Jeffrey Robbins

List of Publications by Year
in descending order

96 papers	12,365 citations	44069 48 h-index	40979 93 g-index
98 all docs	98 docs citations	98 times ranked	20509 citing authors

#	Article	IF	Citations
1	A high-throughput screening identifies ZNF418 as a novel regulator of the ubiquitin-proteasome system and autophagy-lysosomal pathway. Autophagy, 2021, 17, 3124-3139.	9.1	12
2	The N terminus of myosin-binding protein C extends toward actin filaments in intact cardiac muscle. Journal of General Physiology, 2021, 153, .	1.9	30
3	Cardiac myosin binding protein-C phosphorylation accelerates Î²-cardiac myosin detachment rate in mouse myocardium. American Journal of Physiology - Heart and Circulatory Physiology, 2021, 320, H1822-H1835.	3.2	6
4	CYLD exaggerates pressure overload-induced cardiomyopathy via suppressing autolysosome efflux in cardiomyocytes. Journal of Molecular and Cellular Cardiology, 2020, 145, 59-73.	1.9	18
5	Ube2v1 Positively Regulates Protein Aggregation by Modulating Ubiquitin Proteasome System Performance Partially Through K63 Ubiquitination. Circulation Research, 2020, 126, 907-922.	4.5	22
6	In Vivo Remodeling of an Extracellular Matrix Cardiac Patch in an Ovine Model. ASAIO Journal, 2019, 65, 744-752.	1.6	7
7	Myofibroblast-Specific TGFÎ² Receptor II Signaling in the Fibrotic Response to Cardiac Myosin Binding Protein C-Induced Cardiomyopathy. Circulation Research, 2018, 123, 1285-1297.	4.5	39
8	Hypertrophic cardiomyopathy R403Q mutation in rabbit Î²-myosin reduces contractile function at the molecular and myofibrillar levels. Proceedings of the National Academy of Sciences of the United States of America, 2018, 115, 11238-11243.	7.1	23
9	Cardiac Dysfunction in the Sigma 1 Receptor Knockout Mouse Associated With Impaired Mitochondrial Dynamics and Bioenergetics. Journal of the American Heart Association, 2018, 7, e009775.	3.7	54
10	Cardiac Fibrosis in Proteotoxic Cardiac Disease is Dependent Upon Myofibroblast TGFâ€Î² Signaling. Journal of the American Heart Association, 2018, 7, e010013.	3.7	37
11	Desmin and Cardiac Disease. Circulation Research, 2018, 122, 1324-1326.	4.5	18
12	Aberrant Mitochondrial Fission Is Maladaptive in Desmin Mutationâ€“Induced Cardiac Proteotoxicity. Journal of the American Heart Association, 2018, 7, .	3.7	29
13	Activation of Autophagy Ameliorates Cardiomyopathy in <i>Mybpc3</i> -Targeted Knockin Mice. Circulation: Heart Failure, 2017, 10, .	3.9	53
14	Inhibition of Mutant Î±B Crystallinâ€Induced Protein Aggregation by a Molecular Tweezer. Journal of the American Heart Association, 2017, 6, .	3.7	15
15	MMIâ€0100 Inhibits Cardiac Fibrosis in a Mouse Model Overexpressing Cardiac Myosin Binding Protein C. Journal of the American Heart Association, 2017, 6, .	3.7	13
16	Increased susceptibility to structural acute kidney injury in a mouse model of presymptomatic cardiomyopathy. American Journal of Physiology - Renal Physiology, 2017, 313, F699-F705.	2.7	3
17	An Unbiased High-Throughput Screen to Identify Novel Effectors That Impact on Cardiomyocyte Aggregate Levels. Circulation Research, 2017, 121, 604-616.	4.5	13
18	Healing a Heart Through Genetic Intervention. Circulation Research, 2016, 118, 920-922.	4.5	3

