatine kinase and lactate dehydrogenase system changes in rat hearts after myocardial infarction. Journal of the American College of Cardiology, 1996, 27, 487-493.	2.8	36
82	Transcriptional regulation of energy substrate metabolism in normal and hypertrophied heart. Current Hypertension Reports, 2003, 5, 454-458.	3.5	36
83	A PRKAG2 mutation causes biphasic changes in myocardial AMPK activity and does not protect against ischemia. Biochemical and Biophysical Research Communications, 2007, 360, 381-387.	2.1	36
84	Thermodynamic limitation for Ca2+ handling contributes to decreased contractile reserve in rat hearts. American Journal of Physiology - Heart and Circulatory Physiology, 1998, 275, H2064-H2071.	3.2	35
85	Metabolic Therapy at the Crossroad: How to Optimize Myocardial Substrate Utilization?. Trends in Cardiovascular Medicine, 2009, 19, 201-207.	4.9	35
86	Compromised Myocardial Energetics in Hypertrophied Mouse Hearts Diminish the Beneficial Effect of Overexpressing SERCA2a. Journal of Biological Chemistry, 2011, 286, 10163-10168.	3.4	35
87	Heart specific knockout of Ndufs4 ameliorates ischemia reperfusion injury. Journal of Molecular and Cellular Cardiology, 2018, 123, 38-45.	1.9	35
88	Promoting PGC-1Î±-driven mitochondrial biogenesis is detrimental in pressure-overloaded mouse hearts. American Journal of Physiology - Heart and Circulatory Physiology, 2014, 307, H1307-H1316.	3.2	34
89	Elevated MCU Expression by CaMKIIÎ´B Limits Pathological Cardiac Remodeling. Circulation, 2022, 145, 1067-1083.	1.6	34
90	NAD ⁺ Redox Imbalance in the Heart Exacerbates Diabetic Cardiomyopathy. Circulation: Heart Failure, 2021, 14, e008170.	3.9	33

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91	Suppression of 5â€²-AMP-activated protein kinase activity does not impair recovery of contractile function during reperfusion of ischemic hearts. American Journal of Physiology - Heart and Circulatory Physiology, 2009, 297, H313-H321.	3.2	32
92	The Role of Diacylglycerol Acyltransferase (DGAT) 1 and 2 in Cardiac Metabolism and Function. Scientific Reports, 2018, 8, 4983.	3.3	32
93	Another Role for the Celebrity. Circulation Research, 2005, 96, 139-140.	4.5	31
94	Enhancing Cardiac Triacylglycerol Metabolism Improves Recovery From Ischemic Stress. Diabetes, 2015, 64, 2817-2827.	0.6	30
95	Regional Biochemical Remodeling in Non-infarcted Tissue of Rat Heart Post-myocardial Infarction. Journal of Molecular and Cellular Cardiology, 1996, 28, 1531-1538.	1.9	29
96	TNF-Î± induces acyl-CoA synthetase 3 to promote lipid droplet formation in human endothelial cells. Journal of Lipid Research, 2020, 61, 33-44.	4.2	29
97	Boosting NAD+ blunts TLR4-induced type I IFN in control and systemic lupus erythematosus monocytes. Journal of Clinical Investigation, 2022, 132, .	8.2	27
98	Long-term expression of protein kinase C in adult mouse hearts improves postischemic recovery. Proceedings of the National Academy of Sciences of the United States of America, 1999, 96, 13536-13541.	7.1	26
99	GLUT1 overexpression enhances glucose metabolism and promotes neonatal heart regeneration. Scientific Reports, 2021, 11, 8669.	3.3	25
100	Assessment of Cardiac Function and Energetics in Isolated Mouse Hearts Using ³¹P NMR Spectroscopy. Journal of Visualized Experiments, 2010, , .	0.3	24
101	Extending the Scope of ¹H NMR Spectroscopy for the Analysis of Cellular Coenzyme A and Acetyl Coenzyme A. Analytical Chemistry, 2019, 91, 2464-2471.	6.5	22
102	Alterations of Performance and Oxygen Utilization in Chronically Infarcted Rat Hearts. Journal of Molecular and Cellular Cardiology, 1996, 28, 321-330.	1.9	21
