

Lucie Carrier

List of Publications by Year in descending order

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109
papers

8,385
citations

44042

48
h-index

46771

89
g-index

111
all docs

111
docs citations

111
times ranked

7010
citing authors

#	ARTICLE	IF	CITATIONS
1	The detyrosination/re-tyrosination cycle of tubulin and its role and dysfunction in neurons and cardiomyocytes. <i>Seminars in Cell and Developmental Biology</i> , 2023, 137, 46-62.	2.3	20
2	Targeted therapies in genetic dilated and hypertrophic cardiomyopathies: from molecular mechanisms to therapeutic targets. A position paper from the Heart Failure Association (HFA) and the Working Group on Myocardial Function of the European Society of Cardiology (ESC). <i>European Journal of Heart Failure</i> , 2022, 24, 406-420.	2.9	22
3	Animal models and animal-free innovations for cardiovascular research: current status and routes to be explored. Consensus document of the ESC Working Group on Myocardial Function and the ESC Working Group on Cellular Biology of the Heart. <i>Cardiovascular Research</i> , 2022, 118, 3016-3051.	1.8	30
4	Interpretation and actionability of genetic variants in cardiomyopathies: a position statement from the European Society of Cardiology Council on cardiovascular genomics. <i>European Heart Journal</i> , 2022, 43, 1901-1916.	1.0	32
5	CMYA5 is a novel interaction partner of FHL2 in cardiac myocytes. <i>FEBS Journal</i> , 2022, 289, 4622-4645.	2.2	6
6	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.	4.3	12
7	Towards standardization of echocardiography for the evaluation of left ventricular function in adult rodents: a position paper of the ESC Working Group on Myocardial Function. <i>Cardiovascular Research</i> , 2021, 117, 43-59.	1.8	72
8	Targeting the population for gene therapy with MYBPC3. <i>Journal of Molecular and Cellular Cardiology</i> , 2021, 150, 101-108.	0.9	23
9	Proteomic and Functional Studies Reveal Detyrosinated Tubulin as Treatment Target in Sarcomere Mutation-Induced Hypertrophic Cardiomyopathy. <i>Circulation: Heart Failure</i> , 2021, 14, e007022.	1.6	58
10	Cas9-expressing chickens and pigs as resources for genome editing in livestock. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2021, 118, .	3.3	22
11	A Transgenic Mouse Model of Eccentric Left Ventricular Hypertrophy With Preserved Ejection Fraction Exhibits Alterations in the Autophagy-Lysosomal Pathway. <i>Frontiers in Physiology</i> , 2021, 12, 614878.	1.3	2
12	Cardiovascular magnetic resonance detects microvascular dysfunction in a mouse model of hypertrophic cardiomyopathy. <i>Journal of Cardiovascular Magnetic Resonance</i> , 2021, 23, 63.	1.6	3
13	Generation of bi-allelic MYBPC3 truncating mutant and isogenic control from an iPSC line of a patient with hypertrophic cardiomyopathy. <i>Stem Cell Research</i> , 2021, 55, 102489.	0.3	1
14	Translational investigation of electrophysiology in hypertrophic cardiomyopathy. <i>Journal of Molecular and Cellular Cardiology</i> , 2021, 157, 77-89.	0.9	16
15	Ouabain worsens diastolic sarcomere length in myocytes from a cardiomyopathy mouse model. <i>European Journal of Pharmacology</i> , 2021, 904, 174170.	1.7	1
16	A Novel miRNA Screen Identifies miRNA-4454 as a Candidate Biomarker for Ventricular Fibrosis in Patients with Hypertrophic Cardiomyopathy. <i>Biomolecules</i> , 2021, 11, 1718.	1.8	14
17	Autophagy in cardiomyopathies. <i>Biochimica Et Biophysica Acta - Molecular Cell Research</i> , 2020, 1867, 118432.	1.9	29
18	Is Desmin Propensity to Aggregate Part of its Protective Function?. <i>Cells</i> , 2020, 9, 491.	1.8	19

