

Jeffrey Robbins

List of Publications by Year in Descending Order

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The third column is the impact factor (IF) of the journal, and the fourth column is the number of citations of the article.

95 papers	10,086 citations	43 h-index	98 g-index
98 ext. papers	11,468 ext. citations	10 avg, IF	5.69 L-index

#	Paper	IF	Citations
95	A high-throughput screening identifies ZNF418 as a novel regulator of the ubiquitin-proteasome system and autophagy-lysosomal pathway. <i>Autophagy</i> , 2021 , 17, 3124-3139	10.2	2
94	Cardiac myosin binding protein-C phosphorylation accelerates cardiac myosin detachment rate in mouse myocardium. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2021 , 320, H1822-H1835 ^O	5.7	1
93	The N terminus of myosin-binding protein C extends toward actin filaments in intact cardiac muscle. <i>Journal of General Physiology</i> , 2021 , 153,	3.4	4
92	CYLD exaggerates pressure overload-induced cardiomyopathy via suppressing autolysosome efflux in cardiomyocytes. <i>Journal of Molecular and Cellular Cardiology</i> , 2020 , 145, 59-73	5.8	7
91	Ube2v1 Positively Regulates Protein Aggregation by Modulating Ubiquitin Proteasome System Performance Partially Through K63 Ubiquitination. <i>Circulation Research</i> , 2020 , 126, 907-922	15.7	14
90	In Vivo Remodeling of an Extracellular Matrix Cardiac Patch in an Ovine Model. <i>ASAIO Journal</i> , 2019 , 65, 744-752	3.6	5
89	Aberrant Mitochondrial Fission Is Maladaptive in Desmin Mutation-Induced Cardiac Proteotoxicity. <i>Journal of the American Heart Association</i> , 2018 , 7,	6	19
88	Myofibroblast-Specific TGF β Receptor II Signaling in the Fibrotic Response to Cardiac Myosin Binding Protein C-Induced Cardiomyopathy. <i>Circulation Research</i> , 2018 , 123, 1285-1297	15.7	20
87	Hypertrophic cardiomyopathy R403Q mutation in rabbit myosin reduces contractile function at the molecular and myofibrillar levels. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2018 , 115, 11238-11243	11.5	16
86	Cardiac Dysfunction in the Sigma 1 Receptor Knockout Mouse Associated With Impaired Mitochondrial Dynamics and Bioenergetics. <i>Journal of the American Heart Association</i> , 2018 , 7, e009775	6	39
85	Cardiac Fibrosis in Proteotoxic Cardiac Disease is Dependent Upon Myofibroblast TGF β Signaling. <i>Journal of the American Heart Association</i> , 2018 , 7, e010013	6	25
84	Activation of Autophagy Ameliorates Cardiomyopathy in α -Targeted Knockin Mice. <i>Circulation: Heart Failure</i> , 2017 , 10,	7.6	37
83	Inhibition of Mutant β Crystallin-Induced Protein Aggregation by a Molecular Tweezer. <i>Journal of the American Heart Association</i> , 2017 , 6,	6	12
82	MMI-0100 Inhibits Cardiac Fibrosis in a Mouse Model Overexpressing Cardiac Myosin Binding Protein C. <i>Journal of the American Heart Association</i> , 2017 , 6,	6	11
81	Increased susceptibility to structural acute kidney injury in a mouse model of presymptomatic cardiomyopathy. <i>American Journal of Physiology - Renal Physiology</i> , 2017 , 313, F699-F705	4.3	3
80	An Unbiased High-Throughput Screen to Identify Novel Effectors That Impact on Cardiomyocyte Aggregate Levels. <i>Circulation Research</i> , 2017 , 121, 604-616	15.7	11
79	Making the connections: Autophagy and post-translational modifications in cardiomyocytes. <i>Autophagy</i> , 2016 , 12, 2252-2253	10.2	10

