Julie R Mcmullen

List of Publications by Year
in descending order

40 papers	4,111 citations	331259 21 h-index	329751 37 g-index
41 all docs	41 docs citations	41 times ranked	5425 citing authors

#	Article	IF	Citations
1	Protein phosphatase 2A in the healthy and failing heart: New insights and therapeutic opportunities. Cellular Signalling, 2022, 91, 110213.	1.7	4
2	IGF1â€“PI3K-inducedÂphysiological cardiac hypertrophy: Implications for new heart failure therapies, biomarkers, and predicting cardiotoxicity. Journal of Sport and Health Science, 2021, 10, 637-647.	3.3	24
3	Novel Lipid Species for Detecting and Predicting Atrial Fibrillation in Patients With Type 2 Diabetes. Diabetes, 2021, 70, 255-261.	0.3	9
4	FoxO1 is required for physiological cardiac hypertrophy induced by exercise but not by constitutively active PI3K. American Journal of Physiology - Heart and Circulatory Physiology, 2021, 320, H1470-H1485.	1.5	15
5	Proteome characterisation of extracellular vesicles isolated from heart. Proteomics, 2021, 21, e2100026.	1.3	28
6	Prevention of Pathological Atrial Remodeling and Atrial Fibrillation. Journal of the American College of Cardiology, 2021, 77, 2846-2864.	1.2	46
7	Tissue-specific expression of Cas9 has no impact on whole-body metabolism in four transgenic mouse lines. Molecular Metabolism, 2021, 53, 101292.	3.0	5
8	Overexpression of Heat Shock Protein 70 Improves Cardiac Remodeling and Survival in Protein Phosphatase 2A-Expressing Transgenic Mice with Chronic Heart Failure. Cells, 2021, 10, 3180.	1.8	4
9	A Step-By-Step Method to Detect Neutralizing Antibodies Against AAV using a Colorimetric Cell-Based Assay. Journal of Visualized Experiments, 2021, , .	0.2	1
10	Clusterin is regulated by IGF1â€“PI3K signaling in the heart: implications for biomarker and drug target discovery, and cardiotoxicity. Archives of Toxicology, 2020, 94, 1763-1768.	1.9	10
11	CORP: Practical tools for improving experimental design and reporting of laboratory studies of cardiovascular physiology and metabolism. American Journal of Physiology - Heart and Circulatory Physiology, 2019, 317, H627-H639.	1.5	10
12	Inhibition of heat shock protein 70 blocks the development of cardiac hypertrophy by modulating the phosphorylation of histone deacetylase 2. Cardiovascular Research, 2019, 115, 1850-1860.	1.8	23
13	Adeno-Associated Virus Gene Therapy: Translational Progress and Future Prospects in the Treatment of Heart Failure. Heart Lung and Circulation, 2018, 27, 1285-1300.	0.2	30
14	Distinct lipidomic profiles in models of physiological and pathological cardiac remodeling, and potential therapeutic strategies. Biochimica Et Biophysica Acta - Molecular and Cell Biology of Lipids, 2018, 1863, 219-234.	1.2	21
15	Lipidomic Profiles of the Heart and Circulation in Response to Exercise versus Cardiac Pathology: A Resource of Potential Biomarkers and Drug Targets. Cell Reports, 2018, 24, 2757-2772.	2.9	55
16	Understanding Key Mechanisms of Exercise-Induced Cardiac Protection to Mitigate Disease: Current Knowledge and Emerging Concepts. Physiological Reviews, 2018, 98, 419-475.	13.1	120
17	PP2A negatively regulates the hypertrophic response by dephosphorylating HDAC2 S394 in the heart. Experimental and Molecular Medicine, 2018, 50, 1-14.	3.2	22
18	Improving the quality of preclinical research echocardiography: observations, training, and guidelines for measurement. American Journal of Physiology - Heart and Circulatory Physiology, 2018, 315, H58-H70.	1.5	37