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19	Gene therapy for inherited arrhythmias. <i>Cardiovascular Research</i> , 2020, 116, 1635-1650.	1.8	20
20	Depletion of Vasohibin 1 Speeds Contraction and Relaxation in Failing Human Cardiomyocytes. <i>Circulation Research</i> , 2020, 127, e14-e27.	2.0	32
21	Gene therapy strategies in the treatment of hypertrophic cardiomyopathy. <i>Pflugers Archiv European Journal of Physiology</i> , 2019, 471, 807-815.	1.3	52
22	Myoarchitectural disarray of hypertrophic cardiomyopathy begins pre-birth. <i>Journal of Anatomy</i> , 2019, 235, 962-976.	0.9	34
23	Association of Asymmetric Dimethylarginine and Diastolic Dysfunction in Patients with Hypertrophic Cardiomyopathy. <i>Biomolecules</i> , 2019, 9, 277.	1.8	8
24	Analysis of Contractile Function of Permeabilized Human Hypertrophic Cardiomyopathy Multicellular Heart Tissue. <i>Frontiers in Physiology</i> , 2019, 10, 239.	1.3	9
25	Targeted panel sequencing in pediatric primary cardiomyopathy supports a critical role of <i>TNNI3</i> . <i>Clinical Genetics</i> , 2019, 96, 549-559.	1.0	28
26	Disease modeling of a mutation in <i>ACTN2</i> guides clinical therapy in hypertrophic cardiomyopathy. <i>EMBO Molecular Medicine</i> , 2019, 11, e11115.	3.3	88
27	Making Sense of Inhibiting Nonsense in Hypertrophic Cardiomyopathy. <i>Circulation</i> , 2019, 139, 812-814.	1.6	3
28	Biallelic mutation in <i>MYH7</i> and <i>MYBPC3</i> leads to severe cardiomyopathy with left ventricular noncompaction phenotype. <i>Human Mutation</i> , 2019, 40, 1101-1114.	1.1	22
29	Phosphomimetic cardiac myosin-binding protein C partially rescues a cardiomyopathy phenotype in murine engineered heart tissue. <i>Scientific Reports</i> , 2019, 9, 18152.	1.6	13
30	The homozygous K280N troponin T mutation alters cross-bridge kinetics and energetics in human HCM. <i>Journal of General Physiology</i> , 2019, 151, 18-29.	0.9	25
31	Cardiomyopathy phenotypes in human-induced pluripotent stem cell-derived cardiomyocytes—a systematic review. <i>Pflugers Archiv European Journal of Physiology</i> , 2019, 471, 755-768.	1.3	57
32	Mechanistic role of the CREB-regulated transcription coactivator 1 in cardiac hypertrophy. <i>Journal of Molecular and Cellular Cardiology</i> , 2019, 127, 31-43.	0.9	5
33	S100A4 as a Target of the E3-Ligase Asb2 ^{Δ2} and Its Effect on Engineered Heart Tissue. <i>Frontiers in Physiology</i> , 2018, 9, 1292.	1.3	3
34	CRISPR/Cas9 editing in human pluripotent stem cell-cardiomyocytes highlights arrhythmias, hypocontractility, and energy depletion as potential therapeutic targets for hypertrophic cardiomyopathy. <i>European Heart Journal</i> , 2018, 39, 3879-3892.	1.0	176
35	Diltiazem prevents stress-induced contractile deficits in cardiomyocytes, but does not reverse the cardiomyopathy phenotype in <i>Mybpc3</i> knock mice. <i>Journal of Physiology</i> , 2017, 595, 3987-3999.	1.3	18
36	Blinded Contractility Analysis in hiPSC-Cardiomyocytes in Engineered Heart Tissue Format: Comparison With Human Atrial Trabeculae. <i>Toxicological Sciences</i> , 2017, 158, 164-175.	1.4	52

