Yow Keat Tham

List of Publications by Year
in descending order

Source: https://exaly.com/author-pdf/7080422/publications.pdf

Version: 2024-02-01

		687363	996975
16	1,514	13	15
papers	citations	h-index	g-index

16	16	16	2745
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Pathophysiology of cardiac hypertrophy and heart failure: signaling pathways and novel therapeutic targets. Archives of Toxicology, 2015, 89, 1401-1438.	4.2	492
2	Therapeutic inhibition of the miR-34 family attenuates pathological cardiac remodeling and improves heart function. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 17615-17620.	7.1	391
3	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.	3.9	115
4	Enhanced phosphoinositide 3-kinase(p110Î±) activity prevents diabetes-induced cardiomyopathy and superoxide generation in a mouse model of diabetes. Diabetologia, 2012, 55, 3369-3381.	6.3	88
5	The small-molecule BGP-15 protects against heart failure and atrial fibrillation in mice. Nature Communications, 2014, 5, 5705.	12.8	86
6	Therapeutic silencing of miRâ€652 restores heart function and attenuates adverse remodeling in a setting of established pathological hypertrophy. FASEB Journal, 2014, 28, 5097-5110.	0.5	74
7	Silencing of miR-34a Attenuates Cardiac Dysfunction in a Setting of Moderate, but Not Severe, Hypertrophic Cardiomyopathy. PLoS ONE, 2014, 9, e90337.	2.5	67
8	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.	6.4	55
9	Inhibition of miR-154 Protects Against Cardiac Dysfunction and Fibrosis in a Mouse Model of Pressure Overload. Scientific Reports, 2016, 6, 22442.	3.3	43
10	Sex differences in response to miRNAâ€34a therapy in mouse models of cardiac disease: identification of sexâ€, diseaseâ€•and treatmentâ€regulated miRNAs. Journal of Physiology, 2016, 594, 5959-5974.	2.9	40
11	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.	2.4	21
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.	4.3	17
13	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.	3.2	15
14	Novel Lipid Species for Detecting and Predicting Atrial Fibrillation in Patients With Type 2 Diabetes. Diabetes, 2021, 70, 255-261.	0.6	9
15	Forkhead box protein O1 (FoxO1) is required for exercise-induced, but not PI3K-induced, physiological cardiac hypertrophy. Journal of Molecular and Cellular Cardiology, 2018, 124, 93.	1.9	1
16	O166 Inhibition of miRNA-652 Using LNA-antimiRs Improves Cardiac Function in a Mouse Model of Pressure Overload and is Associated with Preserved Angiogenesis and Upregulation of Jagged 1. , 2014, 9, e47.		0