Lewis J Watson

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/6354726/publications.pdf

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19	1,345	16	17
papers	citations	h-index	g-index
19	19	19	2295
all docs	docs citations	times ranked	citing authors

#	Article	IF	CITATIONS
1	Metabolomic Analysis of Pressure-Overloaded and Infarcted Mouse Hearts. Circulation: Heart Failure, 2014, 7, 634-642.	1.6	181
2	Circulating Exosomes Induced by Cardiac Pressure Overload Contain Functional Angiotensin II Type 1 Receptors. Circulation, 2015, 131, 2120-2130.	1.6	177
3	O-linked \hat{I}^2 - <i>N</i> -acetylglucosamine transferase is indispensable in the failing heart. Proceedings of the National Academy of Sciences of the United States of America, 2010, 107, 17797-17802.	3.3	170
4	Augmented O-GlcNAc signaling attenuates oxidative stress and calcium overload in cardiomyocytes. Amino Acids, 2011, 40, 895-911.	1.2	145
5	Non-canonical glycosyltransferase modulates post-hypoxic cardiac myocyte death and mitochondrial permeability transition. Journal of Molecular and Cellular Cardiology, 2008, 45, 313-325.	0.9	106
6	O-GlcNAc signaling is essential for NFAT-mediated transcriptional reprogramming during cardiomyocyte hypertrophy. American Journal of Physiology - Heart and Circulatory Physiology, 2012, 302, H2122-H2130.	1.5	96
7	Cardiomyocyte <i>Ogt</i> is essential for postnatal viability. American Journal of Physiology - Heart and Circulatory Physiology, 2014, 306, H142-H153.	1.5	78
8	High Fat Feeding in Mice Is Insufficient to Induce Cardiac Dysfunction and Does Not Exacerbate Heart Failure. PLoS ONE, 2013, 8, e83174.	1.1	69
9	MicroRNA-539 Is Up-regulated in Failing Heart, and Suppresses O-GlcNAcase Expression. Journal of Biological Chemistry, 2014, 289, 29665-29676.	1.6	63
10	The two-pore domain potassium channel TREK-1 mediates cardiac fibrosis and diastolic dysfunction. Journal of Clinical Investigation, 2018, 128, 4843-4855.	3.9	62
11	Cardiac overexpression of 8-oxoguanine DNA glycosylase 1 protects mitochondrial DNA and reduces cardiac fibrosis following transaortic constriction. American Journal of Physiology - Heart and Circulatory Physiology, 2011, 301, H2073-H2080.	1.5	43
12	Cardiac phosphatase-deficient 6-phosphofructo-2-kinase/fructose-2,6-bisphosphatase increases glycolysis, hypertrophy, and myocyte resistance to hypoxia. American Journal of Physiology - Heart and Circulatory Physiology, 2008, 294, H2889-H2897.	1.5	40
13	Cardiomyocyte Ogt limits ventricular dysfunction in mice following pressure overload without affecting hypertrophy. Basic Research in Cardiology, 2017, 112, 23.	2.5	38
14	Low-dose simvastatin improves survival and ventricular function via eNOS in congestive heart failure. American Journal of Physiology - Heart and Circulatory Physiology, 2006, 291, H2743-H2751.	1.5	30
15	Reduced Cardiac Fructose 2,6 Bisphosphate Increases Hypertrophy and Decreases Glycolysis following Aortic Constriction. PLoS ONE, 2013, 8, e53951.	1.1	24
16	Phosphorylation of Src by phosphoinositide 3-kinase regulates beta-adrenergic receptor-mediated EGFR transactivation. Cellular Signalling, 2016, 28, 1580-1592.	1.7	21
17	Loss of Oâ€GlcNAc transferase activity sensitizes cardiac myocytes to postâ€hypoxic death. FASEB Journal, 2008, 22, 750.10.	0.2	2
18	Oâ€GlcNAcase Exacerbates Postâ€Hypoxic Cardiac Myocyte Death. FASEB Journal, 2007, 21, A1376.	0.2	0

#	Article	IF	CITATIONS
19	Oâ€GlcNAc Transferase is a Proâ€Survival Enzyme in Postâ€Hypoxic Cardiac Myocytes. FASEB Journal, 2007, 21, A800.	0.2	O