Steven Baxter Marston

List of Publications by Year
in descending order

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124

times ranked

3402

citing authors

#	ARTICLE	IF	CITATIONS
1	Evidence From Human Myectomy Samples That <i>MYBPC3</i> Mutations Cause Hypertrophic Cardiomyopathy Through Haploinsufficiency. Circulation Research, 2009, 105, 219-222.	4.5	210
2	Troponin phosphorylation and regulatory function in human heart muscle: Dephosphorylation of Ser23/24 on troponin I could account for the contractile defect in end-stage heart failure. Journal of Molecular and Cellular Cardiology, 2007, 42, 247-259.	1.9	162
3	Atomic model of the human cardiac muscle myosin filament. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 318-323.	7.1	153
4	Dilated Cardiomyopathy Mutations in Three Thin Filament Regulatory Proteins Result in a Common Functional Phenotype. Journal of Biological Chemistry, 2005, 280, 28498-28506.	3.4	133
5	Analysis of cardiac myosin binding protein-C phosphorylation in human heart muscle. Journal of Molecular and Cellular Cardiology, 2010, 49, 1003-1011.	1.9	132
6	Alpha-cardiac actin mutations produce atrial septal defects. Human Molecular Genetics, 2008, 17, 256-265.	2.9	128
7	Alterations in Thin Filament Regulation Induced by a Human Cardiac Troponin T Mutant That Causes Dilated Cardiomyopathy Are Distinct from Those Induced by Troponin T Mutants That Cause Hypertrophic Cardiomyopathy. Journal of Biological Chemistry, 2002, 277, 40710-40716.	3.4	125
8	How Do Mutations in Contractile Proteins Cause the Primary Familial Cardiomyopathies?. Journal of Cardiovascular Translational Research, 2011, 4, 245-255.	2.4	106
9	Purification and properties of Ca2+-regulated thin filaments and F-actin from sheep aorta smooth muscle. Journal of Muscle Research and Cell Motility, 1984, 5, 559-575.	2.0	97
10	In Vitro Motility Analysis of Actin-Tropomyosin Regulation by Troponin and Calcium. Journal of Biological Chemistry, 1995, 270, 7836-7841.	3.4	97
11	Myosin binding protein C phosphorylation in normal, hypertrophic and failing human heart muscle. Journal of Molecular and Cellular Cardiology, 2008, 45, 209-216.	1.9	97
12	How do MYBPC3 mutations cause hypertrophic cardiomyopathy?. Journal of Muscle Research and Cell Motility, 2012, 33, 75-80.	2.0	93
13	Mutation Update and Genotype-Phenotype Correlations of Novel and Previously Described Mutations in<i>TPM2</i>and<i>TPM3</i>Causing Congenital Myopathies. Human Mutation, 2014, 35, 779-790.	2.5	92
14	Genotypeâ€“phenotype correlations in ACTA1 mutations that cause congenital myopathies. Neuromuscular Disorders, 2009, 19, 6-16.	0.6	87
15	Fatal hypertrophic cardiomyopathy and nemaline myopathy associated with ACTA1 K336E mutation. Neuromuscular Disorders, 2006, 16, 548-552.	0.6	83
16	Modulation of Thin Filament Activation by Breakdown or Isoform Switching of Thin Filament Proteins. Circulation Research, 2003, 93, 1170-1178.	4.5	81
17	Investigation of a Truncated Cardiac Troponin T That Causes Familial Hypertrophic Cardiomyopathy. Circulation Research, 2000, 86, 1146-1152.	4.5	75
18	Mutations in repeating structural motifs of tropomyosin cause gain of function in skeletal muscle myopathy patients. Human Molecular Genetics, 2013, 22, 4978-4987.	2.9	75

