

Mark J Kohr

List of Publications by Year in descending order

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Version: 2024-02-01

49
papers

2,121
citations

201674

27
h-index

243625

44
g-index

50
all docs

50
docs citations

50
times ranked

2769
citing authors

#	ARTICLE	IF	CITATIONS
1	Inorganic arsenic induces sex-dependent pathological hypertrophy in the heart. American Journal of Physiology - Heart and Circulatory Physiology, 2021, 320, H1321-H1336.	3.2	15
2	An emerging perspective on sex differences: Intersecting S-nitrosothiol and aldehyde signaling in the heart. Redox Biology, 2020, 31, 101441.	9.0	18
3	Nuclear-mitochondrial communication involving miR-181c plays an important role in cardiac dysfunction during obesity. Journal of Molecular and Cellular Cardiology, 2020, 144, 87-96.	1.9	12
4	Identifying Regulators of O-GlcNAcylation in the Broken Heart. FASEB Journal, 2020, 34, 1-1.	0.5	0
5	Abstract 16165: Inorganic Arsenic Induces Sex-Dependent Pathological Cardiac Hypertrophy. Circulation, 2020, 142, .	1.6	0
6	Sustained elevation of MG53 in the bloodstream increases tissue regenerative capacity without compromising metabolic function. Nature Communications, 2019, 10, 4659.	12.8	47
7	Unlocking the Secrets of Mitochondria in the Cardiovascular System. Circulation, 2019, 140, 1205-1216.	1.6	91
8	Inorganic arsenic exposure induces sex-disparate effects and exacerbates ischemia-reperfusion injury in the female heart. American Journal of Physiology - Heart and Circulatory Physiology, 2019, 316, H1053-H1064.	3.2	16
9	A knock-in mutation at cysteine 144 of TRIM72 is cardioprotective and reduces myocardial TRIM72 release. Journal of Molecular and Cellular Cardiology, 2019, 136, 95-101.	1.9	5
10	miR-181c Activates Mitochondrial Calcium Uptake by Regulating MICU1 in the Heart. Journal of the American Heart Association, 2019, 8, e012919.	3.7	18
11	<i>S</i>-Nitrosoglutathione Reductase Is Essential for Protecting the Female Heart From Ischemia-Reperfusion Injury. Circulation Research, 2018, 123, 1232-1243.	4.5	35
12	Kcnj11 Ablation Is Associated With Increased Nitro-Oxidative Stress During Ischemia-Reperfusion Injury. Circulation: Heart Failure, 2017, 10, .	3.9	6
13	Divergent Effects of miR-181 Family Members on Myocardial Function Through Protective Cytosolic and Detrimental Mitochondrial microRNA Targets. Journal of the American Heart Association, 2017, 6, .	3.7	74
14	Adenosine A1 receptor activation increases myocardial protein S-nitrosothiols and elicits protection from ischemia-reperfusion injury in male and female hearts. PLoS ONE, 2017, 12, e0177315.	2.5	18
15	Post-translational Modifications in the Cardiovascular Proteome. , 2016, , 293-320.		0
16	Characterization of the sex-dependent myocardial <i>S</i>-nitrosothiol proteome. American Journal of Physiology - Heart and Circulatory Physiology, 2016, 310, H505-H515.	3.2	35
17	Effect of Sodium Nitrite on Ischaemia and Reperfusion-Induced Arrhythmias in Anaesthetized Dogs: Is Protein S-Nitrosylation Involved?. PLoS ONE, 2015, 10, e0122243.	2.5	19
18	Mitsugumin-53: Potential biomarker and therapeutic for myocardial ischemic injury?. Journal of Molecular and Cellular Cardiology, 2015, 81, 46-48.	1.9	4

