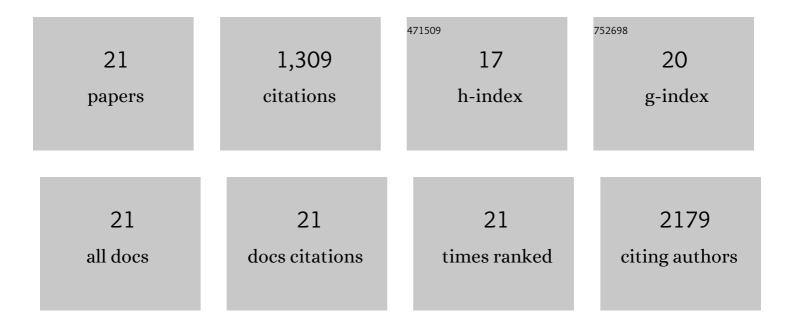
## Xiao-Ming Gao

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/8906243/publications.pdf

Version: 2024-02-01



XIAO-MING GAO

#	Article	IF	CITATIONS
1	Mouse model of post-infarct ventricular rupture: time course, strain- and gender-dependency, tensile strength, and histopathology. Cardiovascular Research, 2005, 65, 469-477.	3.8	156
2	Inhibition of mTOR reduces chronic pressure-overload cardiac hypertrophy and fibrosis. Journal of Hypertension, 2006, 24, 1663-1670.	0.5	142
3	Down-regulation of mitofusin-2 expression in cardiac hypertrophy in vitro and in vivo. Life Sciences, 2007, 80, 2154-2160.	4.3	113
4	Small-molecule-biased formyl peptide receptor agonist compound 17b protects against myocardial ischaemia-reperfusion injury in mice. Nature Communications, 2017, 8, 14232.	12.8	104
5	Deletion of macrophage migration inhibitory factor protects the heart from severe ischemia–reperfusion injury: A predominant role of anti-inflammation. Journal of Molecular and Cellular Cardiology, 2011, 50, 991-999.	1.9	99
6	Relaxin remodels fibrotic healing following myocardial infarction. Laboratory Investigation, 2011, 91, 675-690.	3.7	93
7	Post-infarct cardiac rupture: Recent insights on pathogenesis and therapeutic interventions. , 2012, 134, 156-179.		86
8	Regression of pressure overload-induced left ventricular hypertrophy in mice. American Journal of Physiology - Heart and Circulatory Physiology, 2005, 288, H2702-H2707.	3.2	79
9	Sex Hormones and Cardiomyopathic Phenotype Induced by Cardiac β2-Adrenergic Receptor Overexpression. Endocrinology, 2003, 144, 4097-4105.	2.8	73
10	Long non-coding RNAs H19, MALAT1 and MIAT as potential novel biomarkers for diagnosis of acute myocardial infarction. Biomedicine and Pharmacotherapy, 2019, 118, 109208.	5.6	54
11	Differential roles of cardiac and leukocyte derived macrophage migration inhibitory factor in inflammatory responses and cardiac remodelling post myocardial infarction. Journal of Molecular and Cellular Cardiology, 2014, 69, 32-42.	1.9	52
12	Infarct size and post-infarct inflammation determine the risk of cardiac rupture in mice. International Journal of Cardiology, 2010, 143, 20-28.	1.7	48
13	Endogenous Relaxin Does Not Affect Chronic Pressure Overload-Induced Cardiac Hypertrophy and Fibrosis. Endocrinology, 2008, 149, 476-482.	2.8	38
14	Platelet-Targeted Delivery of Peripheral Blood Mononuclear Cells to the Ischemic Heart Restores Cardiac Function after Ischemia-Reperfusion Injury. Theranostics, 2017, 7, 3192-3206.	10.0	36
15	Preserved ventricular contractility in infarcted mouse heart overexpressing β <sub>2</sub> -adrenergic receptors. American Journal of Physiology - Heart and Circulatory Physiology, 2000, 279, H2456-H2463.	3.2	31
16	Relaxin mitigates microvascular damage and inflammation following cardiac ischemia–reperfusion. Basic Research in Cardiology, 2019, 114, 30.	5.9	28
17	Splenic release of platelets contributes to increased circulating platelet size and inflammation after myocardial infarction. Clinical Science, 2016, 130, 1089-1104.	4.3	20
18	Microvascular leakage in acute myocardial infarction: characterization by histology, biochemistry, and magnetic resonance imaging. American Journal of Physiology - Heart and Circulatory Physiology, 2017, 312, H1068-H1075.	3.2	19

#	Article	IF	CITATIONS
19	Cardioprotective effects of constitutively active MEK1 against H2O2-induced apoptosis and autophagy in cardiomyocytes via the ERK1/2 signaling pathway. Biochemical and Biophysical Research Communications, 2019, 512, 125-130.	2.1	16
20	Mutant DD genotype of NFKB1 gene is associated with the susceptibility and severity of coronary artery disease. Journal of Molecular and Cellular Cardiology, 2017, 103, 56-64.	1.9	11
21	NFKB1 gene rs28362491 polymorphism is associated with the susceptibility of acute coronary syndrome. Bioscience Reports, 2019, 39, .	2.4	11