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19	Small heat shock protein Hsp20 (HspB6) as a partner of 14-3-3Î³. Molecular and Cellular Biochemistry, 2007, 295, 9-17.	3.1	71
20	OBSCN Mutations Associated with Dilated Cardiomyopathy and Haploinsufficiency. PLoS ONE, 2015, 10, e0138568.	2.5	70
21	Troponin phosphorylation and myofilament Ca2+-sensitivity in heart failure: Increased or decreased?. Journal of Molecular and Cellular Cardiology, 2008, 45, 603-607.	1.9	69
22	Familial dilated cardiomyopathy mutations uncouple troponin I phosphorylation from changes in myofibrillar Ca2+ sensitivity. Cardiovascular Research, 2013, 99, 65-73.	3.8	68
23	Effect of Hypertrophic Cardiomyopathy Mutations in Human Cardiac Muscle Î± -tropomyosin (Asp175Asn) Tj ETQq1 1 0.784314 rgBT / Motility Assay. Journal of Molecular and Cellular Cardiology, 2000, 32, 1489-1498.	1.9	67
24	Myofilament dysfunction in cardiac disease from mice to men. Journal of Muscle Research and Cell Motility, 2008, 29, 189-201.	2.0	67
25	Distinct hypertrophic cardiomyopathy genotypes result in convergent sarcomeric proteoform profiles revealed by top-down proteomics. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 24691-24700.	7.1	67
26	A simple method for measuring the relative force exerted by myosin on actin filaments in the in vitro motility assay: evidence that tropomyosin and troponin increase force in single thin filaments. Biochemical Journal, 2000, 350, 693-699.	3.7	66
27	The Effect of Mutations in Î±-Tropomyosin (E40K and E54K) That Cause Familial Dilated Cardiomyopathy on the Regulatory Mechanism of Cardiac Muscle Thin Filaments. Journal of Biological Chemistry, 2007, 282, 13487-13497.	3.4	65
28	The pathogenesis ofACTA1-related congenital fiber type disproportion. Annals of Neurology, 2007, 61, 552-561.	5.3	63
29	Myosin Regulatory Light Chain (RLC) Phosphorylation Change as a Modulator of Cardiac Muscle Contraction in Disease. Journal of Biological Chemistry, 2013, 288, 13446-13454.	3.4	63
30	Normal passive viscoelasticity but abnormal myofibrillar force generation in human hypertrophic cardiomyopathy. Journal of Molecular and Cellular Cardiology, 2010, 49, 737-745.	1.9	61
31	The use of phosphateâ€affinity SDSâ€PAGE to measure the cardiac troponin I phosphorylation site distribution in human heart muscle. Proteomics - Clinical Applications, 2009, 3, 1371-1382.	1.6	58
32	Troponin structure and function: a view of recent progress. Journal of Muscle Research and Cell Motility, 2020, 41, 71-89.	2.0	57
33	Molecular Mechanism of the E99K Mutation in Cardiac Actin (ACTC Gene) That Causes Apical Hypertrophy in Man and Mouse. Journal of Biological Chemistry, 2011, 286, 27582-27593.	3.4	56
34	Investigation of a transgenic mouse model of familial dilated cardiomyopathy. Journal of Molecular and Cellular Cardiology, 2010, 49, 380-389.	1.9	53
35	A simple method for automatic tracking of actin filaments in the motility assay. Journal of Muscle Research and Cell Motility, 1996, 17, 497-506.	2.0	51
36	Investigating the role of uncoupling of troponin I phosphorylation from changes in myofibrillar Ca2+-sensitivity in the pathogenesis of cardiomyopathy. Frontiers in Physiology, 2014, 5, 315.	2.8	51

