

Michael J Daseke

List of Publications by Year in Descending Order

Source: <https://exaly.com/author-pdf/9417837/michael-j-daseke-publications-by-year.pdf>

Version: 2024-04-19

This document has been generated based on the publications and citations recorded by exaly.com. For the latest version of this publication list, visit the link given above.

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

11
papers

186
citations

7
h-index

11
g-index

11
ext. papers

316
ext. citations

6.3
avg, IF

3.58
L-index

#	Paper	IF	Citations
11	Macrophages secrete murinoglobulin-1 and galectin-3 to regulate neutrophil degranulation after myocardial infarction.. <i>Molecular Omics</i> , 2022 ,	4.4	1
10	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
9	Infarct in the Heart: What's MMP-9 Got to Do with It?. <i>Biomolecules</i> , 2021 , 11,	5.9	9
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	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, H706-H714	5.3	7
5	Exogenous IL-4 Promotes Myocardial Infarction Repair by Turning off Pro-Inflammation in Neutrophils while Stimulating Anti-Inflammation in Macrophages to Induce Neutrophil Phagocytosis. <i>FASEB Journal</i> , 2020 , 34, 1-1	0.9	
4	Cardiac fibroblast activation during myocardial infarction wound healing: Fibroblast polarization after MI. <i>Matrix Biology</i> , 2020 , 91-92, 109-116	11.4	23
3	Neutrophil proteome shifts over the myocardial infarction time continuum. <i>Basic Research in Cardiology</i> , 2019 , 114, 37	11.8	41
2	Understanding the mechanisms that determine extracellular matrix remodeling in the infarcted myocardium. <i>Biochemical Society Transactions</i> , 2019 , 47, 1679-1687	5.1	4
1	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