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19	Alterations in Multiâ€Scale Cardiac Architecture in Association With Phosphorylation of Myosin Binding Proteinâ€C. Journal of the American Heart Association, 2016, 5, e002836.	3.7	17
20	UBC9-Mediated Sumoylation Favorably Impacts Cardiac Function in Compromised Hearts. Circulation Research, 2016, 118, 1894-1905.	4.5	57
21	Making the connections: Autophagy and post-translational modifications in cardiomyocytes. Autophagy, 2016, 12, 2252-2253.	9.1	16
22	In vivo definition of cardiac myosin-binding protein Câ€™s critical interactions with myosin. Pflugers Archiv European Journal of Physiology, 2016, 468, 1685-1695.	2.8	20
23	Guidelines for the use and interpretation of assays for monitoring autophagy (3rd edition). Autophagy, 2016, 12, 1-222.	9.1	4,701
24	Cardiac Fibrosis. Circulation Research, 2016, 118, 1021-1040.	4.5	1,136
25	Phosphorylation and calcium antagonistically tune myosin-binding protein Câ€™s structure and function. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, 3239-3244.	7.1	84
26	Functional significance of the discordance between transcriptional profile and left ventricular structure/function during reverse remodeling. JCI Insight, 2016, 1, e86038.	5.0	33
27	Proteotoxicity and Cardiac Dysfunction. Circulation Research, 2015, 116, 1863-1882.	4.5	80
28	Bringing It All Together. Circulation Research, 2015, 117, 987-989.	4.5	0
29	Myosin-binding protein C corrects an intrinsic inhomogeneity in cardiac excitation-contraction coupling. Science Advances, 2015, 1, .	10.3	69
30	Giving credence to controls: Avoiding the false phenotype. Journal of Molecular and Cellular Cardiology, 2015, 86, 136-137.	1.9	3
31	The absence of MuRF1 protects against Calpain1â€induced systolic dysfunction in vivo. FASEB Journal, 2015, 29, 46.1.	0.5	0
32	Desensitization of Myofilaments to Ca ²⁺ as a Therapeutic Target for Hypertrophic Cardiomyopathy With Mutations in Thin Filament Proteins. Circulation: Cardiovascular Genetics, 2014, 7, 132-143.	5.1	61
33	Tubulin hyperacetylation is adaptive in cardiac proteotoxicity by promoting autophagy. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, E5178-86.	7.1	92
34	Disturbance in Z-Disk Mechanosensitive Proteins Induced by a Persistent Mutant Myopalladin Causes Familial Restrictive Cardiomyopathy. Journal of the American College of Cardiology, 2014, 64, 2765-2776.	2.8	39
35	Post-translational control of cardiac hemodynamics through myosin binding protein C. Pflugers Archiv European Journal of Physiology, 2014, 466, 231-236.	2.8	24
36	RAF1 mutations in childhood-onset dilated cardiomyopathy. Nature Genetics, 2014, 46, 635-639.	21.4	69

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37	Proteotoxicity: An underappreciated pathology in cardiac disease. Journal of Molecular and Cellular Cardiology, 2014, 71, 3-10.	1.9	55
38	Sumo E2 Enzyme UBC9 Is Required for Efficient Protein Quality Control in Cardiomyocytes. Circulation Research, 2014, 115, 721-729.	4.5	59
39	Functional dissection of myosin binding protein C phosphorylation. Journal of Molecular and Cellular Cardiology, 2013, 64, 39-50.	1.9	19
40	An Endogenously Produced Fragment of Cardiac Myosin-Binding Protein C Is Pathogenic and Can Lead to Heart Failure. Circulation Research, 2013, 113, 553-561.	4.5	26
41	Enhanced autophagy ameliorates cardiac proteinopathy. Journal of Clinical Investigation, 2013, 123, 5284-5297.	8.2	260
42	Determination of the critical residues responsible for cardiac myosin binding protein C's interactions. Journal of Molecular and Cellular Cardiology, 2012, 53, 838-847.	1.9	42
43	Unique single molecule binding of cardiac myosin binding protein-C to actin and phosphorylation-dependent inhibition of actomyosin motility requires 17 amino acids of the motif domain. Journal of Molecular and Cellular Cardiology, 2012, 52, 219-227.	1.9	77
44	The extent of cardiac myosin binding protein-C phosphorylation modulates actomyosin function in a graded manner. Journal of Muscle Research and Cell Motility, 2012, 33, 449-459.	2.0	34
45	Electron Microscopy and 3D Reconstruction of F-Actin Decorated with Cardiac Myosin-Binding Protein C (cMyBP-C). Journal of Molecular Biology, 2011, 410, 214-225.	4.2	67
46	Autophagy and proteotoxicity in cardiomyocytes. Autophagy, 2011, 7, 1259-1260.	9.1	20
47	Atg7 Induces Basal Autophagy and Rescues Autophagic Deficiency in CryAB ^{R120G} Cardiomyocytes. Circulation Research, 2011, 109, 151-160.	4.5	162
48	A Critical Function for Ser-282 in Cardiac Myosin Binding Protein-C Phosphorylation and Cardiac Function. Circulation Research, 2011, 109, 141-150.	4.5	113
49	Signaling and Myosin-binding Protein C. Journal of Biological Chemistry, 2011, 286, 9913-9919.	3.4	36
50	Twenty Years of Gene Targeting. Circulation Research, 2011, 109, 722-723.	4.5	6
51	Desmin-related cardiomyopathy: an unfolding story. American Journal of Physiology - Heart and Circulatory Physiology, 2011, 301, H1220-H1228.	3.2	118
52	Enhancement of proteasomal function protects against cardiac proteinopathy and ischemia/reperfusion injury in mice. Journal of Clinical Investigation, 2011, 121, 3689-3700.	8.2	169
53	Manipulation of Death Pathways in Desmin-Related Cardiomyopathy. Circulation Research, 2010, 106, 1524-1532.	4.5	60
54	Distinct Sarcomeric Substrates Are Responsible for Protein Kinase D-mediated Regulation of Cardiac Myofilament Ca2+ Sensitivity and Cross-bridge Cycling. Journal of Biological Chemistry, 2010, 285, 5674-5682.	3.4	96