78	In vivo definition of cardiac myosin-binding protein C's critical interactions with myosin. <i>Pflugers Archiv European Journal of Physiology</i> , 2016 , 468, 1685-95	4.6	16
77	Guidelines for the use and interpretation of assays for monitoring autophagy (3rd edition). <i>Autophagy</i> , 2016 , 12, 1-222	10.2	3838
76	Cardiac Fibrosis: The Fibroblast Awakens. <i>Circulation Research</i> , 2016 , 118, 1021-40	15.7	734
75	Phosphorylation and calcium antagonistically tune myosin-binding protein C's structure and function. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2016 , 113, 3239-44	11.5	54
74	Functional significance of the discordance between transcriptional profile and left ventricular structure/function during reverse remodeling. <i>JCI Insight</i> , 2016 , 1, e86038	9.9	21
73	Healing a Heart Through Genetic Intervention. <i>Circulation Research</i> , 2016 , 118, 920-2	15.7	2
72	Alterations in Multi-Scale Cardiac Architecture in Association With Phosphorylation of Myosin Binding Protein-C. <i>Journal of the American Heart Association</i> , 2016 , 5, e002836	6	10
71	UBC9-Mediated Sumoylation Favorably Impacts Cardiac Function in Compromised Hearts. <i>Circulation Research</i> , 2016 , 118, 1894-905	15.7	37
70	Myosin-binding protein C corrects an intrinsic inhomogeneity in cardiac excitation-contraction coupling. <i>Science Advances</i> , 2015 , 1,	14.3	47
69	Proteotoxicity and cardiac dysfunction. <i>Circulation Research</i> , 2015 , 116, 1863-82	15.7	62
68	The absence of MuRF1 protects against Calpain1-induced systolic dysfunction in vivo. <i>FASEB Journal</i> , 2015 , 29, 46.1	0.9	
67	Post-translational control of cardiac hemodynamics through myosin binding protein C. <i>Pflugers Archiv European Journal of Physiology</i> , 2014 , 466, 231-6	4.6	21
66	RAF1 mutations in childhood-onset dilated cardiomyopathy. <i>Nature Genetics</i> , 2014 , 46, 635-639	36.3	54
65	Proteotoxicity: an underappreciated pathology in cardiac disease. <i>Journal of Molecular and Cellular Cardiology</i> , 2014 , 71, 3-10	5.8	41
64	Sumo E2 enzyme UBC9 is required for efficient protein quality control in cardiomyocytes. <i>Circulation Research</i> , 2014 , 115, 721-9	15.7	39
63	Desensitization of myofilaments to Ca ²⁺ as a therapeutic target for hypertrophic cardiomyopathy with mutations in thin filament proteins. <i>Circulation: Cardiovascular Genetics</i> , 2014 , 7, 132-143		46
62	Tubulin hyperacetylation is adaptive in cardiac proteotoxicity by promoting autophagy. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2014 , 111, E5178-86	11.5	73
61	Disturbance in Z-disk mechanosensitive proteins induced by a persistent mutant myopalladin causes familial restrictive cardiomyopathy. <i>Journal of the American College of Cardiology</i> , 2014 , 64, 2765-76	15.1	31

60	Functional dissection of myosin binding protein C phosphorylation. <i>Journal of Molecular and Cellular Cardiology</i> , 2013 , 64, 39-50	5.8	17
59	An endogenously produced fragment of cardiac myosin-binding protein C is pathogenic and can lead to heart failure. <i>Circulation Research</i> , 2013 , 113, 553-61	15.7	23
58	Enhanced autophagy ameliorates cardiac proteinopathy. <i>Journal of Clinical Investigation</i> , 2013 , 123, 5284-97	15.9	211
57	Author response: Bax and Bak function as the outer membrane component of the mitochondrial permeability pore in regulating necrotic cell death in mice 2013 ,		2
56	Determination of the critical residues responsible for cardiac myosin binding protein C β interactions. <i>Journal of Molecular and Cellular Cardiology</i> , 2012 , 53, 838-47	5.8	37
55	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. <i>Journal of Molecular and Cellular Cardiology</i> , 2012 , 52, 219-27	5.8	69
54	The extent of cardiac myosin binding protein-C phosphorylation modulates actomyosin function in a graded manner. <i>Journal of Muscle Research and Cell Motility</i> , 2012 , 33, 449-59	3.5	28
53	Electron microscopy and 3D reconstruction of F-actin decorated with cardiac myosin-binding protein C (cMyBP-C). <i>Journal of Molecular Biology</i> , 2011 , 410, 214-25	6.5	58
52	Autophagy and proteotoxicity in cardiomyocytes. <i>Autophagy</i> , 2011 , 7, 1259-60	10.2	17
51	Atg7 induces basal autophagy and rescues autophagic deficiency in CryABR120G cardiomyocytes. <i>Circulation Research</i> , 2011 , 109, 151-60	15.7	137
50	A critical function for Ser-282 in cardiac Myosin binding protein-C phosphorylation and cardiac function. <i>Circulation Research</i> , 2011 , 109, 141-50	15.7	95
49	Signaling and myosin-binding protein C. <i>Journal of Biological Chemistry</i> , 2011 , 286, 9913-9	5.4	35
48	Twenty years of gene targeting: what we don't know. <i>Circulation Research</i> , 2011 , 109, 722-3	15.7	6
47	Desmin-related cardiomyopathy: an unfolding story. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2011 , 301, H1220-8	5.2	93
46	Enhancement of proteasomal function protects against cardiac proteinopathy and ischemia/reperfusion injury in mice. <i>Journal of Clinical Investigation</i> , 2011 , 121, 3689-700	15.9	144
45	Manipulation of death pathways in desmin-related cardiomyopathy. <i>Circulation Research</i> , 2010 , 106, 1524-32	15.7	50
44	Distinct sarcomeric substrates are responsible for protein kinase D-mediated regulation of cardiac myofilament Ca ²⁺ sensitivity and cross-bridge cycling. <i>Journal of Biological Chemistry</i> , 2010 , 285, 5674-82	5.4	86
43	Autophagy in desmin-related cardiomyopathy: Thoughts at the halfway point. <i>Autophagy</i> , 2010 , 6, 665-666	6.2	14