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37	Evaluation of MYBPC3 trans -Splicing and Gene Replacement as Therapeutic Options in Human iPSC-Derived Cardiomyocytes. <i>Molecular Therapy - Nucleic Acids</i> , 2017, 7, 475-486.	2.3	74
38	Activation of Autophagy Ameliorates Cardiomyopathy in <i>Mybpc3</i> -Targeted Knockin Mice. <i>Circulation: Heart Failure</i> , 2017, 10, .	1.6	53
39	Nebivolol Desensitizes Myofilaments of a Hypertrophic Cardiomyopathy Mouse Model. <i>Frontiers in Physiology</i> , 2017, 8, 558.	1.3	11
40	Epigallocatechin-3-Gallate Accelerates Relaxation and Ca ²⁺ Transient Decay and Desensitizes Myofilaments in Healthy and <i>Mybpc3</i> -Targeted Knock-in Cardiomyopathic Mice. <i>Frontiers in Physiology</i> , 2016, 7, 607.	1.3	16
41	The embryological basis of subclinical hypertrophic cardiomyopathy. <i>Scientific Reports</i> , 2016, 6, 27714.	1.6	29
42	Comparison of the effects of a truncating and a missense MYBPC3 mutation on contractile parameters of engineered heart tissue. <i>Journal of Molecular and Cellular Cardiology</i> , 2016, 97, 82-92.	0.9	48
43	Selective phosphorylation of PKA targets after β^2 -adrenergic receptor stimulation impairs myofilament function in <i>Mybpc3</i> -targeted HCM mouse model. <i>Cardiovascular Research</i> , 2016, 110, 200-214.	1.8	28
44	Serum Matrix Metalloproteinases as Quantitative Biomarkers for Myocardial Fibrosis and Sudden Cardiac Death Risk Stratification in Patients With Hypertrophic Cardiomyopathy. <i>Journal of Cardiac Failure</i> , 2016, 22, 845-850.	0.7	31
45	<i>S</i> -glutathiolation impairs phosphoregulation and function of cardiac myosin-binding protein C in human heart failure. <i>FASEB Journal</i> , 2016, 30, 1849-1864.	0.2	38
46	Ranolazine antagonizes catecholamine-induced dysfunction in isolated cardiomyocytes, but lacks long-term therapeutic effects <i>in vivo</i> in a mouse model of hypertrophic cardiomyopathy. <i>Cardiovascular Research</i> , 2016, 109, 90-102.	1.8	38
47	I-1-deficiency negatively impacts survival in a cardiomyopathy mouse model. <i>IJC Heart and Vasculature</i> , 2015, 8, 87-94.	0.6	3
48	Targeted <i>Mybpc3</i> Knock-Out Mice with Cardiac Hypertrophy Exhibit Structural Mitral Valve Abnormalities. <i>Journal of Cardiovascular Development and Disease</i> , 2015, 2, 48-65.	0.8	9
49	Research priorities in sarcomeric cardiomyopathies. <i>Cardiovascular Research</i> , 2015, 105, 449-456.	1.8	48
50	Targets for therapy in sarcomeric cardiomyopathies. <i>Cardiovascular Research</i> , 2015, 105, 457-470.	1.8	122
51	Animal and in silico models for the study of sarcomeric cardiomyopathies. <i>Cardiovascular Research</i> , 2015, 105, 439-448.	1.8	45
52	<i>PLEKHM2</i> mutation leads to abnormal localization of lysosomes, impaired autophagy flux and associates with recessive dilated cardiomyopathy and left ventricular noncompaction. <i>Human Molecular Genetics</i> , 2015, 24, 7227-7240.	1.4	55
53	Cardiac myosin-binding protein C (MYBPC3) in cardiac pathophysiology. <i>Gene</i> , 2015, 573, 188-197.	1.0	148
54	Changes in the cardiac metabolome caused by perhexiline treatment in a mouse model of hypertrophic cardiomyopathy. <i>Molecular BioSystems</i> , 2015, 11, 564-573.	2.9	34

