Michael J Daseke

List of Publications by Citations

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The third column is the impact factor (IF) of the journal, and the fourth column is the number of citations of the article.

11
papers186
citations7
h-index11
g-index11
ext. papers316
ext. citations6.3
avg, IF3.58
L-index

#	Paper	IF	Citations
11	Fibroblast polarization over the myocardial infarction time continuum shifts roles from inflammation to angiogenesis. <i>Basic Research in Cardiology</i> , 2019 , 114, 6	11.8	72
10	Neutrophil proteome shifts over the myocardial infarction time continuum. <i>Basic Research in Cardiology</i> , 2019 , 114, 37	11.8	41
9	Cardiac fibroblast activation during myocardial infarction wound healing: Fibroblast polarization after MI. <i>Matrix Biology</i> , 2020 , 91-92, 109-116	11.4	23
8	Neutrophil signaling during myocardial infarction wound repair. <i>Cellular Signalling</i> , 2021 , 77, 109816	4.9	15
7	Exogenous IL-4 shuts off pro-inflammation in neutrophils while stimulating anti-inflammation in macrophages to induce neutrophil phagocytosis following myocardial infarction. <i>Journal of Molecular and Cellular Cardiology</i> , 2020 , 145, 112-121	5.8	12
6	Infarct in the Heart: Whates MMP-9 Got to Do with It?. Biomolecules, 2021, 11,	5.9	9
5	The compendium of matrix metalloproteinase expression in the left ventricle of mice following myocardial infarction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2020 , 318, H70	6- 11 714	1 ⁷
4	Understanding the mechanisms that determine extracellular matrix remodeling in the infarcted myocardium. <i>Biochemical Society Transactions</i> , 2019 , 47, 1679-1687	5.1	4
3	S100A9 is a functional effector of infarct wall thinning after myocardial infarction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2021 ,	5.2	2
2	Macrophages secrete murinoglobulin-1 and galectin-3 to regulate neutrophil degranulation after myocardial infarction <i>Molecular Omics</i> , 2022 ,	4.4	1
1	Exogenous IL-4 Promotes Myocardial Infarction Repair by Turning off Pro-Inflammation in Neutrophils while Stimulating Anti-Inflammation in Macrophages to Induce Neutrophil	0.9	