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37	Mutations in troponin T associated with Hypertrophic Cardiomyopathy increase Ca2+-sensitivity and suppress the modulation of Ca2+-sensitivity by troponin I phosphorylation. Archives of Biochemistry and Biophysics, 2016, 601, 113-120.	3.0	49
38	Tropomyosin and Troponin Regulation of Wild Type and E93K Mutant Actin Filaments from Drosophila Flight Muscle. Journal of Biological Chemistry, 1998, 273, 15016-15021.	3.4	48
39	Abnormal actin binding of aberrant Î²-tropomyosins is a molecular cause of muscle weakness in <i>TPM2</i>-related nemaline and cap myopathy. Biochemical Journal, 2012, 442, 231-239.	3.7	48
40	GSK3Î² Phosphorylates Newly Identified Site in the Proline-Alanineâ€“Rich Region of Cardiac Myosinâ€“Binding Protein C and Alters Cross-Bridge Cycling Kinetics in Human. Circulation Research, 2013, 112, 633-639.	4.5	48
41	Functional Analysis of a Unique Troponin C Mutation, GLY159ASP, that Causes Familial Dilated Cardiomyopathy, Studied in Explanted Heart Muscle. Circulation: Heart Failure, 2009, 2, 456-464.	3.9	46
42	The flexibility of two tropomyosin mutants, D175N and E180G, that cause hypertrophic cardiomyopathy. Biochemical and Biophysical Research Communications, 2012, 424, 493-496.	2.1	43
43	The molecular phenotype of human cardiac myosin associated with hypertrophic obstructive cardiomyopathy. Cardiovascular Research, 2008, 79, 481-491.	3.8	41
44	Structure and properties of K141E mutant of small heat shock protein HSP22 (HspB8, H11) that is expressed in human neuromuscular disorders. Archives of Biochemistry and Biophysics, 2006, 454, 32-41.	3.0	40
45	Abnormal contractility in human heart myofibrils from patients with dilated cardiomyopathy due to mutations in TTN and contractile protein genes. Scientific Reports, 2017, 7, 14829.	3.3	40
46	In vitro Motility Analysis of Thin Filaments from Failing and Non-failing Human Heart: Troponin from Failing Human Hearts Induces Slower Filament Sliding and Higher Ca2+ Sensitivity. Journal of Molecular and Cellular Cardiology, 2002, 34, 469-482.	1.9	37
47	<scp><i>TPM</i></scp><i>3</i> deletions cause a hypercontractile congenital muscle stiffness phenotype. Annals of Neurology, 2015, 78, 982-994.	5.3	36
48	Small Molecules Acting on Myofilaments as Treatments for Heart and Skeletal Muscle Diseases. International Journal of Molecular Sciences, 2020, 21, 9599.	4.1	36
49	Myofibrillar Ca2+ sensitivity is uncoupled from troponin I phosphorylation in hypertrophic obstructive cardiomyopathy due to abnormal troponin T. Cardiovascular Research, 2013, 97, 500-508.	3.8	34
50	Effect of mutations in the Î²5â€“Î²7 loop on the structure and properties of human small heat shock protein HSP22 (HspB8, H11). FEBS Journal, 2007, 274, 5628-5642.	4.7	31
51	Two mutations in troponin I that cause hypertrophic cardiomyopathy have contrasting effects on cardiac muscle contractility. Biochemical Journal, 2002, 362, 443-451.	3.7	30
52	The functional effects of mutations Thr673â†’ Asp and Ser702â†’ Asp at the Pro-directed kinase phosophorylation sites in the C-terminus of chicken gizzard caldesmon. FEBS Letters, 1993, 327, 85-89.	2.8	29
53	Uncoupling of myofilament Ca²⁺sensitivity from troponin I phosphorylation by mutations can be reversed by epigallocatechin-3-gallate. Cardiovascular Research, 2015, 108, 99-110.	3.8	29
54	From genotype to phenotype: a longitudinal study of a patient with hypertrophic cardiomyopathy due to a mutation in the MYBPC3 gene. Journal of Muscle Research and Cell Motility, 2008, 29, 239-246.	2.0	28

