Kate L Weeks

List of Publications by Year in descending order

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KATE I MEEKS

#	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	Lymphangiogenesis: A new player in the heart's adaptive response to exercise. Journal of Sport and Health Science, 2022, , .	3.3	0
3	Glycogen-autophagy: Molecular machinery and cellular mechanisms of glycophagy. Journal of Biological Chemistry, 2022, 298, 102093.	1.6	16
4	Maternal obesity: influencing the heart right from the start. Journal of Physiology, 2022, 600, 3007-3008.	1.3	0
5	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
6	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
7	Gene therapy targeting cardiac phosphoinositide 3-kinase (p110α) attenuates cardiac remodeling in type 2 diabetes. American Journal of Physiology - Heart and Circulatory Physiology, 2020, 318, H840-H852.	1.5	32
8	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
9	Histone deacetylases in cardiovascular and metabolic diseases. Journal of Molecular and Cellular Cardiology, 2019, 130, 151-159.	0.9	65
10	HDAC inhibitors and cardioprotection: Homing in on a mechanism of action. EBioMedicine, 2019, 40, 21-22.	2.7	4
11	Role of type 2A phosphatase regulatory subunit B56α in regulating cardiac responses to β-adrenergic stimulation in vivo. Cardiovascular Research, 2019, 115, 519-529.	1.8	9
12	Gene delivery of medium chain acyl-coenzyme A dehydrogenase induces physiological cardiac hypertrophy and protects against pathological remodelling. Clinical Science, 2018, 132, 381-397.	1.8	17
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	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
15	Nucleocytoplasmic shuttling: The ins and outs of quantitative imaging. Clinical and Experimental Pharmacology and Physiology, 2018, 45, 1087-1094.	0.9	5
16	Divergent Effects of PKC (Protein Kinase C) α in the Human and Animal Heart?. Circulation Genomic and Precision Medicine, 2018, 11, e002104.	1.6	3
17	Phosphoinositide 3-kinase (p110α) gene delivery limits diabetes-induced cardiac NADPH oxidase and cardiomyopathy in a mouse model with established diastolic dysfunction. Clinical Science, 2017, 131, 1345-1360.	1.8	49
18	βâ€Adrenergic Stimulation Induces Histone Deacetylase 5 (HDAC5) Nuclear Accumulation in Cardiomyocytes by B55αâ€PP2Aâ€Mediated Dephosphorylation. Journal of the American Heart Association, 2017, 6, .	1.6	29

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19	The IGF1-PI3K-Akt Signaling Pathway in Mediating Exercise-Induced Cardiac Hypertrophy and Protection. Advances in Experimental Medicine and Biology, 2017, 1000, 187-210.	0.8	74
20	HSP70: therapeutic potential in acute and chronic cardiac disease settings. Future Medicinal Chemistry, 2016, 8, 2177-2183.	1.1	10
21	Assessing structural and functional responses of murine hearts to acute and sustained β-adrenergic stimulation in vivo. Journal of Pharmacological and Toxicological Methods, 2016, 79, 60-71.	0.3	14
22	Pathophysiology of cardiac hypertrophy and heart failure: signaling pathways and novel therapeutic targets. Archives of Toxicology, 2015, 89, 1401-1438.	1.9	492
23	Roles and postâ€translational regulation of cardiac class IIa histone deacetylase isoforms. Journal of Physiology, 2015, 593, 1785-1797.	1.3	34
24	Exchanging cardiac phenotype: Is AKT-mediated NHE1 inhibition a permissive switch in physiological hypertrophy?. Journal of Molecular and Cellular Cardiology, 2014, 77, 175-177.	0.9	1
25	Phosphoinositide 3-Kinase p110α 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
26	Follistatin-mediated skeletal muscle hypertrophy is regulated by Smad3 and mTOR independently of myostatin. Journal of Cell Biology, 2012, 197, 997-1008.	2.3	167
27	The Athlete's Heart vs. the Failing Heart: Can Signaling Explain the Two Distinct Outcomes?. Physiology, 2011, 26, 97-105.	1.6	114
28	Molecular distinction between physiological and pathological cardiac hypertrophy: Experimental findings and therapeutic strategies. , 2010, 128, 191-227.		694
29	PI3K(p110α) Protects Against Myocardial Infarction-Induced Heart Failure. Arteriosclerosis, Thrombosis, and Vascular Biology, 2010, 30, 724-732.	1.1	160
30	Role of phosphoinositide 3-kinases in regulating cardiac function. Frontiers in Bioscience - Landmark, 2009, Volume, 2221.	3.0	11
31	The protective effects of exercise and phosphoinositide 3-kinase (p110α) in the failing heart. Clinical Science, 2009, 116, 365-375.	1.8	12
32	PROMOTING PHYSIOLOGICAL HYPERTROPHY IN THE FAILING HEART. Clinical and Experimental Pharmacology and Physiology, 2008, 35, 438-441.	0.9	7