103	Mechanisms behind the Relaxing Effect of Furosemide on the Isolated Rabbit Ear Artery. Basic and Clinical Pharmacology and Toxicology, 1990, 67, 406-410.	0.0	20
104	The Molecular Energetics of the Failing Heart from Animal Modelsâ€•Small Animal Models. , 1999, 4, 245-253.		20
105	The effects of fatty acid composition on cardiac hypertrophy and function in mouse models of diet-induced obesity. Journal of Nutritional Biochemistry, 2017, 46, 137-142.	4.2	20
106	NAD(H) in mitochondrial energy transduction: implications for health and disease. Current Opinion in Physiology, 2018, 3, 101-109.	1.8	20
107	Increasing fatty acid oxidation elicits a sex-dependent response in failing mouse hearts. Journal of Molecular and Cellular Cardiology, 2021, 158, 1-10.	1.9	19
108	Sample preparation methodology for mouse heart metabolomics using comprehensive two-dimensional gas chromatography coupled with time-of-flight mass spectrometry. Talanta, 2013, 108, 123-130.	5.5	18

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109	Rescue of heart lipoprotein lipase-knockout mice confirms a role for triglyceride in optimal heart metabolism and function. American Journal of Physiology - Endocrinology and Metabolism, 2013, 305, E1339-E1347.	3.5	17
110	Upregulation of mitochondrial ATPase inhibitory factor 1 (ATPIF1) mediates increased glycolysis in mouse hearts. Journal of Clinical Investigation, 2022, 132, .	8.2	17
111	Lack of UCP3 does not affect skeletal muscle mitochondrial function under lipid-challenged conditions, but leads to sudden cardiac death. Basic Research in Cardiology, 2014, 109, 447.	5.9	16
112	Amino acid primed mTOR activity is essential for heart regeneration. IScience, 2022, 25, 103574.	4.1	15
113	Genetically encoded biosensors for evaluating NAD+/NADH ratio in cytosolic and mitochondrial compartments. Cell Reports Methods, 2021, 1, 100116.	2.9	14
114	Diabetes Suppresses Glucose Uptake and Glycolysis in Macrophages. Circulation Research, 2022, 130, 779-781.	4.5	13
115	Acetylation of muscle creatine kinase negatively impacts high-energy phosphotransfer in heart failure. JCI Insight, 2021, 6, .	5.0	12
116	How Does Folic Acid Cure Heart Attacks?. Circulation, 2008, 117, 1772-1774.	1.6	11
117	Metabolic Modulation of Macrophage Function Post Myocardial Infarction. Frontiers in Physiology, 2020, 11, 674.	2.8	11
118	Enhancing fatty acid oxidation negatively regulates PPARs signaling in the heart. Journal of Molecular and Cellular Cardiology, 2020, 146, 1-11.	1.9	10
119	A high-carbohydrate diet lowered blood pressure in healthy Chinese male adolescents. BioScience Trends, 2014, 8, 132-137.	3.4	9
120	The Relationship Between KLF5 and PPARÎ± in the Heart. Circulation Research, 2016, 118, 193-195.	4.5	9
121	Angiotensin I conversion and coronary constriction by angiotensin II in ischemic and hypoxic isolated rat hearts. European Journal of Pharmacology, 1991, 203, 71-77.	3.5	7
122	Thermodynamic Limitation for the Sarcoplasmic Reticulum Ca2+-ATPase Contributes to Impaired Contractile Reserve in Heartsa. Annals of the New York Academy of Sciences, 1998, 853, 322-324.	3.8	5
123	Combat Doxorubicin Cardiotoxicity With the Power of Mitochondria Transfer. JACC: CardioOncology, 2021, 3, 441-443.	4.0	5
124	Energetic basis of diastolic dysfunction. Magnetic Resonance Materials in Physics, Biology, and Medicine, 1998, 6, 129-131.	2.0	4
125	Boosting mitochondrial metabolism with dietary supplements in heart failure. Nature Reviews Cardiology, 2021, 18, 685-686.	13.7	4