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55	Skeletal muscle myopathy mutations at the actin tropomyosin interface that cause gain- or loss-of-function. Journal of Muscle Research and Cell Motility, 2013, 34, 165-169.	2.0	27
56	The Molecular Mechanisms of Mutations in Actin and Myosin that Cause Inherited Myopathy. International Journal of Molecular Sciences, 2018, 19, 2020.	4.1	27
57	Evidence against the regulation of caldesmon inhibitory activity by p42/p44erkmitogen-activated protein kinase in vitro and demonstration of another caldesmon kinase in intact gizzard smooth muscle. FEBS Letters, 1999, 452, 254-258.	2.8	26
58	Investigation of changes in skeletal muscle Î±-actin expression in normal and pathological human and mouse hearts. Journal of Muscle Research and Cell Motility, 2010, 31, 207-214.	2.0	25
59	Tropomyosin isoform expression and phosphorylation in the human heart in health and disease. Journal of Muscle Research and Cell Motility, 2013, 34, 189-197.	2.0	25
60	Mechanical and energetic properties of papillary muscle from<i>ACTC</i>E99K transgenic mouse models of hypertrophic cardiomyopathy. American Journal of Physiology - Heart and Circulatory Physiology, 2013, 304, H1513-H1524.	3.2	25
61	Obscurin variants and inherited cardiomyopathies. Biophysical Reviews, 2017, 9, 239-243.	3.2	25
62	The homozygous K280N troponin T mutation alters cross-bridge kinetics and energetics in human HCM. Journal of General Physiology, 2019, 151, 18-29.	1.9	25
63	A simple method for measuring the relative force exerted by myosin on actin filaments in the in vitro motility assay: evidence that tropomyosin and troponin increase force in single thin filaments. Biochemical Journal, 2000, 350, 693.	3.7	24
64	Functional characterisation of a mutant actin (Met132Val) from a patient with nemaline myopathy. Neuromuscular Disorders, 2004, 14, 167-174.	0.6	24
65	Why Is there a Limit to the Changes in Myofilament Ca2+-Sensitivity Associated with Myopathy Causing Mutations?. Frontiers in Physiology, 2016, 7, 415.	2.8	23
66	The Dilated Cardiomyopathy-Causing Mutation ACTC E361G in Cardiac Muscle Myofibrils Specifically Abolishes Modulation of Ca 2+ Regulation by Phosphorylation of Troponin I. Biophysical Journal, 2014, 107, 2369-2380.	0.5	22
67	Troponin structure: its modulation by Ca²⁺and phosphorylation studied by molecular dynamics simulations. Physical Chemistry Chemical Physics, 2016, 18, 20691-20707.	2.8	21
68	Cardiac and skeletal myopathies: can genotype explain phenotype?. , 2001, 22, 1-4.		20
69	Molecular Defects in Cardiac Myofilament Ca2+-Regulation Due to Cardiomyopathy-Linked Mutations Can Be Reversed by Small Molecules Binding to Troponin. Frontiers in Physiology, 2018, 9, 243.	2.8	19
70	Random walks with thin filaments: application of in vitro motility assay to the study of actomyosin regulation. Journal of Muscle Research and Cell Motility, 2003, 24, 149-156.	2.0	18
71	Role of Caldesmon in the Ca2+ Regulation of Smooth Muscle Thin Filaments. Journal of Biological Chemistry, 2008, 283, 47-56.	3.4	18
72	A dilated cardiomyopathy mutation blunts adrenergic response and induces contractile dysfunction under chronic angiotensin II stress. American Journal of Physiology - Heart and Circulatory Physiology, 2015, 309, H1936-H1946.	3.2	18