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55	The E3 ubiquitin ligase Asb2 ¹² is downregulated in a mouse model of hypertrophic cardiomyopathy and targets desmin for proteasomal degradation. <i>Journal of Molecular and Cellular Cardiology</i> , 2015, 87, 214-224.	0.9	35
56	Sexual dimorphic response to exercise in hypertrophic cardiomyopathy-associated MYBPC3-targeted knock-in mice. <i>Pflügers Archiv European Journal of Physiology</i> , 2015, 467, 1303-1317.	1.3	35
57	Automated analysis of contractile force and Ca ²⁺ transients in engineered heart tissue. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2014, 306, H1353-H1363.	1.5	69
58	Proteasome inhibition slightly improves cardiac function in mice with hypertrophic cardiomyopathy. <i>Frontiers in Physiology</i> , 2014, 5, 484.	1.3	24
59	Mybpc3 gene therapy for neonatal cardiomyopathy enables long-term disease prevention in mice. <i>Nature Communications</i> , 2014, 5, 5515.	5.8	131
60	FHL2 expression and variants in hypertrophic cardiomyopathy. <i>Basic Research in Cardiology</i> , 2014, 109, 451.	2.5	58
61	Endothelin ¹ Induces Myofibrillar Disarray and Contractile Vector Variability in Hypertrophic Cardiomyopathy ¹ Induced Pluripotent Stem Cell ¹ Derived Cardiomyocytes. <i>Journal of the American Heart Association</i> , 2014, 3, e001263.	1.6	131
62	MYBPC3 in hypertrophic cardiomyopathy: from mutation identification to RNA-based correction. <i>Pflügers Archiv European Journal of Physiology</i> , 2014, 466, 215-223.	1.3	34
63	Ubiquitin-proteasome system and hereditary cardiomyopathies. <i>Journal of Molecular and Cellular Cardiology</i> , 2014, 71, 25-31.	0.9	64
64	Contractile abnormalities and altered drug response in engineered heart tissue from Mybpc3-targeted knock-in mice. <i>Journal of Molecular and Cellular Cardiology</i> , 2013, 63, 189-198.	0.9	65
65	A novel genetic variant in the transcription factor Islet-1 exerts gain of function on myocyte enhancer factor 2C promoter activity. <i>European Journal of Heart Failure</i> , 2013, 15, 267-276.	2.9	21
66	GSK3 ¹² Phosphorylates Newly Identified Site in the Proline-Alanine ¹ Rich Region of Cardiac Myosin ¹ Binding Protein C and Alters Cross-Bridge Cycling Kinetics in Human. <i>Circulation Research</i> , 2013, 112, 633-639.	2.0	48
67	Impact of ANKRD1 mutations associated with hypertrophic cardiomyopathy on contraction parameters of engineered heart tissue. <i>Basic Research in Cardiology</i> , 2013, 108, 349.	2.5	40
68	Heterozygous LmnadelK32 mice develop dilated cardiomyopathy through a combined pathomechanism of haploinsufficiency and peptide toxicity. <i>Human Molecular Genetics</i> , 2013, 22, 3152-3164.	1.4	72
69	Perturbed Length-Dependent Activation in Human Hypertrophic Cardiomyopathy With Missense Sarcomeric Gene Mutations. <i>Circulation Research</i> , 2013, 112, 1491-1505.	2.0	191
70	Rescue of cardiomyopathy through U7sn ¹ RNA ¹ mediated exon skipping in Mybpc3 ¹ targeted knock ¹ in mice. <i>EMBO Molecular Medicine</i> , 2013, 5, 1128-1145.	3.3	85
71	Repair of Mybpc3 mRNA by 5 ¹ trans-splicing in a Mouse Model of Hypertrophic Cardiomyopathy. <i>Molecular Therapy - Nucleic Acids</i> , 2013, 2, e102.	2.3	61
72	Protein kinase D increases maximal Ca ²⁺ -activated tension of cardiomyocyte contraction by phosphorylation of cMyBP-C-Ser ³¹⁵ . <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2012, 303, H323-H331.	1.5	20

