Michael J Daseke

List of Publications by Year
in descending order

11 papers	424 citations	1162367 8 h-index	1372195 10 g-index
11 all docs	11 docs citations	11 times ranked	540 citing authors

#	Article	IF	Citations
1	Fibroblast polarization over the myocardial infarction time continuum shifts roles from inflammation to angiogenesis. Basic Research in Cardiology, 2019, 114, 6.	2.5	118
2	Neutrophil proteome shifts over the myocardial infarction time continuum. Basic Research in Cardiology, 2019, 114, 37.	2.5	78
3	Cardiac fibroblast activation during myocardial infarction wound healing. Matrix Biology, 2020, 91-92, 109-116.	1.5	61
4	Neutrophil signaling during myocardial infarction wound repair. Cellular Signalling, 2021, 77, 109816.	1.7	44
5	Exogenous IL-4 shuts off pro-inflammation in neutrophils while stimulating anti-inflammation in macrophages to induce neutrophil phagocytosis following myocardial infarction. Journal of Molecular and Cellular Cardiology, 2020, 145, 112-121.	0.9	38
6	Infarct in the Heart: Whatâ€™s MMP-9 Got to Do with It?. Biomolecules, 2021, 11, 491.	1.8	37
7	The compendium of matrix metalloproteinase expression in the left ventricle of mice following myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2020, 318, H706-H714.	1.5	16
8	Understanding the mechanisms that determine extracellular matrix remodeling in the infarcted myocardium. Biochemical Society Transactions, 2019, 47, 1679-1687.	1.6	12
9	S100A9 is a functional effector of infarct wall thinning after myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2022, 322, H145-H155.	1.5	11
10	Macrophages secrete murinoglobulin-1 and galectin-3 to regulate neutrophil degranulation after myocardial infarction. Molecular Omics, 2022, 18, 186-195.	1.4	9
11	Exogenous ILâ€4 Promotes Myocardial Infarction Repair by Turning off Proâ€Inflammation in Neutrophils while Stimulating Antiâ€Inflammation in Macrophages to Induce Neutrophil Phagocytosis. FASEB Journal, 2020, 34, 1-1.	0.2	0