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73	Tropomyosin Must Interact Weakly with Actin to Effectively Regulate Thin Filament Function. Biophysical Journal, 2017, 113, 2444-2451.	0.5	18
74	Back to the future: new techniques show that forgotten phosphorylation sites are present in contractile proteins of the heart whilst intensively studied sites appear to be absent. Journal of Muscle Research and Cell Motility, 2009, 30, 93-95.	2.0	16
75	Titin-truncating mutations associated with dilated cardiomyopathy alter length-dependent activation and its modulation via phosphorylation. Cardiovascular Research, 2022, 118, 241-253.	3.8	16
76	Approaches to High-Throughput Analysis of Cardiomyocyte Contractility. Frontiers in Physiology, 2020, 11, 612.	2.8	16
77	Identification of casein kinase II as a major endogeneous caldesmon kinase in sheep aorta smooth muscle. FEBS Letters, 1993, 334, 18-22.	2.8	15
78	Investigations into the Sarcomeric Protein and Ca2+-Regulation Abnormalities Underlying Hypertrophic Cardiomyopathy in Cats (Felix catus). Frontiers in Physiology, 2017, 8, 348.	2.8	15
79	Phosphorylation of vascular smooth muscle caldesmon by endogenous kinase. FEBS Letters, 1992, 305, 192-196.	2.8	14
80	A novel Ca2+ binding protein associated with caldesmon in Ca2+-regulated smooth muscle thin filaments: evidence for a structurally altered form of calmodulin. Journal of Muscle Research and Cell Motility, 2000, 21, 537-549.	2.0	14
81	Z-band Alternatively Spliced PDZ Motif Protein (ZASP) Is the Major O-Linked Î²-N-Acetylglucosamine-substituted Protein in Human Heart Myofibrils. Journal of Biological Chemistry, 2013, 288, 4891-4898.	3.4	12
82	Modulation of cardiac thin filament structure by phosphorylated troponin-I analyzed by protein-protein docking and molecular dynamics simulation. Archives of Biochemistry and Biophysics, 2022, 725, 109282.	3.0	11
83	Localization of phospholipid-binding sites of caldesmon. FEBS Letters, 1994, 342, 176-180.	2.8	10
84	The Importance of Intrinsically Disordered Segments of Cardiac Troponin in Modulating Function by Phosphorylation and Disease-Causing Mutations. Frontiers in Physiology, 2016, 7, 508.	2.8	10
85	Direct visualisation and kinetic analysis of normal and nemaline myopathy actin polymerisation using total internal reflection microscopy. Journal of Muscle Research and Cell Motility, 2009, 30, 85-92.	2.0	9
86	Small molecule studies: the fourth wave of muscle research. Journal of Muscle Research and Cell Motility, 2019, 40, 69-76.	2.0	9
87	Instrumentation to study myofibril mechanics from static to artificial simulations of cardiac cycle. MethodsX, 2016, 3, 156-170.	1.6	8
88	Age- and strain-related aberrant Ca2+ release is associated with sudden cardiac death in the ACTC E99K mouse model of hypertrophic cardiomyopathy. American Journal of Physiology - Heart and Circulatory Physiology, 2017, 313, H1213-H1226.	3.2	8
89	Introducing a special edition of the Journal of Muscle Research and Cell Motility on tropomyosin: form and function. Journal of Muscle Research and Cell Motility, 2013, 34, 151-153.	2.0	7
90	A post-MI power struggle: adaptations in cardiac power occur at the sarcomere level alongside MyBP-C and RLC phosphorylation. American Journal of Physiology - Heart and Circulatory Physiology, 2016, 311, H465-H475.	3.2	7

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91	DCM-Causing Mutation E361G in Actin Slows Myofibril Relaxation Kinetics and Uncouples Myofibril Ca2+ Sensitivity from Protein Phosphorylation. Biophysical Journal, 2013, 104, 312a.	0.5	3
92	A Repeating Structural Motif in Tropomyosin that is Responsible for Multiple Gain of Function Skeletal Myopathy Mutations. Biophysical Journal, 2013, 104, 646a-647a.	0.5	3
93	Evidence for reduced troponin I phosphorylation and altered troponin function in patients with hypertrophic obstructive cardiomyopathy. Journal of Molecular and Cellular Cardiology, 2006, 40, 939.	1.9	2
94	How does genotype define phenotype? Microphysiology of a tropomyosin mutation <i>in situ</i> shows the limitations of reductionism. Journal of Physiology, 2008, 586, 2821-2821.	2.9	2
95	Cellular dysfunction and altered contractile protein post-translational modification in hypertrophic cardiomyopathy septal tissue. Journal of Molecular and Cellular Cardiology, 2008, 44, 746.	1.9	2
96	Analysis of Cardiac Myofibrillar Troponin I Phosphorylation in Normal and Failing Human Hearts Using Phos-Tags. Biophysical Journal, 2009, 96, 501a.	0.5	2
97	Modulation of the Interaction between Troponin I N-Terminal Peptide and Troponin C by Phosphorylation Studied by Molecular Dynamics. Biophysical Journal, 2014, 106, 349a.	0.5	2
98	The Molecular Defects in Ca2+ Regulation due to Mutations that Cause Hypertrophic Cardiomyopathy can be Reversed by Small Molecules that Bind to Troponin. Biophysical Journal, 2018, 114, 37a.	0.5	2
99	Donor hearts in the Sydney Heart Bank: reliable control but is it â€˜normalâ€™ heart?. Biophysical Reviews, 2020, 12, 799-803.	3.2	2
100	Pressure Overload Is Associated With Low Levels of Troponin I and Myosin Binding Protein C Phosphorylation in the Hearts of Patients With Aortic Stenosis. Frontiers in Physiology, 2020, 11, 241.	2.8	2
101	Cardiac myocytes and the cardiac action potential. , 2010, , 2604-2618.		2
102	Force Measurements From Myofibril to Filament. Frontiers in Physiology, 2021, 12, 817036.	2.8	2
103	Mouse HCM Model Expressing E99K ACTC Mutation Reproduces Phenotypes As Found In Human Patients. Biophysical Journal, 2009, 96, 499a-500a.	0.5	1
104	Effects of EMD57033 and EGCG on Modulation of Ca2+-Sensitivity by Pka Phosphorylation. Biophysical Journal, 2014, 106, 726a-727a.	0.5	1
105	Epigallocatechin-3-Gallate Reverses the Defects in Modulation of Ca2+-Sensitivity by Troponin I Phosphorylation Caused by Hypertrophic and Dilated Cardiomyopathy Mutations in Cardiac Muscle. Biophysical Journal, 2015, 108, 361a-362a.	0.5	1
106	Primary Effects of HCM Mutations in Humans And Cats. Biophysical Journal, 2016, 110, 123a-124a.	0.5	1
107	Troponin Structure and Effects of Phosphorylation and Mutations Studied by Molecular Dynamics Simulations. Biophysical Journal, 2016, 110, 208a.	0.5	1
108	â€˜(De-)sensitizationâ€™ vs. â€˜Uncouplingâ€™: what drives cardiomyopathies in the thin filament? Reply. Cardiovascular Research, 2016, 109, 187-188.	3.8	1

