Rugmani Padmanabhan Iyer

List of Publications by Year
in descending order

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31 papers	2,457 citations	257101 24 h-index	433756 31 g-index
31 all docs	31 docs citations	31 times ranked	3895 citing authors

#	Article	IF	Citations
1	Matrix Metalloproteinase-9: Many Shades of Function in Cardiovascular Disease. Physiology, 2013, 28, 391-403.	1.6	385
2	IL-10 improves cardiac remodeling after myocardial infarction by stimulating M2 macrophage polarization and fibroblast activation. Basic Research in Cardiology, 2017, 112, 33.	2.5	278
3	Temporal neutrophil polarization following myocardial infarction. Cardiovascular Research, 2016, 110, 51-61.	1.8	253
4	Cardiac Fibroblast Activation Post-Myocardial Infarction: Current Knowledge Gaps. Trends in Pharmacological Sciences, 2017, 38, 448-458.	4.0	151
5	A Novel Collagen Matricryptin Reduces Left Ventricular Dilation Post-Myocardial Infarction by Promoting Scar Formation and Angiogenesis. Journal of the American College of Cardiology, 2015, 66, 1364-1374.	1.2	145
6	The history of matrix metalloproteinases: milestones, myths, and misperceptions. American Journal of Physiology - Heart and Circulatory Physiology, 2012, 303, H919-H930.	1.5	134
7	Myofibroblasts and the extracellular matrix network in post-myocardial infarction cardiac remodeling. Pflugers Archiv European Journal of Physiology, 2014, 466, 1113-27.	1.3	94
8	MMP-9 signaling in the left ventricle following myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2016, 311, H190-H198.	1.5	92
9	Matrix metalloproteinases as input and output signals for post-myocardial infarction remodeling. Journal of Molecular and Cellular Cardiology, 2016, 91, 134-140.	0.9	88
10	LXR/RXR signaling and neutrophil phenotype following myocardial infarction classify sex differences in remodeling. Basic Research in Cardiology, 2018, 113, 40.	2.5	86
11	Early matrix metalloproteinase-12 inhibition worsens post-myocardial infarction cardiac dysfunction by delaying inflammation resolution. International Journal of Cardiology, 2015, 185, 198-208.	0.8	85
12	CD36 Is a Matrix Metalloproteinase-9 Substrate That Stimulates Neutrophil Apoptosis and Removal During Cardiac Remodeling. Circulation: Cardiovascular Genetics, 2016, 9, 14-25.	5.1	78
13	Building a better infarct: Modulation of collagen cross-linking to increase infarct stiffness and reduce left ventricular dilation post-myocardial infarction. Journal of Molecular and Cellular Cardiology, 2015, 85, 229-239.	0.9	59
14	Periodontal-induced chronic inflammation triggers macrophage secretion of Ccl12 to inhibit fibroblast-mediated cardiac wound healing. JCI Insight, 2017, 2, .	2.3	55
15	Early matrix metalloproteinase-9 inhibition post-myocardial infarction worsens cardiac dysfunction by delaying inflammation resolution. Journal of Molecular and Cellular Cardiology, 2016, 100, 109-117.	0.9	52
16	Transgenic overexpression of macrophage matrix metalloproteinase-9 exacerbates age-related cardiac hypertrophy, vessel rarefaction, inflammation, and fibrosis. American Journal of Physiology - Heart and Circulatory Physiology, 2017, 312, H375-H383.	1.5	51
17	Osteopontin is proteolytically processed by matrix metalloproteinase 9. Canadian Journal of Physiology and Pharmacology, 2015, 93, 879-886.	0.7	46
18	Translating Kochâ€™s Postulates to Identify Matrix Metalloproteinase Roles in Postmyocardial Infarction Remodeling. Circulation Research, 2014, 114, 860-871.	2.0	41

#	Article	IF	Citations
19	P. gingivalis lipopolysaccharide intensifies inflammation post-myocardial infarction through matrix metalloproteinase-9. Journal of Molecular and Cellular Cardiology, 2014, 76, 218-226.	0.9	41
20	Citrate Synthase Is a NovelIn VivoMatrix Metalloproteinase-9 Substrate That Regulates Mitochondrial Function in the Postmyocardial Infarction Left Ventricle. Antioxidants and Redox Signaling, 2014, 21, 1974-1985.	2.5	38
21	Macrophage overexpression of matrix metalloproteinase-9 in aged mice improves diastolic physiology and cardiac wound healing after myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2018, 314, H224-H235.	1.5	37
22	Exogenous CXCL4 infusion inhibits macrophage phagocytosis by limiting CD36 signalling to enhance post-myocardial infarction cardiac dilation and mortality. Cardiovascular Research, 2019, 115, 395-408.	1.8	36
23	Aliskiren and valsartan mediate left ventricular remodeling post-myocardial infarction in mice through MMP-9 effects. Journal of Molecular and Cellular Cardiology, 2014, 72, 326-335.	0.9	33
24	Defining the sham environment for post-myocardial infarction studies in mice. American Journal of Physiology - Heart and Circulatory Physiology, 2016, 311, H822-H836.	1.5	27
25	Using proteomics to uncover extracellular matrix interactions during cardiac remodeling. Proteomics - Clinical Applications, 2013, 7, 516-527.	0.8	23
26	Matrix metalloproteinaseâ€9â€dependent mechanisms of reduced contractility and increased stiffness in the aging heart. Proteomics - Clinical Applications, 2016, 10, 92-107.	0.8	15
27	The Mouse Heart Attack Research Tool 1.0 database. American Journal of Physiology - Heart and Circulatory Physiology, 2018, 315, H522-H530.	1.5	14
28	Glycoproteomic Profiling Provides Candidate Myocardial Infarction Predictors of Later Progression to Heart Failure. ACS Omega, 2019, 4, 1272-1280.	1.6	10
29	Using the laws of thermodynamics to understand how matrix metalloproteinases coordinate the myocardial response to injury. Metalloproteinases in Medicine, 2015, 2, 75.	1.0	5
30	Identification of a Disulfide Bridge Important for Transport Function of SNAT4 Neutral Amino Acid Transporter. PLoS ONE, 2013, 8, e56792.	1.1	4
31	N-Glycosylation influences transport, but not cellular trafficking, of a neuronal amino acid transporter SNAT1. Biochemical Journal, 2016, 473, 4227-4242.	1.7	1