42	Cardiac myosin binding protein-C phosphorylation in a {beta}-myosin heavy chain background. <i>Circulation</i> , 2009 , 119, 1253-62	16.7	69
41	Inducible expression of active protein phosphatase-1 inhibitor-1 enhances basal cardiac function and protects against ischemia/reperfusion injury. <i>Circulation Research</i> , 2009 , 104, 1012-20	15.7	91
40	Phospholamban overexpression in rabbit ventricular myocytes does not alter sarcoplasmic reticulum Ca transport. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2009 , 296, H698-703	5.2	9
39	Biochemical and mechanical dysfunction in a mouse model of desmin-related myopathy. <i>Circulation Research</i> , 2009 , 104, 1021-8	15.7	42
38	With great power comes great responsibility: using mouse genetics to study cardiac hypertrophy and failure. <i>Journal of Molecular and Cellular Cardiology</i> , 2009 , 46, 130-6	5.8	82
37	Cardiomyocyte expression of a polyglutamine preamyloid oligomer causes heart failure. <i>Circulation</i> , 2008 , 117, 2743-51	16.7	109
36	Role of the acidic NTregion of cardiac troponin I in regulating myocardial function. <i>FASEB Journal</i> , 2008 , 22, 1246-57	0.9	22
35	Phospholamban overexpression in transgenic rabbits. <i>Transgenic Research</i> , 2008 , 17, 157-70	3.3	30
34	Inducible expression of active Inhibitor-1 enhances cardiac function and improves contractility after an ischemic insult. <i>FASEB Journal</i> , 2008 , 22, 970.15	0.9	
33	Overexpressed Cardiac Gs alpha Protects Against Myocardial Ischemic Injury in Conscious Rabbits. <i>FASEB Journal</i> , 2008 , 22, 51-51	0.9	1
32	Distribution and structure-function relationship of myosin heavy chain isoforms in the adult mouse heart. <i>Journal of Biological Chemistry</i> , 2007 , 282, 24057-64	5.4	33
31	Exercise reverses preamyloid oligomer and prolongs survival in alphaB-crystallin-based desmin-related cardiomyopathy. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2007 , 104, 5995-6000	11.5	66
30	Control of in vivo left ventricular [correction] contraction/relaxation kinetics by myosin binding protein C: protein kinase A phosphorylation dependent and independent regulation. <i>Circulation</i> , 2007 , 116, 2399-408	16.7	66
29	Heart failure and protein quality control. <i>Circulation Research</i> , 2006 , 99, 1315-28	15.7	171
28	Cardiac myosin binding protein C phosphorylation is cardioprotective. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2006 , 103, 16918-23	11.5	164
27	PKC-beta11 sensitizes cardiac myofilaments to Ca ²⁺ by phosphorylating troponin I on threonine-144. <i>Journal of Molecular and Cellular Cardiology</i> , 2006 , 41, 823-33	5.8	79
26	Transgenic rabbit model for human troponin I-based hypertrophic cardiomyopathy. <i>Circulation</i> , 2005 , 111, 2330-8	16.7	60
25	Forced expression of alpha-myosin heavy chain in the rabbit ventricle results in cardioprotection under cardiomyopathic conditions. <i>Circulation</i> , 2005 , 111, 2339-46	16.7	65