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55	Autophagy in desmin-related cardiomyopathy: Thoughts at the halfway point. Autophagy, 2010, 6, 665-666.	9.1	15
56	Cardiac Myosin Binding Protein-C Phosphorylation in a Î²-Myosin Heavy Chain Background. Circulation, 2009, 119, 1253-1262.	1.6	81
57	Inducible Expression of Active Protein Phosphatase-1 Inhibitor-1 Enhances Basal Cardiac Function and Protects Against Ischemia/Reperfusion Injury. Circulation Research, 2009, 104, 1012-1020.	4.5	95
58	Phospholamban overexpression in rabbit ventricular myocytes does not alter sarcoplasmic reticulum Ca transport. American Journal of Physiology - Heart and Circulatory Physiology, 2009, 296, H698-H703.	3.2	9
59	Biochemical and Mechanical Dysfunction in a Mouse Model of Desmin-Related Myopathy. Circulation Research, 2009, 104, 1021-1028.	4.5	48
60	With great power comes great responsibility: Using mouse genetics to study cardiac hypertrophy and failure. Journal of Molecular and Cellular Cardiology, 2009, 46, 130-136.	1.9	96
61	Phospholamban overexpression in transgenic rabbits. Transgenic Research, 2008, 17, 157-170.	2.4	30
62	Cardiomyocyte Expression of a Polyglutamine Preamyloid Oligomer Causes Heart Failure. Circulation, 2008, 117, 2743-2751.	1.6	126
63	Role of the acidic Nâ€² region of cardiac troponin I in regulating myocardial function. FASEB Journal, 2008, 22, 1246-1257.	0.5	23
64	Inducible expression of active Inhibitorâ€1 enhances cardiac function and improves contractility after an ischemic insult. FASEB Journal, 2008, 22, 970.15.	0.5	0
65	Overexpressed Cardiac Gs alpha Protects Against Myocardial Ischemic Injury in Conscious Rabbits. FASEB Journal, 2008, 22, 51-51.	0.5	1
66	Distribution and Structure-Function Relationship of Myosin Heavy Chain Isoforms in the Adult Mouse Heart. Journal of Biological Chemistry, 2007, 282, 24057-24064.	3.4	34
67	Exercise reverses preamyloid oligomer and prolongs survival in ÂB-crystallin-based desmin-related cardiomyopathy. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 5995-6000.	7.1	76
68	Control of In Vivo Contraction/Relaxation Kinetics by Myosin Binding Protein C. Circulation, 2007, 116, 2399-2408.	1.6	73
69	PKC-Î²II sensitizes cardiac myofilaments to Ca2+ by phosphorylating troponin I on threonine-144. Journal of Molecular and Cellular Cardiology, 2006, 41, 823-833.	1.9	84
70	Heart Failure and Protein Quality Control. Circulation Research, 2006, 99, 1315-1328.	4.5	205
71	Cardiac myosin binding protein c phosphorylation is cardioprotective. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 16918-16923.	7.1	189
72	Transgenic Rabbit Model for Human Troponin Iâ€"Based Hypertrophic Cardiomyopathy. Circulation, 2005, 111, 2330-2338.	1.6	72

