

# Janet R Manning

## List of Publications by Year in descending order

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Version: 2024-02-01

24  
papers

482  
citations

840776

11  
h-index

713466

21  
g-index

28  
all docs

28  
docs citations

28  
times ranked

673  
citing authors

#	ARTICLE	IF	CITATIONS
1	Myocardial brain-derived neurotrophic factor regulates cardiac bioenergetics through the transcription factor Yin Yang 1. <i>Cardiovascular Research</i> , 2023, 119, 571-586.	3.8	12
2	Diet-induced obese mice are resistant to improvements in cardiac function resulting from short-term adropin treatment. <i>Current Research in Physiology</i> , 2022, 5, 55-62.	1.7	3
3	GPER-dependent estrogen signaling increases cardiac GCN5L1 expression. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2022, 322, H762-H768.	3.2	6
4	Loss of the mitochondrial phosphate carrier SLC25A3 induces remodeling of the cardiac mitochondrial protein acylome. <i>American Journal of Physiology - Cell Physiology</i> , 2021, 321, C519-C534.	4.6	8
5	Phosphoproteomic analysis identifies phospho-Threonine-17 site of phospholamban important in low molecular weight isoform of fibroblast growth factor 2-induced protection against post-ischemic cardiac dysfunction. <i>Journal of Molecular and Cellular Cardiology</i> , 2020, 148, 1-14.	1.9	2
6	Cardiomyocyte-specific deletion of GCN5L1 in mice restricts mitochondrial protein hyperacetylation in response to a high fat diet. <i>Scientific Reports</i> , 2020, 10, 10665.	3.3	17
7	Calreticulin expression in human cardiac myocytes induces ER stress-associated apoptosis. <i>Physiological Reports</i> , 2020, 8, e14400.	1.7	8
8	Increased fatty acid oxidation enzyme activity in the hearts of mice fed a high fat diet does not correlate with improved cardiac contractile function. <i>Current Research in Physiology</i> , 2020, 3, 44-49.	1.7	4
9	Loss of GCN5L1 in cardiac cells disrupts glucose metabolism and promotes cell death via reduced Akt/mTORC2 signaling. <i>Biochemical Journal</i> , 2019, 476, 1713-1724.	3.7	22
10	Adropin reduces blood glucose levels in mice by limiting hepatic glucose production. <i>Physiological Reports</i> , 2019, 7, e14043.	1.7	34
11	Loss of GCN5L1 in cardiac cells limits mitochondrial respiratory capacity under hyperglycemic conditions. <i>Physiological Reports</i> , 2019, 7, e14054.	1.7	9
12	Adropin treatment restores cardiac glucose oxidation in pre-diabetic obese mice. <i>Journal of Molecular and Cellular Cardiology</i> , 2019, 129, 174-178.	1.9	41
13	Cardiac-specific deletion of GCN5L1 restricts recovery from ischemia-reperfusion injury. <i>Journal of Molecular and Cellular Cardiology</i> , 2019, 129, 69-78.	1.9	19
14	The protein acetylase GCN5L1 modulates hepatic fatty acid oxidation activity via acetylation of the mitochondrial $\beta$ -oxidation enzyme HADHA. <i>Journal of Biological Chemistry</i> , 2018, 293, 17676-17684.	3.4	62
15	Rad GTPase Deletion Attenuates Post-Ischemic Cardiac Dysfunction and Remodeling. <i>JACC Basic To Translational Science</i> , 2018, 3, 83-96.	4.1	3
16	Adropin regulates pyruvate dehydrogenase in cardiac cells via a novel GPCR-MAPK-PDK4 signaling pathway. <i>Redox Biology</i> , 2018, 18, 25-32.	9.0	66
17	Acetylation of mitochondrial proteins by GCN5L1 promotes enhanced fatty acid oxidation in the heart. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2017, 313, H265-H274.	3.2	60
18	Rad-deletion Phenocopies Tonic Sympathetic Stimulation of the Heart. <i>Journal of Cardiovascular Translational Research</i> , 2016, 9, 432-444.	2.4	17

#	ARTICLE	IF	CITATIONS
19	Loss of Rad-GTPase produces a novel adaptive cardiac phenotype resistant to systolic decline with aging. American Journal of Physiology - Heart and Circulatory Physiology, 2015, 309, H1336-H1345.	3.2	11
20	Quantitative Phosphoproteomics Using Acetone-Based Peptide Labeling: Method Evaluation and Application to a Cardiac Ischemia/Reperfusion Model. Journal of Proteome Research, 2013, 12, 4268-4279.	3.7	13
21	Low molecular weight fibroblast growth factor-2 signals via protein kinase C and myofibrillar proteins to protect against postischemic cardiac dysfunction. American Journal of Physiology - Heart and Circulatory Physiology, 2013, 304, H1382-H1396.	3.2	12
22	Rad GTPase Deletion Increases L-type Calcium Channel Current Leading to Increased Cardiac Contraction. Journal of the American Heart Association, 2013, 2, e000459.	3.7	42
23	Identification of Divergent Regulatory Mechanisms across the RSK Family of Small GTPases. FASEB Journal, 2013, 27, 598.3.	0.5	0
24	Fibroblast growth factor-2-induced cardioprotection against myocardial infarction occurs via the interplay between nitric oxide, protein kinase signaling, and ATP-sensitive potassium channels. Growth Factors, 2012, 30, 124-139.	1.7	10