

Lewis J Watson

List of Publications by Year in descending order

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Version: 2024-02-01

19
papers

1,345
citations

516215

16
h-index

887659

17
g-index

19
all docs

19
docs citations

19
times ranked

2295
citing authors

#	ARTICLE	IF	CITATIONS
1	The two-pore domain potassium channel TREK-1 mediates cardiac fibrosis and diastolic dysfunction. <i>Journal of Clinical Investigation</i> , 2018, 128, 4843-4855.	3.9	62
2	Cardiomyocyte Ogt limits ventricular dysfunction in mice following pressure overload without affecting hypertrophy. <i>Basic Research in Cardiology</i> , 2017, 112, 23.	2.5	38
3	Phosphorylation of Src by phosphoinositide 3-kinase regulates beta-adrenergic receptor-mediated EGFR transactivation. <i>Cellular Signalling</i> , 2016, 28, 1580-1592.	1.7	21
4	Circulating Exosomes Induced by Cardiac Pressure Overload Contain Functional Angiotensin II Type 1 Receptors. <i>Circulation</i> , 2015, 131, 2120-2130.	1.6	177
5	MicroRNA-539 Is Up-regulated in Failing Heart, and Suppresses O-GlcNAcase Expression. <i>Journal of Biological Chemistry</i> , 2014, 289, 29665-29676.	1.6	63
6	Metabolomic Analysis of Pressure-Overloaded and Infarcted Mouse Hearts. <i>Circulation: Heart Failure</i> , 2014, 7, 634-642.	1.6	181
7	Cardiomyocyte <i>Ogt</i> is essential for postnatal viability. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2014, 306, H142-H153.	1.5	78
8	Reduced Cardiac Fructose 2,6 Bisphosphate Increases Hypertrophy and Decreases Glycolysis following Aortic Constriction. <i>PLoS ONE</i> , 2013, 8, e53951.	1.1	24
9	High Fat Feeding in Mice Is Insufficient to Induce Cardiac Dysfunction and Does Not Exacerbate Heart Failure. <i>PLoS ONE</i> , 2013, 8, e83174.	1.1	69
10	O-GlcNAc signaling is essential for NFAT-mediated transcriptional reprogramming during cardiomyocyte hypertrophy. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2012, 302, H2122-H2130.	1.5	96
11	Cardiac overexpression of 8-oxoguanine DNA glycosylase 1 protects mitochondrial DNA and reduces cardiac fibrosis following transaortic constriction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2011, 301, H2073-H2080.	1.5	43
12	Augmented O-GlcNAc signaling attenuates oxidative stress and calcium overload in cardiomyocytes. <i>Amino Acids</i> , 2011, 40, 895-911.	1.2	145
13	O-linked N-acetylglucosamine transferase is indispensable in the failing heart. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2010, 107, 17797-17802.	3.3	170
14	Non-canonical glycosyltransferase modulates post-hypoxic cardiac myocyte death and mitochondrial permeability transition. <i>Journal of Molecular and Cellular Cardiology</i> , 2008, 45, 313-325.	0.9	106
15	Cardiac phosphatase-deficient 6-phosphofructo-2-kinase/fructose-2,6-bisphosphatase increases glycolysis, hypertrophy, and myocyte resistance to hypoxia. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2008, 294, H2889-H2897.	1.5	40
16	Loss of O-GlcNAc transferase activity sensitizes cardiac myocytes to post-hypoxic death. <i>FASEB Journal</i> , 2008, 22, 750.10.	0.2	2
17	O-GlcNAcase Exacerbates Post-hypoxic Cardiac Myocyte Death. <i>FASEB Journal</i> , 2007, 21, A1376.	0.2	0
18	O-GlcNAc Transferase is a Survival Enzyme in Post-hypoxic Cardiac Myocytes. <i>FASEB Journal</i> , 2007, 21, A800.	0.2	0

#	ARTICLE	IF	CITATIONS
19	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