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73	Forced Expression of Î±-Myosin Heavy Chain in the Rabbit Ventricle Results in Cardioprotection Under Cardiomyopathic Conditions. Circulation, 2005, 111, 2339-2346.	1.6	73
74	Reversal of amyloid-induced heart disease in desmin-related cardiomyopathy. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 13592-13597.	7.1	100
75	Mitochondrial Dysfunction and Apoptosis Underlie the Pathogenic Process in Î±-B-Crystallin Desmin-Related Cardiomyopathy. Circulation, 2005, 112, 3451-3461.	1.6	174
76	Cardiac Myosin-Binding Protein-C Phosphorylation and Cardiac Function. Circulation Research, 2005, 97, 1156-1163.	4.5	203
77	In Vivo and in Vitro Analysis of Cardiac Troponin I Phosphorylation. Journal of Biological Chemistry, 2005, 280, 703-714.	3.4	84
78	Desmin-related cardiomyopathy in transgenic mice: A cardiac amyloidosis. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 10132-10136.	7.1	262
79	Overexpression of phospholamban in slowâ€twitch skeletal muscle is associated with depressed contractile function and muscle remodeling. FASEB Journal, 2004, 18, 974-976.	0.5	15
80	Impact of beta-myosin heavy chain expression on cardiac function during stress. Journal of the American College of Cardiology, 2004, 44, 2390-2397.	2.8	215
81	Genetic modification of the heart: exploring necessity and sufficiency in the past 10Byears. Journal of Molecular and Cellular Cardiology, 2004, 36, 643-652.	1.9	22
82	Reengineering Inducible Cardiac-Specific Transgenesis With an Attenuated Myosin Heavy Chain Promoter. Circulation Research, 2003, 92, 609-616.	4.5	227
83	Î±B-Crystallin Modulates Protein Aggregation of Abnormal Desmin. Circulation Research, 2003, 93, 998-1005.	4.5	114
84	Ischemic Protection and Myofibrillar Cardiomyopathy. Circulation Research, 2002, 91, 741-748.	4.5	78
85	Desmin filaments and cardiac disease: Establishing causality. Journal of Cardiac Failure, 2002, 8, S287-S292.	1.7	44
86	Molecular mechanics of mouse cardiac myosin isoforms. American Journal of Physiology - Heart and Circulatory Physiology, 2002, 283, H1446-H1454.	3.2	97
87	Expression of R120Gâ€"Î±B-Crystallin Causes Aberrant Desmin and Î±B-Crystallin Aggregation and Cardiomyopathy in Mice. Circulation Research, 2001, 89, 84-91.	4.5	282
88	Mouse Model of Desmin-Related Cardiomyopathy. Circulation, 2001, 103, 2402-2407.	1.6	184
89	Remodeling the Cardiac Sarcomere Using Transgenesis. Annual Review of Physiology, 2000, 62, 261-287.	13.1	42
90	Transgenic Modeling of a Cardiac Troponin I Mutation Linked to Familial Hypertrophic Cardiomyopathy. Circulation Research, 2000, 87, 805-811.	4.5	141

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91	Cardiotrophic Effects of Protein Kinase C Îµ. Circulation Research, 2000, 86, 1173-1179.	4.5	199
92	In Vivo Modeling of Myosin Binding Protein C Familial Hypertrophic Cardiomyopathy. Circulation Research, 1999, 85, 841-847.	4.5	93
93	Transgenic over-expression of a motor protein at high levels results in severe cardiac pathology. Transgenic Research, 1999, 8, 9-22.	2.4	35
94	Anin vivo analysis of transcriptional elements in the mouse Î±-myosin heavy chain gene promoter. Transgenic Research, 1995, 4, 397-405.	2.4	40
95	Position independent expression and developmental regulation is directed by the Î² myosin heavy chain gene's 5â€² upstream region in transgenic mice. Nucleic Acids Research, 1995, 23, 3301-3309.	14.5	40
96	In Vivo Definition of a Cardiac Specific Promoter and Its Potential Utility in Remodeling the Heart. Annals of the New York Academy of Sciences, 1995, 752, 492-505.	3.8	24