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109	Direct Evidence In Man For Haploinsufficiency As The Mechanism Of Action Of Myosin-binding Protein C Mutations That Cause Hypertrophic Cardiomyopathy. Biophysical Journal, 2009, 96, 371a.	0.5	0
110	Introducing a series of topical special issues of the Journal of Muscle Research and Cell Motility. Journal of Muscle Research and Cell Motility, 2012, 33, 1-3.	2.0	0
111	Using FRET to Characterize the Actomyosin Complex in Cardiac Muscle. Biophysical Journal, 2013, 104, 16a.	0.5	0
112	There is a Limit to the Changes in Myofilament Ca2+-Sensitivity due to Myopathies. Biophysical Journal, 2013, 104, 312a.	0.5	0
113	Age-Related Cardiac Dysfunction in Transgenic Mice Carrying Actin E99K Mutation. Biophysical Journal, 2014, 106, 344a.	0.5	0
114	Dcm-Causing Mutation E361G in Actin Uncouples Myofibril Ca2+ Sensitivity from Protein Phosphorylation. Biophysical Journal, 2014, 106, 774a-775a.	0.5	0
115	DCM Mutation ACTCE361G Causes Uncoupling of Myofibril Sensitivity from TnI Phosphorylation that can be Reversed by Epigallocatechin-3-Gallate. Biophysical Journal, 2015, 108, 292a.	0.5	0
116	Molecular Dynamics Studies on Phosphorylated and Unphosphorylated Cardiac Troponin. Biophysical Journal, 2015, 108, 447a.	0.5	0
117	Obscurin Mutations Cause Haploinsufficiency and are Common in Patients with Familial Dilated Cardiomyopathy (FDCM). Biophysical Journal, 2015, 108, 292a.	0.5	0
118	Important announcement: a rational nomenclature for tropomyosin variants. Journal of Muscle Research and Cell Motility, 2015, 36, 145-145.	2.0	0
119	Molecular Mechanism of Novel Deletions in TPM3 that cause a Hypercontractile Phenotype with Congenital Muscle Stiffness. Biophysical Journal, 2016, 110, 14a-15a.	0.5	0
120	Effect of Truncated Mutations in the Titin Gene on Cardiac Function. Biophysical Journal, 2018, 114, 498a.	0.5	0
121	The European Muscle Conference 2019 Special Issue. Journal of Muscle Research and Cell Motility, 2019, 40, 67-67.	2.0	0