126	Metabolism and Inflammation in Cardiovascular Health and Diseases: Mechanisms to Therapies. Journal of Molecular and Cellular Cardiology, 2021, 157, 113-114.	1.9	3

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127	Myocardial contractile efficiency increases in proportion to a fetal enzyme shift in chronically infarcted rat hearts. Basic Research in Cardiology, 2005, 100, 171-178.	5.9	2
128	Failed Power Plant Turns Into Mass Murder. Circulation Research, 2018, 122, 11-13.	4.5	2
129	Remodeling of cardiac metabolism in heart failure with preserved ejection fraction. Current Opinion in Physiology, 2022, 27, 100559.	1.8	2
130	Transcript variant dictates Prkag2 cardiomyopathy?. Journal of Molecular and Cellular Cardiology, 2012, 53, 317-319.	1.9	1
131	Meeting highlights from the 2013 <scp>E</scp>uropean <scp>S</scp>ociety of <scp>C</scp>ardiology <scp>H</scp>eart <scp>F</scp>ailure <scp>A</scp>ssociation <scp>W</scp>inter <scp>M</scp>eeting on <scp>T</scp>ranslational <scp>H</scp>eart <scp>F</scp>ailure <scp>R</scp>esearch. European Journal of Heart Failure, 2014, 16, 6-14.	7.1	1
132	Glucose Promotes Cell Growth by Suppressing Branched-chain Amino Acid Degradation. Journal of Molecular and Cellular Cardiology, 2017, 112, 156.	1.9	1
133	Abstract 282: Increasing Cardiac Fatty Acid Oxidation Protects Against High Fat Diet Induced Mitochondria Dysfunction and Cardiomyopathy in Mice. Circulation Research, 2018, 123, .	4.5	1
134	Energetic basis of diastolic dysfunction. Magnetic Resonance Materials in Physics, Biology, and Medicine, 1998, 6, 129-131.	2.0	0
135	Response to Letter Regarding Article, â€œImpaired Mitochondrial Biogenesis Precedes Heart Failure in Right Ventricular Hypertrophy in Congenital Heart Diseaseâ€ Circulation: Heart Failure, 2012, 5, .	3.9	0
136	Increased Glucose Transport Prevents the Development of Contractile Dysfunctions in Diabetic Hearts. FASEB Journal, 2009, 23, 856.9.	0.5	0
137	Abstract 19826: Cardiac DGAT1 Deficiency Decreases Triglyceride Turnover and Alters Substrate Utilization. Circulation, 2014, 130, .	1.6	0
138	Metabolic Interventions to Treat Mitochondrial Cardiomyopathy: Roles of NAD + and Protein Acetylation in Leigh Syndrome. FASEB Journal, 2018, 32, 900.2.	0.5	0
139	Abstract 543: Uncovering the Mechanisms by Which Fatty Acid Oxidation Suppresses Cardiomyocyte Hypertrophy. Circulation Research, 2018, 123, .	4.5	0
140	Abstract 275: Quantification of the Mitochondrial Protein Interactome in Failing Hearts. Circulation Research, 2018, 123, .	4.5	0
141	Abstract 417: Targeting the NAD/NADH Ratio for Heart Failure Therapy. Circulation Research, 2018, 123, .	4.5	0
142	Abstract 413: NAD + -dependent Pathogenic Mechanisms and Metabolic Interventions for Mitochondrial Disease and its Associated Cardiomyopathy. Circulation Research, 2018, 123, .	4.5	0
143	Abstract 411: Mitochondrial NAD(H) Signaling in Cardiac Physiology and Pathology. Circulation Research, 2018, 123, .	4.5	0
144	1921-P: The Role of BCAA Catabolism in Glucose Homeostasis. Diabetes, 2019, 68, 1921-P.	0.6	0

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145	Metabolic Modulation of Cardiac Health: The Role of Glucose and Amino Acids. FASEB Journal, 2020, 34, 1-1.	0.5	0
146	Roles of NAD Metabolism in Diabetic Cardiomyopathy. FASEB Journal, 2020, 34, 1-1.	0.5	0
147	Abstract 15465: Upregulation of Mitochondrial Atpase Inhibitory Factor 1 Mediates Increased Glycolysis in Pathological Cardiac Hypertrophy. Circulation, 2020, 142, .	1.6	0