24	Reversal of amyloid-induced heart disease in desmin-related cardiomyopathy. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2005 , 102, 13592-7	11.5	93
23	Mitochondrial dysfunction and apoptosis underlie the pathogenic process in alpha-B-crystallin desmin-related cardiomyopathy. <i>Circulation</i> , 2005 , 112, 3451-61	16.7	162
22	Cardiac myosin-binding protein-C phosphorylation and cardiac function. <i>Circulation Research</i> , 2005 , 97, 1156-63	15.7	182
21	In vivo and in vitro analysis of cardiac troponin I phosphorylation. <i>Journal of Biological Chemistry</i> , 2005 , 280, 703-14	5.4	72
20	Desmin-related cardiomyopathy in transgenic mice: a cardiac amyloidosis. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2004 , 101, 10132-6	11.5	222
19	Overexpression of phospholamban in slow-twitch skeletal muscle is associated with depressed contractile function and muscle remodeling. <i>FASEB Journal</i> , 2004 , 18, 974-6	0.9	14
18	Impact of beta-myosin heavy chain expression on cardiac function during stress. <i>Journal of the American College of Cardiology</i> , 2004 , 44, 2390-7	15.1	188
17	Genetic modification of the heart: exploring necessity and sufficiency in the past 10 years. <i>Journal of Molecular and Cellular Cardiology</i> , 2004 , 36, 643-52	5.8	17
16	AlphaB-crystallin modulates protein aggregation of abnormal desmin. <i>Circulation Research</i> , 2003 , 93, 998-1005	15.7	102
15	Reengineering inducible cardiac-specific transgenesis with an attenuated myosin heavy chain promoter. <i>Circulation Research</i> , 2003 , 92, 609-16	15.7	210
14	Molecular mechanics of mouse cardiac myosin isoforms. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2002 , 283, H1446-54	5.2	77
13	Ischemic protection and myofibrillar cardiomyopathy: dose-dependent effects of in vivo deltaPKC inhibition. <i>Circulation Research</i> , 2002 , 91, 741-8	15.7	71
12	Desmin filaments and cardiac disease: establishing causality. <i>Journal of Cardiac Failure</i> , 2002 , 8, S287-92	3.3	39
11	Expression of R120G-alphaB-crystallin causes aberrant desmin and alphaB-crystallin aggregation and cardiomyopathy in mice. <i>Circulation Research</i> , 2001 , 89, 84-91	15.7	248
10	Mouse model of desmin-related cardiomyopathy. <i>Circulation</i> , 2001 , 103, 2402-7	16.7	166
9	Remodeling the cardiac sarcomere using transgenesis. <i>Annual Review of Physiology</i> , 2000 , 62, 261-87	23.1	40
8	Transgenic modeling of a cardiac troponin I mutation linked to familial hypertrophic cardiomyopathy. <i>Circulation Research</i> , 2000 , 87, 805-11	15.7	130
7	Cardiotrophic effects of protein kinase C epsilon: analysis by in vivo modulation of PKCepsilon translocation. <i>Circulation Research</i> , 2000 , 86, 1173-9	15.7	189

6	In vivo modeling of myosin binding protein C familial hypertrophic cardiomyopathy. <i>Circulation Research</i> , 1999 , 85, 841-7	15.7	82
5	Transgenic over-expression of a motor protein at high levels results in severe cardiac pathology. <i>Transgenic Research</i> , 1999 , 8, 9-22	3.3	31
4	An in vivo analysis of transcriptional elements in the mouse alpha-myosin heavy chain gene promoter. <i>Transgenic Research</i> , 1995 , 4, 397-405	3.3	36
3	Position independent expression and developmental regulation is directed by the beta myosin heavy chain gene's 5' upstream region in transgenic mice. <i>Nucleic Acids Research</i> , 1995 , 23, 3301-9	20.1	38
2	In vivo definition of a cardiac specific promoter and its potential utility in remodeling the heart. <i>Annals of the New York Academy of Sciences</i> , 1995 , 752, 492-505	6.5	24
1	Isolation of Neonatal and Adult Rat Cardiomyocytes 117-124		