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19	Divergent Effects of PKC (Protein Kinase C) Î± in the Human and Animal Heart?. Circulation Genomic and Precision Medicine, 2018, 11, e002104.	1.6	3
20	The Interplay of Protein Coding and Non-Coding RNAs (circRNAs, lncRNAs) During Cardiac Differentiation. EBioMedicine, 2017, 25, 9-10.	2.7	9
21	Molecular Aspects of Exercise-induced Cardiac Remodeling. Cardiology Clinics, 2016, 34, 515-530.	0.9	30
22	<i>Smad7</i> gene delivery prevents muscle wasting associated with cancer cachexia in mice. Science Translational Medicine, 2016, 8, 348ra98.	5.8	70
23	Therapeutic potential of targeting microRNAs to regulate cardiac fibrosis: miR-433 a new fibrotic player. Annals of Translational Medicine, 2016, 4, 548-548.	0.7	8
24	Therapeutic targeting of oxidative stress with coenzyme Q10 counteracts exaggerated diabetic cardiomyopathy in a mouse model of diabetes with diminished PI3K(p110Î±) signaling. Free Radical Biology and Medicine, 2015, 87, 137-147.	1.3	63
25	Spontaneous ventricular tachyarrhythmias in Î²₂-adrenoceptor transgenic mice in relation to cardiac interstitial fibrosis. American Journal of Physiology - Heart and Circulatory Physiology, 2015, 309, H946-H957.	1.5	35
26	Long-Term Overexpression of Hsp70 Does Not Protect against Cardiac Dysfunction and Adverse Remodeling in a MURC Transgenic Mouse Model with Chronic Heart Failure and Atrial Fibrillation. PLoS ONE, 2015, 10, e0145173.	1.1	15
27	The small-molecule BGP-15 protects against heart failure and atrial fibrillation in mice. Nature Communications, 2014, 5, 5705.	5.8	86
28	Diabetic cardiomyopathy: Mechanisms and new treatment strategies targeting antioxidant signaling pathways. , 2014, 142, 375-415.		437
29	Silencing of miR-34a Attenuates Cardiac Dysfunction in a Setting of Moderate, but Not Severe, Hypertrophic Cardiomyopathy. PLoS ONE, 2014, 9, e90337.	1.1	67
30	Phosphoinositide 3-Kinase p110Î± Is a Master Regulator of Exercise-Induced Cardioprotection and PI3K Gene Therapy Rescues Cardiac Dysfunction. Circulation: Heart Failure, 2012, 5, 523-534.	1.6	115
31	Molecular distinction between physiological and pathological cardiac hypertrophy: Experimental findings and therapeutic strategies. , 2010, 128, 191-227.		694
32	PI3K(p110Î±) Protects Against Myocardial Infarction-Induced Heart Failure. Arteriosclerosis, Thrombosis, and Vascular Biology, 2010, 30, 724-732.	1.1	160
33	Reduced Phosphoinositide 3-Kinase (p110Î±) Activation Increases the Susceptibility to Atrial Fibrillation. American Journal of Pathology, 2009, 175, 998-1009.	1.9	151
34	Protective effects of exercise and phosphoinositide 3-kinase(p110Â) signaling in dilated and hypertrophic cardiomyopathy. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 612-617.	3.3	269
35	PI3K(p110Î±) Inhibitors as Anti-Cancer Agents: Minding the Heart. Cell Cycle, 2007, 6, 910-913.	1.3	35
36	Modulation of TLR2 induces cardioprotection through a Phosphoinositide 3â€Kinase Dependent Mechanism. FASEB Journal, 2007, 21, A867.	0.2	0

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37	Inhibition of mTOR Signaling With Rapamycin Regresses Established Cardiac Hypertrophy Induced by Pressure Overload. Circulation, 2004, 109, 3050-3055.	1.6	456
38	Deletion of Ribosomal S6 Kinases Does Not Attenuate Pathological, Physiological, or Insulin-Like Growth Factor 1 Receptor-Phosphoinositide 3-Kinase-Induced Cardiac Hypertrophy. Molecular and Cellular Biology, 2004, 24, 6231-6240.	1.1	111
39	The Insulin-like Growth Factor 1 Receptor Induces Physiological Heart Growth via the Phosphoinositide 3-Kinase(p110Î±) Pathway. Journal of Biological Chemistry, 2004, 279, 4782-4793.	1.6	350
40	Phosphoinositide 3-kinase(p110Â) plays a critical role for the induction of physiological, but not pathological, cardiac hypertrophy. Proceedings of the National Academy of Sciences of the United States of America, 2003, 100, 12355-12360.	3.3	483