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19	Ischaemic preconditioning preferentially increases protein S-nitrosylation in subsarcolemmal mitochondria. <i>Cardiovascular Research</i> , 2015, 106, 227-236.	3.8	74
20	S-nitrosylation of TRIM72 at cysteine 144 is critical for protection against oxidation-induced protein degradation and cell death. <i>Journal of Molecular and Cellular Cardiology</i> , 2014, 69, 67-74.	1.9	61
21	Signaling by S-nitrosylation in the heart. <i>Journal of Molecular and Cellular Cardiology</i> , 2014, 73, 18-25.	1.9	79
22	Postconditioning leads to an increase in protein S-nitrosylation. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2014, 306, H825-H832.	3.2	48
23	Glyceraldehyde-3-Phosphate Dehydrogenase Acts as a Mitochondrial Trans-S-Nitrosylase in the Heart. <i>PLoS ONE</i> , 2014, 9, e111448.	2.5	45
24	Essential role of nitric oxide in acute ischemic preconditioning: S-Nitros(yl)ation versus sGC/cGMP/PKG signaling?. <i>Free Radical Biology and Medicine</i> , 2013, 54, 105-112.	2.9	59
25	S-Nitrosylation: Specificity, Occupancy, and Interaction with Other Post-Translational Modifications. <i>Antioxidants and Redox Signaling</i> , 2013, 19, 1209-1219.	5.4	56
26	Cardioprotective Role of Caveolae in Ischemia-Reperfusion Injury. <i>Translational Medicine (Sunnyvale)</i> , 2013, 1, 1-7.	0.4	7
27	Modulation of myocardial contraction by peroxynitrite. <i>Frontiers in Physiology</i> , 2012, 3, 468.	2.8	15
28	Measurement of S-Nitrosylation Occupancy in the Myocardium With Cysteine-Reactive Tandem Mass Tags. <i>Circulation Research</i> , 2012, 111, 1308-1312.	4.5	70
29	Cysteine 203 of cyclophilin D is critical for cyclophilin D activation of the mitochondrial permeability transition pore.. <i>Journal of Biological Chemistry</i> , 2012, 287, 34496-34498.	3.4	0
30	Prolonged Action Potential and After depolarizations Are Not due to Changes in Potassium Currents in NOS3 Knockout Ventricular Myocytes. <i>Journal of Signal Transduction</i> , 2012, 2012, 1-8.	2.0	6
31	S-nitrosylation: A radical way to protect the heart. <i>Journal of Molecular and Cellular Cardiology</i> , 2012, 52, 568-577.	1.9	92
32	Disruption of Caveolae Blocks Ischemic Preconditioning-Mediated S-Nitrosylation of Mitochondrial Proteins. <i>Antioxidants and Redox Signaling</i> , 2012, 16, 45-56.	5.4	61
33	Characterization of potential S-nitrosylation sites in the myocardium. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2011, 300, H1327-H1335.	3.2	129
34	Mechanism of Cardioprotection: What Can We Learn from Females?. <i>Pediatric Cardiology</i> , 2011, 32, 354-359.	1.3	30
35	Simultaneous Measurement of Protein Oxidation and S-Nitrosylation During Preconditioning and Ischemia/Reperfusion Injury With Resin-Assisted Capture. <i>Circulation Research</i> , 2011, 108, 418-426.	4.5	150
36	Cysteine 203 of Cyclophilin D Is Critical for Cyclophilin D Activation of the Mitochondrial Permeability Transition Pore. <i>Journal of Biological Chemistry</i> , 2011, 286, 40184-40192.	3.4	163

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37	S-nitrosylation of cyclophilin D alters mitochondrial permeability transition pore. <i>FASEB Journal</i> , 2011, 25, 1033.1.	0.5	2
38	Regulation of myocyte contraction via neuronal nitric oxide synthase: role of ryanodine receptor S-nitrosylation. <i>Journal of Physiology</i> , 2010, 588, 2905-2917.	2.9	80
39	cAMP-independent activation of protein kinase A by the peroxynitrite generator SIN-1 elicits positive inotropic effects in cardiomyocytes. <i>Journal of Molecular and Cellular Cardiology</i> , 2010, 48, 645-648.	1.9	34
40	Nitroxyl enhances myocyte Ca ²⁺ transients by exclusively targeting SR Ca ²⁺ -cycling. <i>Frontiers in Bioscience - Elite</i> , 2010, E2, 614-626.	1.8	36
41	Peroxynitrite increases protein phosphatase activity and promotes the interaction of phospholamban with protein phosphatase 2a in the myocardium. <i>Nitric Oxide - Biology and Chemistry</i> , 2009, 20, 217-221.	2.7	27
42	Phosphodiesterase 5 restricts NOS3/Soluble guanylate cyclase signaling to L-type Ca ²⁺ current in cardiac myocytes. <i>Journal of Molecular and Cellular Cardiology</i> , 2009, 47, 304-314.	1.9	42
43	Nitroxyl (HNO) Modifies Cysteine Residues in Phospholamban to Increase Myocyte Ca ²⁺ -Cycling and Contractility. <i>Biophysical Journal</i> , 2009, 96, 515a-516a.	0.5	0
44	Biphasic effect of SIN-1 is reliant upon cardiomyocyte contractile state. <i>Free Radical Biology and Medicine</i> , 2008, 45, 73-80.	2.9	18
45	Nitric oxide signaling and the regulation of myocardial function. <i>Journal of Molecular and Cellular Cardiology</i> , 2008, 45, 625-632.	1.9	130
46	Endothelial nitric oxide synthase decreases β^2 -adrenergic responsiveness via inhibition of the L-type Ca ²⁺ current. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2008, 294, H1473-H1480.	3.2	70
47	Targeting of phospholamban by peroxynitrite decreases β^2 -adrenergic stimulation in cardiomyocytes. <i>Cardiovascular Research</i> , 2008, 77, 353-361.	3.8	41
48	Neuronal nitric oxide synthase signaling within cardiac myocytes targets phospholamban. <i>American Journal of Physiology - Cell Physiology</i> , 2008, 294, C1566-C1575.	4.6	58
49	Synthesis and biological testing of aminoxyls designed for long-term retention by living cells. <i>Organic and Biomolecular Chemistry</i> , 2005, 3, 645.	2.8	25