

Michael Kohlhaas

List of Publications by Year in descending order

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Version: 2024-02-01

12
papers

1,359
citations

933447

10
h-index

1281871

11
g-index

14
all docs

14
docs citations

14
times ranked

2337
citing authors

#	ARTICLE	IF	CITATIONS
1	Reversal of Mitochondrial Transhydrogenase Causes Oxidative Stress in Heart Failure. <i>Cell Metabolism</i> , 2015, 22, 472-484.	16.2	307
2	Mitofusin 2-Containing Mitochondrial-Reticular Microdomains Direct Rapid Cardiomyocyte Bioenergetic Responses Via Interorganellar Ca ²⁺ Crosstalk. <i>Circulation Research</i> , 2012, 111, 863-875.	4.5	286
3	Elevated Cytosolic Na ⁺ Increases Mitochondrial Formation of Reactive Oxygen Species in Failing Cardiac Myocytes. <i>Circulation</i> , 2010, 121, 1606-1613.	1.6	273
4	Mitochondrial reactive oxygen species production and elimination. <i>Journal of Molecular and Cellular Cardiology</i> , 2014, 73, 26-33.	1.9	243
5	Adverse Bioenergetic Consequences of Na ⁺ -Ca ²⁺ Exchanger-Mediated Ca ²⁺ Influx in Cardiac Myocytes. <i>Circulation</i> , 2010, 122, 2273-2280.	1.6	76
6	Mitochondrial energetics and calcium coupling in the heart. <i>Journal of Physiology</i> , 2017, 595, 3753-3763.	2.9	67
7	Metabolic alterations in a rat model of takotsubo syndrome. <i>Cardiovascular Research</i> , 2022, 118, 1932-1946.	3.8	31
8	Selective NADH communication from $\hat{\pm}$ -ketoglutarate dehydrogenase to mitochondrial transhydrogenase prevents reactive oxygen species formation under reducing conditions in the heart. <i>Basic Research in Cardiology</i> , 2020, 115, 53.	5.9	28
9	Loss of autophagy protein ATG5 impairs cardiac capacity in mice and humans through diminishing mitochondrial abundance and disrupting Ca ²⁺ cycling. <i>Cardiovascular Research</i> , 2022, 118, 1492-1505.	3.8	18
10	Endogenous nitric oxide formation in cardiac myocytes does not control respiration during $\hat{\pm}$ -adrenergic stimulation. <i>Journal of Physiology</i> , 2017, 595, 3781-3798.	2.9	16
11	The $\hat{\pm}$ 2-isoform of the Na ⁺ /K ⁺ -ATPase protects against pathological remodeling and $\hat{\pm}$ 2-adrenergic desensitization after myocardial infarction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2021, 321, H650-H662.	3.2	12
12	Reply to Blaustein et al.. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2021, 321, H119-H1120.	3.2	0