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73	Localization of Islet-1-Positive Cells in the Healthy and Infarcted Adult Murine Heart. <i>Circulation Research</i> , 2012, 110, 1303-1310.	2.0	87
74	Contractile Dysfunction Irrespective of the Mutant Protein in Human Hypertrophic Cardiomyopathy With Normal Systolic Function. <i>Circulation: Heart Failure</i> , 2012, 5, 36-46.	1.6	127
75	Evidence for FHL1 as a novel disease gene for isolated hypertrophic cardiomyopathy. <i>Human Molecular Genetics</i> , 2012, 21, 3237-3254.	1.4	106
76	Genetics of Hypertrophic and Dilated Cardiomyopathy. <i>Current Pharmaceutical Biotechnology</i> , 2012, 13, 2467-2476.	0.9	23
77	Increased myofilament Ca ²⁺ sensitivity and diastolic dysfunction as early consequences of Mybpc3 mutation in heterozygous knock-in mice. <i>Journal of Molecular and Cellular Cardiology</i> , 2012, 52, 1299-1307.	0.9	118
78	The miRNA-212/132 family regulates both cardiac hypertrophy and cardiomyocyte autophagy. <i>Nature Communications</i> , 2012, 3, 1078.	5.8	518
79	How do MYBPC3 mutations cause hypertrophic cardiomyopathy?. <i>Journal of Muscle Research and Cell Motility</i> , 2012, 33, 75-80.	0.9	93
80	Adrenergic stress reveals septal hypertrophy and proteasome impairment in heterozygous Mybpc3-targeted knock-in mice. <i>Journal of Muscle Research and Cell Motility</i> , 2012, 33, 5-15.	0.9	41
81	Defective proteolytic systems in Mybpc3-targeted mice with cardiac hypertrophy. <i>Basic Research in Cardiology</i> , 2012, 107, 235.	2.5	91
82	Genetics of Hypertrophic and Dilated Cardiomyopathy. <i>Current Pharmaceutical Biotechnology</i> , 2012, 13, 2467-2476.	0.9	25
83	Distinction Between Two Populations of Islet-1-Positive Cells in Hearts of Different Murine Strains. <i>Stem Cells and Development</i> , 2011, 20, 1043-1052.	1.1	32
84	Cardiac myosin-binding protein C in hypertrophic cardiomyopathy: Mechanisms and therapeutic opportunities. <i>Journal of Molecular and Cellular Cardiology</i> , 2011, 50, 613-620.	0.9	96
85	The ubiquitin-proteasome system in cardiomyopathies. <i>Current Opinion in Cardiology</i> , 2011, 26, 190-195.	0.8	63
86	Atrogin-1 and MuRF1 regulate cardiac MyBP-C levels via different mechanisms. <i>Cardiovascular Research</i> , 2010, 85, 357-366.	1.8	81
87	The ubiquitin-proteasome system and nonsense-mediated mRNA decay in hypertrophic cardiomyopathy. <i>Cardiovascular Research</i> , 2010, 85, 330-338.	1.8	73
88	Nonsense-Mediated mRNA Decay and Ubiquitin-Proteasome System Regulate Cardiac Myosin-Binding Protein C Mutant Levels in Cardiomyopathic Mice. <i>Circulation Research</i> , 2009, 105, 239-248.	2.0	152
89	A new polymorphism in human calmodulin III gene promoter is a potential modifier gene for familial hypertrophic cardiomyopathy. <i>European Heart Journal</i> , 2009, 30, 1648-1655.	1.0	39
90	Cardiac Myosin-Binding Protein C Mutations and Hypertrophic Cardiomyopathy. <i>Circulation</i> , 2009, 119, 1473-1483.	1.6	275

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91	Prevention of Myofilament Dysfunction by β -Blocker Therapy in Postinfarct Remodeling. <i>Circulation: Heart Failure</i> , 2009, 2, 233-242.	1.6	38
92	Ubiquitin-Proteasome System Impairment Caused by a Missense Cardiac Myosin-binding Protein C Mutation and Associated with Cardiac Dysfunction in Hypertrophic Cardiomyopathy. <i>Journal of Molecular Biology</i> , 2008, 384, 896-907.	2.0	80
93	The ubiquitin-proteasome system in cardiac dysfunction. <i>Biochimica Et Biophysica Acta - Molecular Basis of Disease</i> , 2008, 1782, 749-763.	1.8	129
94	Cardiac Myosin-Binding Protein C Is Required for Complete Relaxation in Intact Myocytes. <i>Circulation Research</i> , 2007, 101, 928-938.	2.0	117
95	Decreased phosphorylation levels of cardiac myosin-binding protein-C in human and experimental heart failure. <i>Journal of Molecular and Cellular Cardiology</i> , 2007, 43, 223-229.	0.9	141
96	Length and protein kinase A modulations of myocytes in cardiac myosin binding protein C-deficient mice. <i>Cardiovascular Research</i> , 2006, 69, 370-380.	1.8	112
97	Impairment of the ubiquitin-proteasome system by truncated cardiac myosin binding protein C mutants. <i>Cardiovascular Research</i> , 2005, 66, 33-44.	1.8	139
98	Expression of cardiac myosin-binding protein-C (cMyBP-C) in <i>Drosophila</i> as a model for the study of human cardiomyopathies. <i>Human Molecular Genetics</i> , 2005, 14, 7-17.	1.4	12
99	Asymmetric septal hypertrophy in heterozygous cMyBP-C null mice. <i>Cardiovascular Research</i> , 2004, 63, 293-304.	1.8	129
100	Human homozygous R403W mutant cardiac myosin presents disproportionate enhancement of mechanical and enzymatic properties. <i>Journal of Molecular and Cellular Cardiology</i> , 2004, 36, 355-362.	0.9	75
101	Hypertrophic Cardiomyopathy. <i>Circulation</i> , 2003, 107, 2227-2232.	1.6	1,129
102	Effect of MyBP-C Binding to Actin on Contractility in Heart Muscle. <i>Journal of General Physiology</i> , 2003, 122, 761-774.	0.9	109
103	Homozygotes for a R869G Mutation in the β -myosin Heavy Chain Gene have a Severe Form of Familial Hypertrophic Cardiomyopathy. <i>Journal of Molecular and Cellular Cardiology</i> , 2000, 32, 1575-1583.	0.9	59
104	COOH-terminal truncated cardiac myosin-binding protein C mutants resulting from familial hypertrophic cardiomyopathy mutations exhibit altered expression and/or incorporation in fetal rat cardiomyocytes. <i>Journal of Molecular Biology</i> , 1999, 294, 443-456.	2.0	68
105	Cardiac Myosin-Binding Protein C and Hypertrophic Cardiomyopathy. <i>Trends in Cardiovascular Medicine</i> , 1998, 8, 151-157.	2.3	11
106	Cardiac Myosin Binding Protein C Gene Is Specifically Expressed in Heart During Murine and Human Development. <i>Circulation Research</i> , 1998, 82, 130-133.	2.0	57
107	Clinical Features and Prognostic Implications of Familial Hypertrophic Cardiomyopathy Related to the Cardiac Myosin-Binding Protein C Gene. <i>Circulation</i> , 1998, 97, 2230-2236.	1.6	241
108	Organization and Sequence of Human Cardiac Myosin Binding Protein C Gene (MYBPC3) and Identification of Mutations Predicted to Produce Truncated Proteins in Familial Hypertrophic Cardiomyopathy. <i>Circulation Research</i> , 1997, 80, 427-434.	2.0	240

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109	Cardiac myosin binding proteinâ€C gene splice acceptor site mutation is associated with familial hypertrophic cardiomyopathy. <i>Nature Genetics</i> , 1995, 11, 438-440.	9.4	417